Atrial Tachycardia Arising Adjacent to Noncoronary Aortic Sinus
Distinctive Atrial Activation Patterns and Anatomic Insights

Xingpeng Liu, MD,*† Jianzeng Dong, MD,* Siew Yen Ho, PriD,‡ Ashok Shah, MD,† Deyong Long, MD,* Ronghui Yu, MD,* Ribo Tang, MD,* Meleze Hocini, MD,† Michel Haissaguerre, MD,† Changsheng Ma, MD*

Beijing, China; Bordeaux, France; and London, United Kingdom

Objectives
We sought to determine whether atrial tachycardia arising adjacent to the noncoronary aortic sinus (NCAS-AT) has distinctive atrial activation patterns in relation to targeted anatomic imaging.

Background
The knowledge of atrial activation patterns of the NCAS-AT and its anatomic basis is very limited.

Methods
Three-dimensional electroanatomic mapping was performed during NCAS-AT in 13 patients and during sequentially pacing from the noncoronary aortic sinus (NCAS) and the para-Hisian atrial area in 15 reference patients. The spatial relationship between the NCAS and the contiguous atria was studied in another 25 reference patients using computed tomography and in 12 human hearts using gross and microscopic anatomic examination.

Results
During NCAS-AT, the para-Hisian area of the right atrium (RA) and the anteroseptal region of the left atrium were activated almost simultaneously. The initial activation area (within first 20 ms of atrial depolarization) was relatively wide (9.3 ± 2.6 cm² on the RA map and 8.1 ± 2.1 cm² on the left atrium map). In reference patients, NCAS pacing reproduced a biatrial activation pattern of NCAS-AT and resulted in a wider initial activation area than the para-Hisian atrial pacing within first 20 ms of RA activation (10.1 ± 3.0 cm² vs. 3.9 ± 1.7 cm²; p < 0.001). Anatomically, the wall of NCAS did not contain myocardial tissue, but was intimately related to the paraseptal regions of the atria such that the shortest distances from the NCAS to the RA and the left atrium were 1.7 ± 0.6 mm and 2.3 ± 0.9 mm (p < 0.01), respectively.

Conclusions
NCAS-AT has distinct atrial activation patterns that can be explained in part by its spatial anatomic relationship with the atria. (J Am Coll Cardiol 2010;56:796–804) © 2010 by the American College of Cardiology Foundation

Focal atrial tachycardia (AT) preferentially arises from certain anatomic regions such as crista terminalis, ostium of the coronary sinus (CS), pulmonary veins, and atrial appendages. Most of them are amenable to catheter ablation with a high success rate (1). Over the past several years, AT arising adjacent to the noncoronary aortic sinus (NCAS), an uncommon source, has been reported (2–5), but the knowledge of local anatomic substrate of AT arising adjacent to the NCAS (NCAS-AT) is very limited. Besides, the diagnosis of NCAS-AT may be challenging because there are no specific electrophysiologic characteristics, suggesting that the focus lies in the NCAS, and the site of earliest atrial activation during NCAS-AT consistently is found in the para-Hisian location mimicking a true para-Hisian AT. Inappropriate diagnosis potentially can damage atrioventricular conduction if ablation is performed near the bundle of His. Hence, we aimed to investigate the characteristics and implications of atrial activation patterns of NCAS-AT with a 3-dimensional (3D) electroanatomic mapping system and to perform detailed imaging of the spatial relationship between the NCAS and the atria using computed tomography (CT) and histologic techniques.

Methods

Study population. The study consisted of 2 sections: 3D atrial mapping and the clinical anatomic examination and...
imaging. Thirteen consecutive patients with NCAS-AT (AT group) and 15 patients with paroxysmal atrial fibrillation who underwent pulmonary vein isolation (reference group) were enrolled in the former group. Another 25 patients with paroxysmal atrial fibrillation were enrolled in the latter group. With the exception of 1 patient who had tachycardia-mediated cardiomyopathy, all patients with NCAS-AT had structurally normal hearts. Mean duration of symptomatic AT was 2.2 ± 0.6 years. Three patients in the AT group previously underwent unsuccessful catheter ablation elsewhere. All antiarrhythmic drugs were withdrawn for at least 5 half-lives before electrophysiologic examination. No patient received amiodarone. The characteristics of all the patients are summarized groupwise in Table 1.

