Analysis of the Anatomical Tachycardia Circuit in Verapamil-Sensitive Atrial Tachycardia Originating From the Vicinity of the Atrioventricular Node

Hiroshige Yamabe, MD; Yasuaki Tanaka, MD; Kenji Morihisa, MD; Takashi Uemura, MD; Koji Enomoto, MD; Hiroaki Kawano, MD; Hisao Ogawa, MD

Background—Calcium channel–dependent tissue has been suggested to be involved in the circuit of verapamil-sensitive atrial tachycardia originating from the atrioventricular (AV) node vicinity (V-AT), but little information exists.

Methods and Results—To examine the tachycardia circuit of V-AT, a single extrastimulus was delivered during tachycardia to 10 sites of the intraatrial septum: the earliest atrial activation site; His bundle (HB) site; 3 arbitrarily divided sites on the AV junction extending from the HB site to the coronary sinus ostium (CSOS) (sites S, M, and I); the internal-CSOS, inferior-CSOS, superior-CSOS, posterior-CSOS, and posteroinferior-CSOS in 10 patients with V-AT. The longest coupling interval that reset V-AT and subsequent return cycle were measured. The longest coupling interval at earliest atrial activation site was significantly longer than the longest coupling interval at the HB site, site S, M, and I, internal-CSOS, inferior-CSOS, superior-CSOS, posterior-CSOS, and posteroinferior-CSOS, respectively ($P<0.001$ for HB site and $P<0.0001$ for the remaining 8 sites). The return cycle at earliest atrial activation site did not differ from the tachycardia cycle length, whereas those at the remaining 9 sites were significantly longer than tachycardia cycle length ($P<0.001$). Furthermore, a single extrastimulus delivered from sites inferior to the HB site advanced His potential without resetting V-AT in 7 patients in whom AV block was not observed during tachycardia.

Conclusions—Atrial tissue within the Koch’s triangle extending from the HB site to posteroinferior-CSOS is not involved in the tachycardia circuit. Verapamil-sensitive atrial tissue close to the AV node but not the AV nodal conducting system forms the tachycardia circuit of V-AT. (Circ Arrhythm Electrophysiol. 2010;3:54-62.)

Key Words: atrioventricular node ■ mapping ■ tachyarrhythmias

Previous studies have reported the presence of focal atrial tachycardia (AT) arising from the apex of the triangle of Koch’s, in close vicinity to the atrioventricular (AV) node.² It has also been suggested that the underlying mechanism of this form of AT is due to reentry, which involves the AV node and/or AV nodal transitional tissues, because verapamil and/or adenosine are effective in terminating tachycardia³; however, less information exists regarding the anatomic tachycardia circuit of this verapamil-sensitive AT originating from the vicinity of the AV node. The purpose of the present study is to define the anatomic tachycardia circuit of this form of AT. Specifically, we examined whether atrial tissue within the Koch’s triangle, including the AV nodal conduction system, is involved in the circuit.

**Methods**

**Patients**

Among 12 consecutive patients with verapamil-sensitive AT who were referred for electrophysiological study and radiofrequency catheter ablation, 10 patients with stable tachycardia cycle length, which fulfilled the inclusion criteria, (5 men and 5 women; mean age, 67 years, range, 40 to 85 years) were included in this study. 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 from each patient. The protocol was approved by the Hospital Human Research Committee. The sensitivity of AT to verapamil was assessed before electrophysiological study by intravenous administration of verapamil at a dosage between 2.5 and 5 mg. The response of AT to verapamil was defined as sensitive if AT was terminated within 5 minutes after the administration of verapamil occurring in the absence of an atrial premature complex.³ Neither structural heart disease nor enlargement of the right or left atrium was found by transthoracic echocardiography in any patients.

