Atypical Fast-Slow Atrioventricular Nodal Reentrant Tachycardia Incorporating a “Superior” Slow Pathway

A Distinct Supraventricular Tachyarrhythmia

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Background—The existence of an atypical fast-slow (F/S) atrioventricular nodal reentrant tachycardia (AVNRT) including a superior (sup) pathway with slow conductive properties and an atrial exit near the His bundle has not been confirmed.

Methods and Results—We studied 6 women and 2 men (age, 74±7 years) with sup-F/S-A VNRT who underwent successful radiofrequency ablation near the His bundle. Programmed ventricular stimulation induced retrograde conduction over a superior SP with an earliest atrial activation near the His bundle, a mean shortest spike-atrial interval of 378±119 milliseconds, and decremental properties in all patients. sup-F/S-A VNRT was characterized by a long-RP interval; a retrograde atrial activation sequence during tachycardia identical to that over a sup-SP during ventricular pacing; ventriculoatrial dissociation during ventricular overdrive pacing of the tachycardia in 5 patients or atrioventricular block occurring during tachycardia in 3 patients, excluding atrioventricular reentrant tachycardia; termination of the tachycardia by ATP; and a V-A-V activation sequence immediately after ventricular induction or entrainment of the tachycardia, including dual atrial responses in 2 patients. Elimination or modification of retrograde conduction over the sup-SP by ablation near the right perinodal region or from the noncoronary cusp of Valsalva eliminated and confirmed the diagnosis of AVNRT in 4 patients each.

Conclusions—sup-F/S-A VNRT is a distinct supraventricular tachycardia, incorporating an SP located above the Koch triangle as the retrograde limb, that can be eliminated by radiofrequency ablation. (Circulation. 2016;133:114-123. DOI: 10.1161/CIRCULATIONAHA.115.018443.)

Key Words: ablation techniques ■ arrhythmias, cardiac ■ atrioventricular node ■ electrophysiology ■ tachycardia

Atrioventricular nodal reentrant tachycardia (AVNRT), which uses pathways within the atrioventricular node as critical limbs of reentrant circuits, has been divided among slow-fast, fast-slow (F/S), and slow-slow forms on the basis of the relative duration of atrio-His (AH) versus His-atrial (HA) conduction and the site of earliest atrial activation during the tachycardia.1 F/S-AVNRT, characterized by a longer HA than AH interval and an earliest site of atrial activation in the posteroseptal region, incorporates a fast pathway (FP) as the anterograde limb and a typical slow pathway (SP) oriented inferiorly as the retrograde limb.1 Several investigators, however, have described a rare subtype of F/S-AVNRT, with a site of earliest activation located in the superior aspect of the Koch triangle near the His bundle (HB), referred to here as sup-F/S-AVNRT, which has not been widely accepted as a distinct supraventricular tachyarrhythmia. Furthermore, an atypical and distinct arrhythmogenic atrioventricular nodal substrate responsible for sup-F/S-AVNRT has not been confirmed electrophysiologically, although an atypical SP anterior to the FP has been suspected in rare cases of AVNRT.4,5 This retrospective, multicenter study was designed to confirm the existence of a sup-F/S-AVNRT, with a focus on the presence and characteristics of a “superior” SP located in the HB region and used as the retrograde limb of the reentrant circuit.

Clinical Perspective on p 123

Methods

We retrospectively identified 6 women and 2 men 74±7 years of age (range, 59–82 years) who underwent successful ablation of a
sup-FS-AVNRT at 5 Japanese medical institutions. This study complied with the guidelines of the Declaration of Helsinki and was approved by the institutional review board of the Gunma University Hospital. Written informed consent to participate in this study was obtained from all patients.