**Study protocols. 3-DIMENSIONAL ATRIAL MAPPING.** Electrophysiologic examination was performed in the AT and reference groups. A decapolar and 3 quadripolar mapping catheters were used to map the CS, high right atrium, right ventricular apex, and His bundle, respectively. A 3.5-mm, deflectable quadripolar saline-irrigated catheter compatible with the 3D electroanatomic mapping system (Navi-Star Thermo-Cool and CARTO XP, respectively, Biosense-Webster, Inc., Diamond Bar, California) was used for mapping and ablation. The diagnosis of focal AT arising from the atrial septal region was based on the criteria described elsewhere (6). First, 3D right and left atrial activation maps were created sequentially during sustained AT and were displayed together in multiple-map view. Then, earliest atrial activation sites were tagged on the right atrium (RA) and left atrium (LA) maps. Next, the aortic root map was created under angiographic guidance. The diagnosis of NCAS-AT was confirmed by the fact that the earliest activation was earlier in the NCAS than in the RA or LA (Fig. 1). Finally, the activation area within first 20 ms of atrial depolarization was calculated automatically by the software built into the CARTO XP system and displayed on the isochronal maps. The initial atrial activation area was considered diffuse if it was 6 cm² or more. We selected this value based on the following considerations. During electrophysiologic examination, Lin et al. (7) found mean atrial septal conduction velocity of 98 cm/s in 21 subjects with structurally normal hearts. Thus, an impulse originating from a focal source will propagate over 1.96 cm radial distance within 20 ms, and for a focal AT originating from the septum, atrial activation in the first 20 ms occurs over approximately a 12-cm² area. Because the para-Hisian area is contiguous with tricuspid annulus, a barrier for atrial wave propagation, the atrial activation area within the first 20 ms for para-Hisian AT would be one-half that value, that is, 6 cm². Other parameters measured on 3D maps include the proportion (percentage) of area activated in first 20 ms in each atrium, the distance between the earliest activation sites in NCAS and each atrium, and the distance between earliest activation sites in the RA and LA.

In the reference group, patients were subjected to the following protocol after pulmonary vein isolation. Unipolar pacing (MicroPace Cardiac Stimulator, Micropace EP, Inc., Santa Ana, California) via the distal electrode of mapping catheter was carried out at threshold output and 2-ms pulse duration to minimize the capture of surrounding tissue. Pacing was performed at a mean cycle length of 400 ms. First, we created biatrial 3D activation maps during NCAS pacing. The pacing site was located at the inferoposterior wall of NCAS on the aortic root angiogram. Then, the RA map was created during para-Hisian pacing. The pacing site was in accordance with the site of earliest RA activation during NCAS pacing and was located posterosuperior to the His bundle. Finally, the atrial activation area within the first 20 ms of atrial depolarization was displayed on isochronal maps. Intra-atrial conduction times also were measured.

In the AT group, radiofrequency catheter ablation was attempted at the earliest activation site in NCAS with an average power of 30 W, temperature control set at 43°C, and saline irrigation at 17 ml/min. Ablation was considered effective if tachycardia terminated within 10 s of application. A successful end point was defined as noninducibility of AT from rapid atrial pacing combined with administration of isoproterenol.

**CLINICAL ANATOMY AND IMAGING.** A dual-source CT scan (Somatom Definition, Siemens, Forchheim, Germany) was undertaken in 25 reference patients during sinus rhythm. The heart was scanned from the middle of the ascending aorta to the diaphragm with 120 ml intravenous iopromide (Ultravist, Schering, Berlin, Germany) injected at 4 to 5 ml/s during a single breathhold. Reformatted 2-dimensional and 3D images were obtained by post-processing and volume-rendered reconstructions, respectively. By post-processing, slices that were perpendicular to long axis of aortic root were reconstructed from the bottom of NCAS (near aortic valve) to the midpoint of NCAS (plane with maximal convexity) with slice thickness and distance of 1 mm each. In each slice, the smallest distances between the endocardium of NCAS and the endocardium of RA and LA, respectively, were measured by digital calipers. According to the slice number,
the entire measuring zone was divided into 2 equal parts, the higher segment and the lower segment. The smallest distance between the NCAS and each atrium within each segment was calculated by averaging the measurements obtained from each slice within the segment (Fig. 2).

Histologic sections held in the tissue archive of the Cardiac Morphology unit at the Royal Brompton Hospital, London, were retrieved. Serial sections from 12 structurally normal hearts were reviewed to establish the composition of the sinusal portions of the aortic wall and the proximity of the sinuses to atrial tissues.