**Electrophysiological Study**

The present study was performed with patients in a fasting, nonsedated state. Two 6F quadripolar electrode catheters (St Jude Medical, St Paul, Minn) were percutaneously inserted via the right femoral vein and positioned in the His bundle (HB) region and the right ventricular apex. A 6F 20-pole or decapolar or quadripolar electrode catheter (St Jude Medical or USCI, Billerica, Mass) was percutaneously inserted via the subclavian or jugular vein and introduced into

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the coronary sinus. One or 2 7F large-tip (4 mm in length), deflectable quadrupolar electrode catheters with a 2-mm interelectrode distance (Biosense Webster, Inc, Diamond Bar, Calif, or Japan Lifeline, Tokyo, Japan) were percutaneously inserted via the femoral vein and advanced to the right atrium for atrial mapping, pacing and ablation. Bipolar electrograms from the coronary sinus, HB region, right atrial apex and sequential right atrium were filtered between 30 and 600 Hz and recorded along with the surface ECG with the use of a polygraph (RMC-2000; Nihon Kohden, Tokyo, Japan, or EP-workmate; EP Med Systems, Inc, Mt Arlington, NJ). The right atrium was paced at an output of 2-fold the diastolic threshold and a pulse width of 2 ms using a cardiac stimulator (SEC-4103; Nihon Kohden). To evaluate antegrade and retrograde AV conduction and the mode of induction and termination of tachycardia, atrial and ventricular burst pacing and premature extra-stimulation were delivered from the high right atrium, coronary sinus, and right ventricular apex.

After the baseline electrophysiological study, atrial mapping was performed during tachycardia to identify the earliest atrial activation site (EAAS), using right and left anterior oblique fluoroscopic views. The EAAS was identified by measuring the activation time relative to the onset of P wave on the surface ECG. If P-wave onset was indistinct or obscured by T waves, a stable intracardiac signal (such as from the coronary sinus or HB area, or both) was initially used as a reference. AT was diagnosed using the following criteria: (1) atrial activation sequence during tachycardia was different from that during sinus rhythm; (2) tachycardia initiation was independent of the critical AH interval; (3) induction of tachycardia was independent of AV block; (4) perpetuation of tachycardia was independent of AV block; (5) retrograde atrial activation sequence during ventricular pacing was different from that during tachycardia; (6) ventricular pacing delivered during tachycardia demonstrated AV dissociation without affecting the tachycardia cycle length; (7) change in the A-A interval during tachycardia was reflected in the following V-V interval; (8) tachycardia induction by ventricular pacing was associated with the V-A-A-V activation sequence; and (9) the activation sequence on cessation of ventricular pacing delivered during tachycardia was associated with the A-A-V pattern.

The sensitivity of tachycardia to the administration of adenosine triphosphate was examined during the electrophysiological study. Adenosine triphosphate (5 mg) was administered intravenously as a rapid bolus injection, followed by a 10-ml bolus of normal saline flush. The response of AT to adenosine triphosphate was defined as sensitive if AT was terminated within 20 seconds after the administration of adenosine triphosphate occurring in the absence of an atrial premature complex.9

Study Protocol
After right atriotomy in a biplane view, atrial mapping was performed during AT. To define the atrial activation sequence during AT, atrial mapping was performed within the triangle of Koch’s, including the EAAS and 9 sites in the region of the Koch’s triangle: the HB site, 3 equidistantly divided sites of the AV junction extending from the HB site to the coronary sinus ostium (CSOS) (sites S, M, and I), superior-CSOS, inferior-CSOS, posterior-CSOS, and posteroinferior-CSOS, and internal-CSOS (Figure 1). The location of the EAAS was expressed relative to the location of the HB site. The location of the EAAS relative to the HB site was divided into 6 areas: anterior, posterior, superior, inferior, lateral, and septal portions of the HB site.4 In 2 patients, atrial mapping was also performed using a noncontact mapping system (EnSite 3000; St Jude Medical). A 9F multi-electrode array catheter was introduced from the right femoral 10F sheath into the right atrium, deployed over a 0.035-inch guide wire, and its distal tip was fixed in the right ventricular outflow. The details of EnSite 3000 system have been described previously.10

After atrial mapping, a single extrastimulus was delivered to the EAAS and these 9 sites in the region of the Koch’s triangle during tachycardia (Figure 1). A single extrastimulus was delivered beginning with the tachycardia cycle length and decreased by 10 ms until tachycardia was reset.11 Resetting was defined by the presence of a noncompensatory pause after the extrastimulus was delivered.12 The longest coupling interval of the single extrastimulus that reset the tachycardia and the following return cycle were measured at each site. The coupling interval was measured as the interval from the pacing artifact of the single extrastimulus to the preceding local electrogram, and the return cycle as the interval from the pacing artifact to the following local electrogram. The single extrastimulation was performed during the same tachycardia in all patients. The pacing protocol was performed at least twice at each site. Subsequently, rapid atrial pacing at a rate 5 bpm faster than the tachycardia rate was delivered from the EAAS to demonstrate the concealed entrainment in all patients.