Electrophysiological Study

The patients underwent electrophysiological study and catheter ablation after the discontinuation of all antiarrhythmic drugs for ≥5 half-lives. Four multipolar electrode catheters were placed in the high right atrium (RA), HB region, right ventricular (RV) apex, and coronary sinus for the recording of endocardial electrograms and for programmed atrial and ventricular stimulation. In each patient, atrioventricular nodal and ventriculoatrial conduction was studied in detail, and tachycardia was induced by atrial or ventricular overdrive or extrastimulation at baseline, during infusion of isoproterenol, or both. If ventriculoatrial conduction was not detected during this stimulation protocol, RV apical extrastimuli were delivered after simultaneous high RA and RV apical overdrive pacing with or without isoproterenol infusion to promote ventriculoatrial conduction. The retrograde activation sequence during tachycardia, RV pacing, or both was determined by the recording of the atrial electrogram in the HB region and near the coronary sinus, and the earliest site of retrograde activation was confirmed by mapping around the Koch triangle with an ablation catheter during tachycardia or RV pacing. RV apical extrastimulation or entrainment pacing was performed during sustained tachycardia to determine whether it was reset or entrained, and a bolus of ATP was administered in an initial dose of 2 mg and gradually increased until the tachycardia was terminated to examine its sensitivity to ATP.

A superior SP was strongly suspected when the earliest retrograde atrial activation immediately after cessation of RV pacing appeared reproducibly in the HB region and near the coronary sinus, and the earliest site of retrograde activation was confirmed by mapping around the Koch triangle with an ablation catheter during tachycardia or RV pacing. RV apical extrastimulation or entrainment pacing was performed during sustained tachycardia to determine whether it was reset or entrained, and a bolus of ATP was administered in an initial dose of 2 mg and gradually increased until the tachycardia was terminated to examine its sensitivity to ATP.

Catheter Ablation

During ongoing tachycardia, activation mapping, starting in the RA, was performed with a 7F, 4-mm tip, nonirrigated ablation catheter. A CARTO 3-dimensional mapping system (Biosense Webster, Diamond Bar, CA) was used in 4 patients, whereas catheter activation mapping was performed in the other 4 patients. The delivery of radiofrequency energy was targeted to the earliest site of atrial activation during ongoing tachycardia. When mapping in the RA failed to identify an optimally early ablation site relative to the onset of the P wave or when the delivery of radiofrequency at the earliest site of RA activation failed to eliminate the tachycardia, the aortic cusps, visualized by aortic root angiography, were mapped with a catheter advanced from the right femoral artery. When the earliest activation site was in the RA, a power of 5 to 10 W was delivered initially.
If the application was ineffective, the radiofrequency power was gradually increased to 15 to 20 W for 30 seconds, with the temperature limited to 55°C. If the site of earliest activation was in an aortic cusp, the delivery of radiofrequency began at 20 W, with a target temperature of 55°C and a power limited to 35 W. The timing of the local atrial activation relative to the P wave and the amplitude of the local atrial electrogram relative to the RV electrogram (A/V ratio) at the successful ablation site were measured. We did not use cryoablation or other means such as intracardiac or transesophageal echocardiography to identify the earliest site of activation. An ablation attempt was successful when the tachycardia was noninducible by programmed stimulation with and without the administration of isoproterenol.

### Electrocardiographic Analysis

Surface 12-lead electrocardiograms of the spontaneous or inducible tachycardia were used to determine the P-wave polarity. P waves that were not fused with the previous T wave or QRS complex were chosen for analysis. The P wave was visually assigned by consensus of 2 observers among positive, negative, or biphasic (+/− or −/+ morphologies.

### Patient Follow-Up

The patients were followed up at 2 to 3 weeks after the ablation procedure and at 6-month intervals thereafter. The success of radiofrequency catheter ablation was verified by history and by 24-hour ambulatory electrocardiography recordings to exclude recurrences of the tachycardia. Values are expressed as mean±SD.

### Results

#### Patient Characteristics

All patients were free of structural heart disease. All complained of palpitations, and 3 had experienced syncope. Symptoms had been present for a mean of 15±22 months (range, 1–60 months). The ventricular rate during tachycardia was 148±11 bpm (range, 135–162 bpm). No pre-excitation was observed during sinus rhythm in any patient. The arrhythmia was paroxysmal with prolonged periods of normal sinus rhythm in all patients but was occasionally incessant in 2 patients. A single patient was unsuccessfully treated with pilsicainide.

#### Baseline Atrioventricular and Ventriculoatrial Nodal Conduction

Baseline anterograde atrioventricular nodal conduction was normal in all but patient 7, who presented with first-degree atrioventricular block (Table 1). Anterograde dual atrioventricular nodal physiology, evidenced by a sudden increase in the AH interval, was observed in 2 patients. Retrograde conduction over the FP was observed in 4 patients, and retrograde conduction over a typical SP, with a site of earliest atrial activation at the ostium of the coronary sinus, was observed in 1 patient.