**Statistical analysis.** Data are expressed as mean ± SD. Baseline characteristics among the 3 patient groups and the CT data of 4 segments were subjected to analysis of variance with a post hoc test for continuous and chi-square or Fisher exact tests for categorical data. The mapping data from the NCAS-AT and the pacing groups were compared using the Student t test. Data obtained from the NCAS and para-
Hisian pacing performed sequentially were analyzed using a paired $t$ test. A value of $p < 0.05$ was considered statistically significant. SPSS software version 11.5 (SPSS, Inc., Chicago, Illinois) was used for statistical analysis.

**Results**

**P-wave morphology during NCAS-AT.** During NCAS-AT, the P-wave in lead I was isoelectric in 10 patients, slightly negative in 1 patient, and positive in 2 patients. Lead aVL showed a slightly positive P-wave in 10 and an isoelectric P-wave in the remaining 3 patients. In the inferior leads, the P-wave was predominantly negative in 12 patients; however, 7 of them had a slight positive deflection terminally in lead II or III. One patient had an isoelectric P-wave in the inferior leads. In lead V1, the P-wave showed a negative–positive (biphasic) pattern in 11 patients and was exclusively positive in the remaining 2 patients (Fig. 3).

**3D atrial activation mapping.** In the AT group, sustained AT (mean cycle length, 391 ± 79 ms) was induced easily and was terminated by programmed atrial stimulation. RA activation mapping localized earliest atrial activity to the para–Hisian area associated with His bundle potential. Similarly, LA activation mapping localized earliest atrial activity to the anteroseptal LA that was not associated with His bundle potential (Fig. 1). The linear distance between the earliest sites in the RA and LA was a mean ± SD of 8 ± 3 mm. The earliest activation site on the RA map preceded the onset of tachycardia P-wave by 23 ± 3 ms (range 19 to 27 ms), whereas the earliest activation site on the LA map preceded the onset of P-wave by 20 ± 2 ms (range 16 to 23 ms) ($p = \text{NS}$). Notably, the earliest activation site consistently was earlier in the RA than the LA by 3 ± 2 ms in all but 1 patient (LA preceded RA by 4 ms). Further mapping within the NCAS recorded activity that preceded the onset of tachycardia P-wave by 37 ± 3 ms (range 29 to 43 ms) and the earliest sites in the RA and LA by 15 ± 4 ms (range 6 to 20 ms) and 17 ± 4 ms (range 9 to 22 ms), respectively (all $p < 0.01$) (Fig. 1). The linear distances from the earliest site in NCAS to the earliest sites in RA and LA were 7 ± 2 mm and 8 ± 3 mm, respectively. Globally, both the atria were depolarized simultaneously, but complete atrial activation took more time in the RA than in the LA (118 ± 11 ms vs. 77 ± 23 ms) ($p < 0.05$).

Remarkably, the activation area within the first 20 ms of atrial depolarization was a diffuse zone in the paraseptal areas of the atria (Fig. 4). The initial activation areas in the RA and the LA were 9.3 ± 2.6 cm² and 8.1 ± 2.1 cm², respectively (Table 2).

Regarding ablation during NCAS-AT, first application of radiofrequency energy targeting the earliest site in NCAS terminated AT in 12 patients. The mean duration of ablation was 4.6 ± 1.6 s (range 1.7 to 6.7 s). Acceleration of AT before termination was observed in 2 of 13 patients (Fig. 5). After 2 ± 1 radiofrequency applications, the ablation end point was achieved in all patients. No compli-
cation was noted. At 13 ± 3 months of follow-up, no recurrence has been reported.

In the reference group, the pacing threshold in the NCAS was 8 ± 3 mA. The earliest atrial activation was located in the para-Hisian area in 87% (13 of 15) of patients. The atrial activation area within the first 20 ms of atrial depolarization and various intra-atrial activation times were comparable between the NCAS pacing and the NCAS-AT (Tables 2 and 3). The mean pacing threshold in the para-Hisian area was 2 ± 1 mA. As summarized in

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**Figure 3** Representative P-Wave Morphologic Features on 12-Lead ECG in 2 Patients With NCAS-AT

Each boxed P-wave is enlarged in the pane on the right to show the negative-positive P-wave in lead V1 (blue arrow) and the negative-positive P-wave (blue arrow) and negative P-wave (red arrow) in lead III. ECG = electrocardiogram; NCAS-AT = atrial tachycardia arising adjacent to the noncoronary aortic sinus; other abbreviations as in Figure 1.