In 2 patients (patients 9 and 10), rapid atrial pacing was delivered during tachycardia to demonstrate manifest entrainment.13,14 A 7F large-tip deflectable catheter was positioned at the EAAS. Then, rapid atrial pacing at a rate 5 bpm faster than the tachycardia rate was delivered from multiple sites of the right atrium using another 7F large-tip, deflectable quadrupolar catheter.

Catheter Ablation
Radiofrequency energy was delivered as a continuous, unmodulated sine waveform at 500 kHz in unipolar mode between the tip of the ablation catheter and a large skin electrode placed under the patient’s back using a radiofrequency energy generator (CABL-IT; Central Inc, Ichikawa, Chiba). The energy was delivered to the earliest site of atrial activation during AT. A current of 5 to 10 W was delivered initially. If the application was ineffective, the radiofrequency power was then increased gradually. A current of 15 to 20 W for 30 seconds was delivered with the temperature limit set at 55°C. An ablation attempt was regarded as successful if the noninducibility by programmed stimulation was confirmed even after the administration of isoproterenol at 1.0 μg/min.

Statistics
Values are expressed as the mean±SD. Statistical difference in electrophysiological data among different extrastimulation sites was analyzed using repeated-measures ANOVA. A paired Student t test with Bonferroni correction was used to examine whether the electrophysiological data at the EAAS differ from those at other sites. Differences between the tachycardia cycle length and the return cycle at each extrastimulation site were analyzed using paired a Student t test. A value of P<0.05 was considered significant.

Results
Termination of AT by the administration of verapamil was observed in all patients before electrophysiological study. Gradual prolongation of AT cycle length was observed
Table. Electrophysiologic Characteristics of AT

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age-Sex</th>
<th>TCL, ms</th>
<th>Earliest Atrial Activation Site</th>
<th>Induction Mode</th>
<th>Induction With AH Jump</th>
<th>Termination Mode</th>
<th>VA Block Rate, bpm</th>
<th>RV Burst During AT</th>
<th>Termination by Verapamil</th>
<th>Termination by ATP</th>
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<td>1</td>
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<td>AE, RAP</td>
<td>VA (−)</td>
<td>VA dissociation</td>
<td>(+)</td>
<td>(+)</td>
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<tr>
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<td>AE, RAP</td>
<td>(−)</td>
<td>AE, RAP</td>
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<td>VA dissociation</td>
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<td>(+)</td>
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<tr>
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<td>AE, RAP</td>
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<td>AE, RAP</td>
<td>VA (−)</td>
<td>VA dissociation</td>
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<td>(+)</td>
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<tr>
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<td>(+)</td>
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<td>AE, RAP</td>
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<td>AE, RAP</td>
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<td>VA dissociation</td>
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<td>AE, RAP</td>
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<td>AE, RAP</td>
<td>160</td>
<td>VA dissociation</td>
<td>(+)</td>
<td>(+)</td>
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</tbody>
</table>

395±61

AE indicates atrial extrastimulation; AH, atrio-His; ATP, adenosine triphosphate; RAP, rapid atrial pacing; RV, right ventricular; TCL, tachycardia cycle length; VA, ventriculoatrial; M, male; F, female.