#### Conduction Properties of the Superior SP

At baseline, retrograde conduction over the superior SP was observed during ventricular overdrive pacing in 5 patients and exclusively after ventricular extrastimulation preceded by a simultaneous atrioventricular pacing drive in 3 other patients (Table 2 and Figure 1A). A DAR was elicited during ventricular overdrive stimulation in 2 patients and after

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### Table 1. Atrioventricular Nodal Function Before and After Ablation

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, Sex</th>
<th>Before Ablation</th>
<th>After Ablation</th>
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<td>Mean</td>
<td>74±7</td>
<td>104±33</td>
<td>42±10</td>
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A/G DAVNP indicates anterograde dual atrioventricular nodal physiology; AH, atrio-His; ERPFP, anterograde effective refractory period of fast pathway; HV, His-ventricular; NM, not measured; R/G FP, retrograde conduction over fast pathway; R/G SP, retrograde conduction over slow pathway with the earliest atrial activation site at the proximal coronary sinus; and WBCL, maximum pacing cycle inducing Wenckebach type AH block.

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### Figure 2.

Magnified P waves recorded in leads I, II, and V1 during tachycardia (top to bottom tracings) in patients 1, 2, 3, 5, 6, 7, and 8. The P wave is negative in lead II in patients 1 and 8 and biphasic in the other patients. A long-RP tachycardia was present in all patients but patient 7.
ventricular extrastimulation preceded by a simultaneous atrio-
ventricular pacing drive in 1 patient. Decremental conduction
over the superior SP was demonstrable in all patients. A ventricular atrioventricular conduction curve was constructed, and the effective refractory period of the superior SP was measured in 6 patients who underwent ventricular extrastimulation. The occurrence of anterograde conduction over the superior SP was equivocal in 2 patients presenting with anterograde dual AVN physiology.

**Surface ECG During Tachycardia**
A 12-lead ECG was recorded during spontaneous tachycardia in all patients (Figure 2). All patients but 1 (patient 7), whose atrioventricular conduction was fragile, was presented with a long-RP tachycardia. The P wave was narrow, consistent with a septal origin, and its polarity in lead II was biphasic in 5 and deeply negative in 2 patients.

**Electrophysiological Diagnosis of AVNRT**
Atrial extrastimulation reproducibly induced the tachycardia in 5 patients; atrial overdrive pacing, in 1 patient; and both techniques, in 2 patients (Table 2). Ventricular overdrive pacing induced the tachycardia in 2 patients, ventricular extrastimulation in 2 patients, and both techniques in 1 patient. Isoproterenol was needed to induce the tachycardia in a single patient. The arrhythmia induced in the electrophysiological laboratory was a long-RP tachycardia in 7 patients, manifested by a ratio of HA interval to tachycardia cycle length \(>0.5\), except in patient 7, whose AH and HA intervals were identical. The atrial activation sequence during tachycardia was identical to that over a superior SP during ventricular pacing in all patients (Figure 1). The overdrive pacing criterion was fulfilled in 5 patients. Transient second-degree atrioven-
tricular block during tachycardia, the strongest sign excluding the participation of the ventricles in the reentry circuit, developed during spontaneous or induced tachycardia in 3 patients. In 7 patients, atrioventricular reentrant tachycardia was excluded by fulfillment of the overdrive pacing criterion, development of second-degree atrioventricular block during ongoing tachycardia, or both. In the remaining patient, atrio-
ventricular reentrant tachycardia was excluded only by differ-
tential entrainment pacing. Atrial tachycardia was positively excluded by the observation of a V-A-V activation sequence after induction or entrainment of the tachycardia from the RV in 6 patients (Figure 3A), DAR after induction of the tachycardia from the RV in the other 2 patients, and termination of the tachycardia by ventricular pacing without atrial capture in 1 of the 2 other patients (Figure 4). In 6 patients, a V-A-V response was elicited on induction of the tachycardia during ventricular pacing. In 2 of these 6 patients, ventricular entrainment was confirmed by 1:1 retrograde conduction over the superi-
or SP, followed by a V-A-V response. Multiple DARs were induced by extrastimulation at a wide range of coupling inter-
vals in patient 6, and a single DAR was induced after over-
drive pacing in patient 1. In patient 2, both a V-A-V activation sequence and a DAR were observed during an electrophysi-
ological study (Figure 3). The injection of ATP during ongo-
ing tachycardia, in an average 3-mg bolus, gradually increased the atrial cycle length and HA interval before terminating the tachycardia, which ended with a ventricular electrogram in 7 patients, consistent with a sensitivity to ATP of the superior SP. In patient 7, whose anterograde conduction over the FP was fragile, the tachycardia ended with an atrial electrogram, consistent with a diagnosis of AVNRT.
Catheter Ablation and Follow-Up