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**Figure 4** Isochronal Atrial Maps During NCAS-AT

(Left) Initial atrial activation area (brown) within the first 20 ms was 10.7 cm² in the RA. (Right) Initial atrial activation area within the first 20 ms was 11.4 cm² in the LA. Abbreviations as in Figures 1 and 3.
was localized (3.9 ± 0.6 mm) during para-Hisian pacing (34 ± 13 ms) than NCAS pacing (16 ± 6 ms) (p < 0.001). In contrast to the diffuse area of initial RA activation during NCAS pacing (10.1 ± 3.0 cm²), it was localized (3.9 ± 1.7 cm²) during para-Hisian pacing (p < 0.001) (Fig. 6).

Clinical anatomy and imaging. There were 11 ± 0.9 slices (range 10 to 13 slices) from the bottom to the middle of NCAS. In all the patients, NCAS was adjacent to the paraseptal regions of the atria. However, in 88% (22 of 25) of patients, NCAS was closer to the RA than the LA, with shortest distance of 1.7 ± 0.6 mm (range 1 to 3.3 mm) versus 2.3 ± 0.9 mm (range 1.4 to 4.6 mm) (p < 0.01) in all the segments, 1.8 ± 0.7 mm versus 2.5 ± 0.9 mm (p < 0.01) in the higher segment and 1.6 ± 0.6 mm versus 2.1 ± 0.9 mm (p = 0.025) in the lower segment. Moreover, the minimum distance (1 mm) between NCAS and atria was always documented in the lower part of the NCAS, particularly in the rightward NCAS.

In the anatomic specimens, the NCAS can be seen adjacent to the atrial myocardium. We found the deepest part of the NCAS overlying the paraseptal areas of the atria in 3 hearts and abutting this area in the remainder (Fig. 7). On histologic examination, it can be seen that the wall of the NCAS does not contain myocardium. Instead, the deepest sinusal wall adjoins atrial walls in some hearts or is separated by a thin layer of fibrofatty tissues in others. The upper parts of the NCAS are further away from the atria, concurring with the observations made on CT imaging.

### Table 2: 3-Dimensional Electroanatomic Atrial Mapping Data

<table>
<thead>
<tr>
<th></th>
<th>NCAS-AT (n = 13)</th>
<th>NCAS Pacing (n = 15)</th>
<th>Para-Hisian Pacing (n = 15)</th>
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</thead>
<tbody>
<tr>
<td>Left atrium</td>
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<td></td>
<td></td>
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<tr>
<td>Sample points</td>
<td>56 ± 14</td>
<td>50 ± 16</td>
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</tr>
<tr>
<td>Volume (ml)</td>
<td>65.1 ± 14.2</td>
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</tr>
<tr>
<td>IAA (cm²)</td>
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<td>9.0 ± 3.1</td>
<td>—</td>
</tr>
<tr>
<td>IAA%</td>
<td>12.1 ± 3.9</td>
<td>12.7 ± 4.2</td>
<td>—</td>
</tr>
<tr>
<td>Right atrium</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sample points</td>
<td>66 ± 27</td>
<td>70 ± 18</td>
<td>68 ± 17</td>
</tr>
<tr>
<td>Volume (ml)</td>
<td>72.1 ± 16.1</td>
<td>70.3 ± 15.1</td>
<td>71.4 ± 14.1</td>
</tr>
<tr>
<td>IAA (cm²)</td>
<td>9.3 ± 2.6*</td>
<td>10.1 ± 3.0†</td>
<td>3.9 ± 1.7</td>
</tr>
<tr>
<td>IAA%</td>
<td>9.0 ± 3.5*</td>
<td>9.8 ± 3.1†</td>
<td>4.2 ± 1.6</td>
</tr>
</tbody>
</table>

* p < 0.001 (during NCAS-AT versus during para-Hisian pacing). †p < 0.001 (during NCAS pacing versus during para-Hisian pacing).

AT = atrial tachycardia; IAA = initial activation area within the first 20 ms; NCAS = noncoronary aortic sinus.