Response to Single Extrastimulation
AT was reset by a single extrastimulus with an inverse relation between the coupling interval and the following return cycle in all patients. The longest coupling intervals of the single extrastimulus that reset AT at the EAAS, the HB site, sites S, M, and I, inferior-CSOS, internal-CSOS, superior-CSOS, posterior-CSOS, and posteroinferior-CSOS were 385±61, 367±61, 357±67, 349±65, 343±67, 334±61, 322±68, 323±65, 297±57, and 296±60 ms, respectively. Repeated-measures ANOVA revealed a significant difference in the longest coupling interval among different extrastimulus sites (P<0.0001). The longest coupling intervals at the EAAS were significantly longer than at other sites (P<0.01 versus HB site, P<0.001, versus sites S, M, and I, inferior-CSOS, and superior-CSOS, and P<0.0001 versus internal-CSOS, posterior-CSOS, and posteroinferior-CSOS). Repeated-measures ANOVA also revealed a significant difference in the difference between the tachycardia cycle length and longest coupling interval among different extrastimulus sites (P<0.0001). The difference between the tachycardia cycle length and longest coupling interval at the EAAS was shorter than at the other 9 sites (P<0.01 versus HB site, P<0.001, versus sites S, M, and I, inferior-CSOS, and superior-CSOS, and P<0.0001 versus internal-CSOS, posterior-CSOS, and posteroinferior-CSOS) (Figure 3, upper panel).

The return cycle at the EAAS, the HB site, sites S, M, and I, inferior-CSOS, internal-CSOS, superior-CSOS, posterior-CSOS, and posteroinferior-CSOS were 396±60, 415±62, 421±57, 428±60, 435±57, 445±70, 456±61, 453±69, 479±66, and 481±65 ms, respectively. Repeated-measures

during administration of verapamil in all patients. AT was terminated during administration of verapamil in 8 patients (2.5 mg in 5 patients and 5 mg in 3 patients). In the remaining 2 patients, AT was terminated immediately after administration of 5 mg verapamil. AT was induced without an accompanying AH jump and terminated by atrial rapid and extrastimulus pacing, and the atrial activation sequence during tachycardia was different from that during sinus rhythm in all patients. During the induction of AT by atrial extrastimulus pacing, an inverse relationship between A1A2 and A2Ae was observed in all patients. The mean tachycardia cycle length was 395±61 ms (Table). EAAS was observed in the vicinity of the HB site in all patients. The local atrial electrograms near the EAAS did not show fractionated or split potentials in any of the patients. In 2 patients in whom noncontact mapping was used, the EAAS localized by manual mapping coincided with that by the noncontact mapping system (patients 9 and 10). The right atrial endocardial activation times during AT in patients 9 and 10 were 76 ms and 88 ms, respectively. The percentage of right atrial endocardial activation times that spanned the tachycardia cycle length were 18% (patient 9) and 26% (patient 10), respectively. The location of the EAAS relative to the HB site is shown in the Table. Rapid ventricular pacing delivered at a rate faster than the tachycardia cycle length resulted in VA dissociation in all patients (Table). AT was terminated by intravenous administration of 5 mg adenosine triphosphate in all patients.

Atrial Activation Sequence During AT
The intraatrial conduction intervals measured between the onset of the atrial electrogram at the EAAS and at each mapping site during AT are shown in Figure 2.
ANOVA revealed a significant difference in the return cycle among different extrastimulation sites \((P<0.0001)\). The return cycle at the EAAS was significantly shorter than at the HB site, sites S, M, and I, inferior-CSOS, internal-CSOS, superior-CSOS, posterior-CSOS, and posteroinferior-CSOS \((P<0.01\) versus HB site, sites S, M, and I, \(P<0.001\), versus inferior-CSOS, internal-CSOS, and superior-CSOS, and \(P<0.0001\) versus posterior-CSOS and posteroinferior-CSOS). The return cycles at the EAAS did not differ from the tachycardia cycle length, whereas those at the HB site, sites S, M, and I, inferior-CSOS, internal-CSOS, superior-CSOS, posterior-CSOS, and posteroinferior-CSOS were significantly longer than the tachycardia cycle length \((P<0.001\) versus HB site, sites S, M, and I, and \(P<0.0001\) versus remaining 5 sites). Repeated-measures ANOVA revealed a significant difference in the difference between the return cycle length and tachycardia cycle length among different extrastimulation sites \((P<0.0001)\). The difference between the return cycle and the tachycardia cycle length at the EAAS was significantly shorter than at the remaining sites (Figure 3, lower panel).

A single extrastimulus reset the tachycardia as soon as it was delivered during late diastole at the EAAS, whereas a single extrastimulus was unable to reset the tachycardia without capturing the atrial electrograms at the HB site when it was delivered from the site inferior to the HB site. In addition, advancement of His potential without resetting was observed in 7 patients in whom 1:1 AV conduction was observed during tachycardia. These findings suggest that the atrial tissue within the Koch’s triangle, including the AV conduction system, is not involved in the tachycardia circuit of AT.