The tachycardia was successfully ablated from the noncoronary sinus of Valsalva (NCSV) in 4 patients (Figure 5) and from the right-sided perinodal region in 4 patients (Figure 6 and Table 3). In the 4 patients whose successful ablation was from the NCSV, activation mapping in the RA and the aortic cusps revealed the site of earliest activation in the NCSV with a mean interval between the local atrial electrogram and P-wave onset of $-26\pm4$ milliseconds and a mean A/V of $1.7\pm0.7$. In 1 of these 4 patients (patient 4), ablation of a typical SP was attempted in the right posterior septum, which failed to eliminate the tachycardia. In 3 patients whose activation mapping was limited to the RA and in patient 2 whose site of earliest activation was in the NCSV, ablation was successfully performed in the right-sided anterosetal perinodal region, posterior and superior to the HB recording site, at the earliest atrial electrogram, with a mean interval between the local atrial electrogram and P-wave onset of $-35\pm24$ milliseconds and a mean A/V of $1.3\pm0.3$. An HB electrogram was never detected at the site of successful ablation. The tachycardia was terminated at a mean of $1.5\pm0.7$ seconds after radiofrequency delivery in 6 patients to whom it was delivered during ongoing tachycardia. Ectopic junctional complexes developed during radiofrequency delivery in 3 patients (Table 3 and Figure 6B). Retrograde conduction over the superior SP was completely eliminated after successful ablation in all patients but patient 3, in whom retrograde conduction over the superior SP was slower after ablation (Table 3). After ablation, neither

Figure 3. Induction of tachycardia with an initiating V-A-V (A) and V-A-A-V (B) activation sequence in patient 2. A. After the first and second stimulation of triple right ventricular apical (RVA) stimulation at an S-S cycle length of 320 milliseconds, retrograde conduction over the fast pathway (FP) is visible, with the earliest site of atrial activation in the His bundle (HB) region and a gradual increase in the S-atrial interval. When the third S caused a marked increase in the ventriculoatrical interval, retrograde conduction over the superior slow pathway (SP) appears, with the site of earliest atrial activation in the HB region (dashed arrow), followed by anterograde conduction over the FP. B. During double RVA stimulation at an S-S cycle length of 290 milliseconds, no retrograde conduction to the atria was observed after the first S, whereas the first and second atrial electrograms in response to the second S were activated retrogradely (dashed arrows) via the FP and the superior SP, respectively. Note the interval between first and second atrial electrograms of 296 milliseconds, which is shorter than the tachycardia cycle length of 363 milliseconds. I, II, V$_1$, and V$_6$ are surface ECG; HB electrogram (HBE) 1-2 and 9-10 are distal to proximal HB region; and CS9-10 to 1-2 are proximal to distal coronary sinus (CS) recording. HRA indicates high right atrium.
Kaneko et al
Atypical AVNRT and Superior Slow Pathway

Atrioventricular block nor abnormal anterograde conduction over the FP was observed in any patient (Table 1), and AVNRT was no longer inducible by atrial or ventricular stimulation before or during the infusion of isoproterenol. Over a follow-up of 38±32 months, no patient complained of a recurrence of tachycardia (Table 3).

Discussion

Presence and Characteristics of the Superior SP

Although the anatomic existence of a superior SP has never been confirmed,19 several investigators have hypothesized its presence as the retrograde limb of a reentrant circuit of atypical AVNRT.2,3 Nawata et al3 reported 3 cases of atypical AVNRT identical to our sup-F/S-A VNRT. However, they were unable to confirm the presence of retrograde conduction over the SP by ventricular stimulation and did not proceed with its ablation. Otomo et al2 described 9 cases of atypical AVNRT with electrophysiological characteristics similar to those of our sup-F/S-AVNRT. Successful ablation was achieved in the mid septum in the majority of patients, which did not fully support the presence of a superior SP. Among 6 patients who underwent intraoperative ice mapping, Keim et al5 observed a single case of typical AVNRT in which the SP was anterior to the FP. It is noteworthy that, in our study, the presence of the superior SP was confirmed by the successful elimination or modification of the putative superior SP by the delivery of radiofrequency to its atrial insertion, near the HB.