### Table 3: Endocardial Conduction During NCAS-AT and Pacing Protocol

<table>
<thead>
<tr>
<th></th>
<th>NCAS-AT (n = 13)</th>
<th>NCAS Pacing (n = 15)</th>
<th>Para-Hisian Pacing (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NCAS to EAS in RA</td>
<td>15 ± 4</td>
<td>18 ± 6</td>
<td>—</td>
</tr>
<tr>
<td>NCAS to EAS in LA</td>
<td>17 ± 4</td>
<td>20 ± 7</td>
<td>—</td>
</tr>
<tr>
<td>NCAS/para-Hisian to HRA</td>
<td>37 ± 7</td>
<td>40 ± 5</td>
<td>43 ± 8</td>
</tr>
<tr>
<td>NCAS/para-Hisian to CS</td>
<td>35 ± 10</td>
<td>36 ± 12</td>
<td>34 ± 13</td>
</tr>
<tr>
<td>His to CS (ms)</td>
<td>14 ± 3*</td>
<td>16 ± 6†</td>
<td>34 ± 13</td>
</tr>
<tr>
<td>CS to CS (ms)</td>
<td>25 ± 7</td>
<td>26 ± 9</td>
<td>29 ± 11</td>
</tr>
</tbody>
</table>

*p < 0.001 (during NCAS-AT versus during para-Hisian pacing); †p < 0.001 (during NCAS pacing versus during para-Hisian pacing).

CSp = proximal pair (CS9-10) of coronary sinus electrodes; CSd = distal pair (CS1-2) of coronary sinus electrodes; EAS = earliest activation site; HRA = high right atrium; RA = right atrium; other abbreviations as in Tables 1 and 2.

Table 3, pacing from the NCAS and the para-Hisian area resulted in comparable times to activate the CS ostium and the high RA. However, the time of impulse propagation from the His bundle to the CS ostium was longer during para-Hisian pacing (34 ± 13 ms) than NCAS pacing (16 ± 6 ms) (p < 0.001). In contrast to the diffuse area of initial RA activation during NCAS pacing (10.1 ± 3.0 cm²), it was localized (3.9 ± 1.7 cm²) during para-Hisian pacing (p < 0.001) (Fig. 6).

### Figure 5: Ablation in 1 Patient With NCAS-AT

(Left) The CARTO map displays the activation of right atrium, left atrium, and NCAS during NCAS-AT. Note that the earliest activation site during tachycardia was located at the NCAS. During ablation targeting the earliest site in the NCAS (red point), AT terminated after 4.1 s of ablation. Note that the AT cycle length did not change before AT termination (Right). MAP = mapping catheter; RF = radiofrequency; other abbreviations as in Figures 1 and 3.
Fibrofatty tissues fill the increasing gap. On routine histologic examination, the atrial myocardium against the outside of the sinus wall does not show specialization.

Discussion

Main findings. This study described 3D atrial activation patterns of NCAS-AT and explored the anatomic relationship between the NCAS and the atria. Our major findings are as follows. First, during NCAS-AT, the initial atrial activation patterns have 2 distinct characteristics: 1) the atrial activation starts in the right atrial para-Hisian area followed a little later by the activation of the left atrial anteroseptal area; and 2) the activation area within the first 20 ms is diffuse. Second, the NCAS pacing was able to replicate the initial atrial activation patterns of NCAS-AT and resulted in a diffuse and larger initial RA activation area compared with the para-Hisian pacing suggestive of the origin lying adjacent to the NCAS. Third, NCAS is closely related to the paraseptal regions of the atria and can provide an easy access to focal sources during the ablation of paraseptal AT.

P-wave morphology: NCAS-AT versus septal AT. It is unknown whether P-wave morphologic features can be used as a tool to distinguish septal AT from the NCAS-AT or the right- or left-sided ATs. Marrouche et al. (8) reported that the P-wave in V1 and inferior leads was positive (n = 3), positive-negative (n = 1), and negative (n = 1) in 5 patients with left septal AT. Most NCAS-AT patients in our study displayed a negative-positive P-wave in lead V1 and a negative-positive or negative P-wave in the inferior leads. As reported by Chen et al. (9), right mid-septal AT also showed biphasic P-wave in lead V1 and negative P-wave in the inferior leads, although the authors did not specify if the biphasic P-wave in their study was negative-positive or positive-negative. Hence, we speculate that based on surface P-wave morphologic features, it may be difficult to differentiate NCAS-AT from right mid-septal AT.