Figure 4 shows the tracing during resetting of AT by the single extrastimulus delivered from the EAAS in patient 10. The earliest atrial activation was observed in the posterior portion of the HB recording site during tachycardia. A single extrastimulus with a coupling interval of 330 ms delivered from the EAAS reset the tachycardia and the subsequent return cycle was identical to the tachycardia cycle length (Figure 4).

Concealed and Manifest Entrainment

The pacing train delivered from the EAAS accelerated tachycardia to the pacing rate without altering P-wave morphology and the atrial activation sequence in all patients, suggesting entrainment with concealed fusion. The postpacing interval after concealed entrainment was not different from the tachycardia cycle length \((396\pm60\) versus \(395\pm61\) ms, \(P=NS\)). Manifest entrainment was demonstrated by rapid atrial pacing delivered during AT in 2 patients in whom manifest entrainment was attempted. In these patients, the earliest atrial electrogram was orthodromically captured by rapid atrial pacing delivered from the high anterior right atrium (patient 9) and from the high posterior septum (patient 10), respectively. Atrial rapid pacing delivered from the remaining sites of the right atrium, such as the low anterior, low posterior and low septal right atrium, cavotricuspid isthmus, and coronary sinus was unable to capture EAAS orthodromically in both patients. Figure 6 shows the tracing during manifest entrainment in patient 9. During pacing from the high anterolateral right atrium, the atrial electrograms at
the EAAS and the coronary sinus (CS 7–8) were orthodromically captured. Thus, the intervals of the atrial electrograms at the EAAS and CS 7–8 were equal to the pacing interval (400 ms) but were shorter than the AT cycle length (420 ms) (Figure 6). Furthermore, the electrogram morphologies of the EAAS and CS 7–8 during pacing were the same as during AT, suggesting orthodromic capture of the EAAS and CS 7–8 (solid arrow) by pacing from the high anterolateral right atrium. Meanwhile, the atrial electrograms at HRA 7 to 8 were observed 5 ms later than those at CS 7–8 during pacing but were observed 25 ms later than those at CS 7–8 during AT. Furthermore, the interval of the atrial electrogram at HRA 7 to 8 just after pacing was longer than the pacing cycle length and the electrogram morphologies during pacing were different from those during AT at HRA 7 to 8 (Figure 6), suggesting antidromic capture of the electrogram at HRA 7 to 8.
8 (dashed arrow). In addition, the surface P-wave morphologies in leads II and V1, indicated by open arrows, are slightly different from those during tachycardia, indicated by closed arrows, suggesting fusion of the surface P-wave during entrainment (Figure 6). These findings suggest that the high right atrium region including HRA 7 to 8 was antidromically captured, whereas the EAAS and CS regions were orthodromically captured, suggesting fusion of atrial electrograms during entrainment.

Catheter Ablation
The application of radiofrequency energy to the EAAS successfully terminated tachycardia in all patients. The mean number of radiofrequency applications required for successful ablation was 3 ± 2. Accelerated junctional rhythm was observed during energy application in 7 of 10 patients. In 5 patients in whom VA conduction was present before ablation, VA conduction via the AV node persisted after elimination of AT (patients 2, 6, 8, 9, and 10). Also, impairment of AV conduction was not observed in any of the patients. Thus, junctional rhythm might be caused by heating of the anterior transitional zone located in the vicinity of the AV node. There were no complications associated with ablation. During a postablation follow-up period of 25 ± 11 months, there was no recurrence of tachycardia.

Discussion
In the present study, we found that the tachycardia circuit of verapamil-sensitive AT does not involve atrial tissue within the Koch’s triangle extending from the HB site to posteroinferior CSOS. Furthermore, it was shown that the AV conduction system does not participate in the tachycardia circuit of this form of AT. Calcium channel–dependent tissue located close to the AV node but not the AV conducting system was suggested to form the substrate of verapamil-sensitive AT originating from the vicinity of the AV node.