The origin of the superior SP remains unclear. Although a recent study of the embryological development of the specialized conduction system hypothesized the presence of an unspecified arrhythmogenic substrate in the space between the atrial myocardium adjacent to the NCSV and the central fibrous body,20 we found no evidence in favor of a superior SP as an anatomic structure already formed in the developmental process. Instead, we hypothesize that the advanced age of our population might have promoted a structural or electrophysiological (or both) remodeling of the atrioventricular nodal tissue, acquiring the properties of superior SP.

The electrophysiological properties of the superior SP, including slow and decremental conduction, ATP sensitivity, and the development of an accelerated junctional rhythm during radiofrequency delivery, correspond to those of a typical atrioventricular nodal SP. Moreover, considering a 3-dimensional relationship between the site of successful ablation and the HB, the superior SP may consist of atrioventricular nodal tissue that extends from the compact atrioventricular node superiorly toward the NCSV along the tricuspid annulus. The atrial insertion of the superior SP seemed electrically connected to the adjacent atrial muscle, which, during the

Figure 4. Termination of tachycardia at an atrial cycle length of 410 milliseconds by right ventricular (RV) overdrive pacing at a cycle length of 380 milliseconds in patient 1. A long ventriculoatrial interval and an earliest site of atrial activation in the His bundle (HB) region were observed in response to the first 2 ventricular stimuli, consistent with retrograde conduction over the superior slow pathway (SP). After a slight rate-dependent increase in ventriculoatrial conduction over the superior SP between the first and second paced cycles, the third and fourth stimuli are blocked. In response to the fifth ventricular stimulus, a short ventriculoatrial interval and the earliest site of retrograde atrial activation in the HB region (HBE 1–2) were observed, consistent with retrograde conduction over a fast pathway (FP), followed by 2:1 ventriculoatrial conduction over that pathway. The numbers between atrial electrograms at the high right atrium (HRA) indicate the cycle length in milliseconds. The perpendicular dotted lines indicate the onset of the earliest atrial electrogram at HBE1-2 during retrograde conduction over the superior SP and FP, respectively. I, II, V, and V are surface ECG; HBE1-2 and 9-10 are distal to proximal HB region; and CS9-10 to 1-2 are proximal to distal coronary sinus (CS) recording. Reprinted from Kaneko et al4 with permission of the publisher. Copyright © 2014, the International Heart Journal Association.
Tachycardia, was detected as the atrial electrogram at the site of earliest activation. Interindividual variations in the distance measured between the site of successful ablation and the HB (patients 1–4 in Table 3) seem to reflect interindividual variations in the length of the superior SP.

**Diagnosis of AVNRT**

In our study, atrial tachycardia was excluded in all patients by a V-A-V response, including DAR. Furthermore, the V-A-V response observed in our patients confirmed that the FP and the superior SP are the anterograde and retrograde limbs, respectively, of the sup-F/S-AVNRT.

In contrast to the high success rate of ventricular entrainment pacing of the typical F/S-AVNRT, the successful entrainment or termination of the sup-F/S-AVNRT by ventricular pacing was evidently more challenging, despite the confirmation of retrograde conduction over the superior SP before the onset of tachycardia. Ventriculoatrial dissociation during ventricular entrainment pacing was, in fact, observed in 5 of 8 patients, and termination of the tachycardia without atrial capture was observed in a single patient. The inability to entrain the tachycardia with ventricular overdrive pacing is an important criterion to exclude atrioventricular reentry using an accessory pathway. Although it is not specific, this phenomenon is characteristic of the sup-F/S-AVNRT and may be attributable to a functional ventriculoatrial conduction block below the atroventricular nodal reentrant circuit (the lower common pathway) during ongoing tachycardia. The cause of a high rate of ventriculoatrial
block during tachycardia at the level of the lower common pathway is uncertain, although its length, which varies among forms of AVNRT, may be related to its conduction properties.23

Similarity to Adenosine-Sensitive Atrial Tachycardia Originating From the Perinodal Region