Atrial activation patterns during NCAS-AT. Atrial activation patterns are critical for localizing the origin of focal AT. During NCAS-AT, the depolarization of atria occurs almost simultaneously in the para-Hisian RA and anteroseptal LA. Considering a mean distance of 8 mm and a time difference of 3 ms between the earliest atrial sites in RA and LA, theoretical transseptal conduction velocity would be 260 cm/s. However, as we mentioned above, the mean atrial septal conduction velocity is 98 cm/s in normal human hearts (7). Therefore, it is unlikely that during NCAS-AT, atrial activation occurred by impulse conduction from the earliest site in one atrium to the other across the septum. The only explanation for this phenomenon is that the earliest sites in the 2 atria are becoming activated simultaneously from an adjacent source in the NCAS (Fig. 7). Also, it would explain why NCAS activation consistently preceded atrial activation during AT.

Activation of focal AT usually begins and radiates from a focal source. However, there is limited information on the characteristics of initial propagation of wavefront in focal AT. More recently, 2 studies reported that the origin and the surrounding area during tachycardia resulting from focal AT were characterized by a higher
it can be stated that during the initial phase of propagation of a typical focal AT, the atrial activation area should be smaller than during subsequent propagation. Our study showed that the mean interval from the origin of NCAS-AT to its breakthrough sites in the atria was 15 to 17 ms. This finding indicates that the so-called initial atrial activation during NCAS-AT actually occurs after the initial slow conduction phase is finished. This may explain why a diffuse area of atrial activation could be recorded within the first 20 ms on 3D isochronal maps during NCAS-AT.

Correlation between atrial activation patterns and anatomic features. Atrial activation characteristics of NCAS-AT probably are explained by the anatomic findings. Spatially, NCAS is related closely to the paraseptal regions of the atria, and its lower rightward part lies closest to the RA. In accordance with the anatomic features, para-Hisian RA was the earliest atrial activation site, and anteroseptal LA was only slightly later. In the minority of patients in the AT (1 of 13) and reference (2 of 15) groups, the LA was activated slightly earlier than the RA, which is consistent with the CT findings, where NCAS was found to be located closer to the LA than RA in 3 of 25 patients.

As shown in Figure 7, a peculiar histologic feature between the NCAS and the contiguous atria provides a possible explanation for the origin and activation pattern of NCAS-AT and also an anatomic basis for some unusual epicardial anteroseptal accessory pathways (14). Instead from NCAS, the NCAS-AT seems to originate from epicardially located paraseptal atrial myocardial tissue lying adjacent to NCAS. Notably, this myocardial tissue lies in closer proximity to the lower part of NCAS than to the atrial endocardium, causing access from the atria to be difficult during endocardial ablation. However, the energy delivered from the NCAS during epicardial approach results in successful termination of tachycardia with greater ease. In addition, these muscle bundles are interspersed by fibrofatty tissues, which can explain the initial slow conduction occurring before biatrial breakthrough of NCAS-AT.

Clinical implications of atrial activation patterns of NCAS-AT. The clinical implications of these results are as follows: 1) if both the atria are mapped during AT, it facilitates the diagnosis of NCAS-AT based on its distinctive biatrial activation pattern; and 2) although it is difficult to make a diagnosis of NCAS-AT based on the results of RA mapping, our results strongly suggest that a diffuse initial activation in the para-Hisian RA highly favors mapping in the NCAS before LA. If it is inconclusive, then LA mapping is recommended.

Study limitations. It would have been ideal to subject patients with clinical AT to the pacing protocol. Because it would have been an unduly long procedural time for the patients in the AT group, we recruited a reference group to study atrial activation patterns using the pacing protocol. Also, the absolute number of patients with clinical AT is
small. Nevertheless, this is the largest series of patients among the studies on NCAS-AT published so far.

**Conclusions**

NCAS-AT has distinctive initial atrial activation patterns, including almost simultaneous activation of the biatrial paraseptal region and diffuse area of initial activation in each of the atria, in concurrence with the characteristic anatomic features of this region. These activation patterns have implications in improved diagnosis of NCAS-AT, thereby minimizing the risk of inadvertent ablation in the parahisian region. Moreover, in the absence of myocardial tissue in the NCAS, peri-NCAS-AT may be an appropriate terminology.

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**References**


**Key Words:** activation pattern • anatomy • atrial tachycardia • noncoronary aortic sinus • 3-dimensional.