Mechanism of AT and Its Substrate
Tachycardia was induced and terminated by atrial rapid and extrastimulus pacing. An inverse relation of tachycardia induction and resetting was also observed in all patients. In addition, concealed entrainment was shown in all patients. Furthermore, manifest entrainment was demonstrated in 2 patients in whom rapid atrial pacing was attempted from multiple sites of the atrium during tachycardia. These findings suggest that the mechanism of V-AT is due to reentry. Previous studies also suggested reentry as the possible underlying mechanism.1–4,8

Regarding the sensitivity of adenosine and the mechanism of focal AT, Markowitz et al15 proposed that adenosine sensitive focal AT is commonly due to triggered activity or automaticity; however, their adenosine-insensitive focal reentrant ATs showed low-amplitude, fractionated electrograms at the site of origin, which occupy a large percent of tachycardia cycle length,15 different from the ATs presented in this study or those reported previously.1–4,8 In addition, Horie et al16 reported 7 cases of adenosine-sensitive focal reentrant ATs originating from the proximal coronary sinus. Although fractionated electrograms occupying a large percentage of tachycardia cycle length were not observed in their cases, a single extrastimulus delivered from the slow pathway region, distant from the EAAS, reset the AT with a postpacing interval identical to the AT cycle length.16 Furthermore, radiofrequency energy application to this extrastimulation site terminated AT, suggesting that the slow pathway region is part of the reentrant circuit of adenosine-sensitive focal AT originating from the proximal coronary sinus. Thus, adeno-
sine sensitivity is consistent with either calcium channel–
dependent microreentry or c-AMP–dependent triggered ac-
tivity, as suggested by Iwai et al.17

Regarding the substrate of AT originating from the vicinity
of the AV node, previous studies suggested that a calcium
channel–dependent substrate is involved in the reentry circuit
because verapamil and adenosine triphosphate were both
effective in terminating tachycardia.1,3,5,8 Iesaka et al1 sug-
gested that the AV node or its transitional tissue is involved
in this form of tachycardia. Lai et al2 reported that radiofre-
quency energy application to the AT origin near the apex of
the Koch’s triangle produced accelerated junctional rhythm in
4 of 6 patients, suggesting the participation of AV nodal
tissue in the tachycardia substrate. Accelerated junctional
rhythm was also observed in the present 7 patients. In this
study, the extrastimulus began to reset the AT as soon as it
was delivered during late diastole at the EAAS but not at the
remaining 9 sites. The return cycle at the EAAS was almost
identical to the tachycardia cycle length, whereas those at the
remaining sites were longer than the tachycardia cycle length.
These results strongly suggest that atrial and AV nodal tissue
within the Koch’s triangle, extending from the HB site to
posteroinferior-CSOS is not involved in the tachycardia
circuit. In addition, the single extrastimulus delivered from
sites inferior to the HB site was unable to reset AT without
capturing the atrial electrogram at the HB site. Also, advance-
ment of His potential without resetting was observed in
patients in whom 1:1 AV conduction was observed. These
findings suggest that the AV conduction system is not
involved in the tachycardia circuit. Indeed, radiofrequency
energy application selectively eliminated tachycardia without
impairing the AV and retrograde ventriculoatrial conduction
via the AV node. These findings suggest that verapamil-
sensitive atrial tissue close to the AV node but not the AV
conducting system within the Koch’s triangle forms the
substrate of this form of tachycardia.

Alternative AT Origin and
Radiofrequency Ablation

Previously, Ouyang et al18 reported that atrial activation in the
noncoronary sinus was earlier than in the right atrium (8
patients) or the same (1 patient) in 9 patients with focal AT
near the HB region. Although 6 of 9 patients had received
previous unsuccessful ablation of the right atrium, radiofre-
quency ablation to the noncoronary sinus site terminated the
AT in all patients. Thus, they suggest that careful mapping in
the noncoronary aortic sinus is necessary to minimize the
potential risk of injury to the AV node in focal AT near the
AV node.18 Subsequently, Das et al19 reported the usefulness
of noncoronary cusp ablation in peri-AV nodal AT. Radio-
frequency ablation in the noncoronary aortic cusp was suc-
cessful in 7 patients but not in the remaining 3 patients and
required left atrial septal ablation.19 Their result indicates that
the noncoronary sinus is not the only effective site of
ablation, but there is variable location of the AT origin, as
reported previously.8 Although there were no patients in
whom radiofrequency energy application to the EAAS near
the AV node was unsuccessful in this study, the possible
location of AT origin in the left atrial septum or noncoronary
sinus aortic cusp should be considered for ablation of this
form of AT, as suggested previously.8,18,19

Regarding the energy source of catheter ablation, Bastani
et al20 reported that cryoablation of AT near the AV node is
a safe and effective alternative to radiofrequency ablation.
Although impairment of AV conduction was not observed in
this study using radiofrequency energy, cryoablation may be
safer than radiofrequency energy because cryothermal energy
has the advantage of reversible cryothermal mapping.