The clinical entity of ATP-sensitive atrial tachycardia originating near the HB is widely recognized.17,18,25,26 Intra-atrial reentry, the circuit of which includes ATP-sensitive perinodal atrial tissue, has been proposed as the mechanism of that arrhythmia.17,25 The electrophysiological characteristics of this atrial tachycardia, including a long RP, an earliest site of atrial activation near the HB, ATP sensitivity, and a successful ablation site in the perinodal region or NCSV,17,18,25,26 are strikingly similar to those of the sup-F/S-AVNRT. Moreover, as described earlier, the following key diagnostic criteria of this atrial tachycardia are satisfied in some cases of sup-F/S-AVNRT: a V-A-A-V activation sequence after ventricular induction of the tachycardia as a result of DAR14–16,21 and ventriculoatrial dissociation produced by ventricular overdrive.

Figure 6. Intracardiac recordings during tachycardia immediately before (A) and during (B) successful radiofrequency delivery and fluoroscopic views showing the position of the catheters (C and D) and CARTO activation maps of the right atrium and noncoronary aortic cusp during tachycardia (E and F) in the right and left oblique projections, respectively, in patient 2, whose successful ablation was in the right-sided perinodal region. A, The atrial electrogram at the distal pole of the ablation catheter (ABL1-2) precedes the onset of the P wave by 12 milliseconds. B, The tachycardia ended 1.3 seconds after the radiofrequency delivery (RF on), followed by the development of several ectopic junctional cycles (asterisks) with the earliest activation in the His bundle region (HBE) before returning to sinus rhythm (SR). CS indicates coronary sinus catheter; HRA, high right atrium; and RVA, right ventricular apical catheter. E and F, The distance between the successful site (red tag and solid arrows) and the nearest HB (yellow tags) measured 10.7 mm.
stirrulation during the tachycardia caused by ventriculoaortial block in the lower common pathway. Therefore, one might hypothesize that the reentrant circuit of an ATP-sensitive atrial tachycardia originating from the HB region is the same as that used in sup-F/S-A VNRT. However, this hypothesis may be difficult to verify because, if ventriculoaortial block is consistently observed during ongoing tachycardia, ventricular stimulation cannot ensnare or terminate an ongoing tachycardia, neither confirming a diagnosis of AVNRT nor excluding a diagnosis of atrial tachycardia. Further studies are needed to identify differences in the components of the reentrant circuit of a sup-F/S-A VNRT compared with an ATP-sensitive atrial tachycardia.

Catheter Ablation of the sup-F/S-A VNRT

Our study showed that the sup-F/S-A VNRT is curable by catheter ablation of the atrial insertion of the superior SP, at the site of earliest atrial activation, or at the optimal site during tachycardia from either the NCSV or the right-sided perinodal region, without impairing atrioventricular nodal conduction. The incomplete elimination of retrograde conduction over the superior SP may be an acceptable endpoint, as in the case of catheter ablation of typical F/S-A VNRT. However, this was a small, retrospective study that limited enrollment to successful ablations. Therefore, the overall safety and efficacy of this therapy and the proper selection of right-sided perinodal ablation sites compared with NCSV remain to be firmly established. A larger, prospective study is needed to clarify these issues. In contrast to the catheter ablation of typical SP, the development of an accelerated junctional rhythm during ablation of the superior SP was infrequent and was not a reliable indicator of the heating effect on the superior SP. We hypothesize that ablation at precisely the atrial insertion of the superior SP caused exit block of retrograde conduction, concealing the accelerated junctional complexes during radiofrequency delivery.

Conclusions

The sup-F/S-A VNRT is a distinct clinical entity that involves a superior SP located above the Koch triangle as the retrograde limb that can be eliminated by radiofrequency ablation. We recommend systematically mapping the retrograde activation of the SP before attempting its ablation in patients with confirmed F/S-A VNRT.

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Disclosures

None.

References

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The usual arrhythmogenic substrate of atrioventricular nodal reentrant tachycardia (AVNRT) is a slow pathway (SP) that
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F/S-AVNRT is a distinct supraventricular tachycardia, incorporating an SP above the Koch triangle as the retrograde limb,
that can be eliminated by radiofrequency ablation. Its formal inclusion in the differential diagnosis of long-RP tachycardia
should be reflected in a corresponding adaptation of the ablation therapy for refractory AVNRT.
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