Tachycardia Circuit of AT

As emphasized previously,21,22 the location of the pacing site
relative to the reentry circuit is critical to demonstrate
manifest entrainment. Demonstration of the orthodromic
capture of EAAS implies that the pacing site is proximal to an
entrance site of the slow conduction area of the reentry
circuit.21 This suggests that the entrance of the slow conduc-
tion area proximal to the EAAS is located in the direction of
the high anterolateral right atrium in patient 9 and high
posterior septum in patient 10, respectively. The inability to
demonstrate manifest entrainment by pacing delivered from
sites inferior to the HB site, such as CSOS and cavotricuspid
isthmus, is also consistent with the results of single extra-
stimulation. Thus, it was suggested that the slow conduction
area of the circuit, which extends from the EAAS, runs in the
high anterolateral and high posterior septal direction in patients 9 and 10 but not in the direction of the Koch’s triangle. The location of the slow conduction area of the circuit was not clear in the remaining 8 patients because manifest entrainment was not attempted; however, the results of single extrastimulation suggest that it also extends from the EAAS in the direction outside the Koch’s triangle in the remaining 8 patients. Although we could not determine the size of the reentry circuit in this study, delivery of a single extrastimulation to the site proximal to the EAAS, which was confirmed by manifest entrainment, or delivery of radiofrequency energy to that site, might be useful to determine the location of the entrance of the circuit.

Regarding the substrate of this form of AT, it has been shown that cells with AV nodal-type or transitional-type action potentials are present in the AV valve. It has been shown that cells with AV nodal-type or transitional-type action potentials are present in the AV valve.23,24 McGuire et al25,26 noted that a sleeve of AV nodal-type tissue, which responds to adenosine, was present around the tricuspid annulus. This AV nodal-like tissue may be a candidate for the substrate of this form of AT.

Figure 6. Tracing during manifest entrainment by rapid atrial pacing delivered during tachycardia from the high anterolateral right atrium (HAL-RA) in patient 9. See text for the discussion. HRA indicates high right atrium; CS, coronary sinus; MAP, mapping catheter; RV, right ventricle.

Conclusion
Mapping the region of the Koch’s triangle using a single extrastimulation method revealed that perinodal atrial tissue extending from the HB site to the posteroinferior-CSOS is not involved in the tachycardia circuit. Verapamil-sensitive atrial tissue close to the AV node but not the AV conduction system forms the tachycardia circuit of the AT originating from the vicinity of the AV node. The slow conduction area of the circuit of AT may extend in the direction outside of the Koch’s triangle.

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Disclosures
None.

References
Calcium channel–dependent tissue has been suggested to be involved in the circuit of verapamil-sensitive atrial tachycardia; however, the results of single extrastimulation revealed that atrial tissue within the Koch triangle extending from His bundle site to posteroinferior coronary sinus ostium, including the atrioventricular nodal conducting system, was not involved in the tachycardia circuit. Orthodromic capture of the earliest atrial activation site by rapid pacing from the sites opposite the direction of he Koch triangle also suggests that the slow conduction area of the circuit of atrial tachycardia extends in the direction opposite of the Koch triangle. Verapamil-sensitive atrial tissue close to the atrioventricular node but not the atrioventricular nodal conducting system forms the tachycardia circuit of verapamil-sensitive atrial tachycardia. These findings suggest that verapamil-sensitive atrial tachycardia is a distinct entity of tachycardia different from the atrioventricular nodal reentrant tachycardia.
Analysis of the Anatomical Tachycardia Circuit in Verapamil-Sensitive Atrial Tachycardia Originating From the Vicinity of the Atrioventricular Node
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