Atypical Fast-Slow Atrioventricular Nodal Reentrant Tachycardia
Incorporating a “Superior” Slow Pathway:
A Distinct Supraventricular Tachyarrhythmia

Running title: Kaneko et al.; Atypical AVNRT and superior slow pathway

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Abstract

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

Methods and Results—We studied 6 women and 2 men (74 ± 7 years of age) with sup-F/S-AVNRT who underwent successful radiofrequency ablation near the HB. Programmed ventricular stimulation induced retrograde conduction over a sup-SP with an earliest atrial activation near the HB, a mean shortest spike-atrial interval of 378 ± 119 ms and decremental properties in all patients. sup-F/S-AVNRT was characterized by 1) a long RP interval, 2) a retrograde atrial activation sequence during tachycardia identical to that over a sup-SP during ventricular pacing, 3) ventriculoatrial dissociation during ventricular overdrive pacing of the tachycardia in 5 patients, or AV block occurring during tachycardia in 3 patients, excluding AV reentrant tachycardia, 4) termination of the tachycardia by adenosine triphosphate and 5) 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 1) near the right perinodal region, or 2) from the non-coronary cusp of Valsalva, eliminated and confirmed the diagnosis of AVNRT in 4 patients each.

Conclusions—sup-F/S-AVNRT is a distinct supraventricular tachycardia, incorporating a SP located above Koch’s triangle as the retrograde limb, which can be eliminated by radiofrequency ablation.

Key words: ablation; arrhythmia (heart rhythm disorders); atrioventricular node; electrophysiology test; tachyarrhythmia; atrioventricular nodal reentrant tachycardia; superior slow pathway; supraventricular tachycardia ablation
Atrioventricular (AV) nodal reentrant tachycardia (NRT), which utilizes pathways within the AV node as critical limbs of reentrant circuits, has been divided among slow-fast, fast-slow (F/S) and slow-slow forms, based on the relative duration of atrio-His (AH) versus His-atrial (HA) conduction, and on the site of earliest atrial activation during the tachycardia. 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. Several investigators, however, have described a rare subtype of F/S AVNRT, with a site of earliest activation located in the superior (sup) aspect of Koch’s triangle, near the His bundle (HB), referred to here as superior type of F/S AVNRT (sup-F/S-AVNRT), which has not been widely accepted as a distinct supraventricular tachyarrhythmia. Furthermore, an atypical and distinct arrhythmogenic AV 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. 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 utilized as the retrograde limb of the reentrant circuit.

Methods

We retrospectively identified 6 women and 2 men, 74 ± 7 years of age (range 59 to 82 years), who underwent successful ablation of a sup-F/S-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 electrophysiologic 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, respectively, for the recording of endocardial electrograms (EGM) and for programmed atrial and ventricular stimulation. In each patient, AV nodal and ventriculoatrial (VA) conduction was studied in detail, and the tachycardia was induced by atrial or ventricular overdrive or extrastimulation at baseline, or during infusion of isoproterenol, or both. If VA conduction was not detected during this stimulation protocol, RV apical extrastimuli were delivered following simultaneous high RA and RV apical overdrive pacing with or without isoproterenol infusion to promote VA conduction. The retrograde activation sequence during tachycardia, RV pacing or both was determined by the recording of the atrial EGM in the HB region and near the coronary sinus, and the earliest site of retrograde activation was confirmed by mapping around Koch’s triangle, using 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 adenosine triphosphate (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, followed by anterograde conduction over a FP at least once, excluding retrograde conduction over the FP pathway (figure 1A). The existence of a superior SP was ultimately confirmed by the successful elimination of VA conduction following its ablation in the HB region. When possible, its retrograde conductive properties, including conduction time and effective refractory period were evaluated by
means of a VA conduction curve with ventricular premature stimuli or overdrive stimulation. The presence of a slowly conducting AV accessory pathway was excluded, as described later.

The diagnosis of sup-F/S-AVNRT, utilizing a FP as the anterograde and the superior SP as the retrograde pathway was made according to previous reports.\textsuperscript{2,3} Briefly, the intracardiac EGM during tachycardia were characterized by 1) a longer HA than AH interval, and 2) an earliest site of atrial activation in the HB region during tachycardia, identical to the retrograde conduction observed over the superior SP during RV pacing (Figure 1). Moreover, a diagnosis of orthodromic AV reentrant tachycardia incorporating a slowly conducting AV or nodoventricular AP\textsuperscript{7} as the retrograde limb was excluded by one or all of the following observations: 1) inability to modify or entrain the tachycardia by ventricular overdrive pacing, i.e. presence of VA dissociation during ventricular overdrive pacing of the tachycardia\textsuperscript{8,9} (overdrive pacing criterion), 2) development of 2\textsuperscript{nd} degree AV block during ongoing tachycardia,\textsuperscript{10} and 3) a shorter stimulus-to-atrial-EGM interval during entrainment pacing from the RV apex than from the RV base (differential entrainment pacing).\textsuperscript{11} The diagnosis of atrial tachycardia was excluded by one or both of the following observations: 1) termination of the tachycardia by ventricular pacing without atrial capture,\textsuperscript{12} and 2) V-A-V activation sequence after ventricular induction/re-initiation of the tachycardia, resulting from retrograde conduction over the superior SP followed by anterograde conduction over the FP,\textsuperscript{13} including dual atrial responses (DAR) from simultaneous retrograde conduction over FP and superior SP after the last ventricular stimulus. A DAR was diagnosed as a V-A-A-V activation sequence with an A-A interval shorter than the subsequent tachycardia cycle length and both earliest atrial activations in the HB region.\textsuperscript{14-16}

**Catheter ablation**

During ongoing tachycardia, activation mapping, starting in the RA, was performed using a 7F,
4-mm tip, non-irrigated ablation catheter. A CARTO™ 3-dimensional mapping system (Biosense Webster, Diamond Bar, CA) was used in 4 patients, while 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 sec, 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 degree 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 EGM relative to the RV EGM (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 non-inducible 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 the analysis. The P wave was visually assigned by consensus of two observers among positive, negative, or biphasic (+/- or -/+ ) morphologies.
Patient follow-up

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

Results

Patient characteristics

All patients were free from structural heart disease. All complained of palpitations and 3 had experienced syncope. Symptoms had been present for a mean 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, though was occasionally incessant in 2 patients. A single patient was unsuccessfully treated with pilsicainide.

Baseline atrioventricular and ventriculoatrial nodal conduction

Baseline anterograde AV nodal conduction was normal in all but patient no 7, who presented with 1st degree AV block (table 1). Anterograde dual AV nodal physiology, evident 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 slow pathway

At baseline, retrograde conduction over the superior SP was observed during ventricular overdrive pacing in 5 patients and, in 3 other patients, exclusively after ventricular
extrastimulation preceded by a simultaneous AV pacing drive (table 2; figure 1A). A DAR was elicited during ventricular overdrive stimulation in 2 patient and after ventricular extrastimulation preceded by a simultaneous AV pacing drive in 1 patient. Decremental conduction over the superior SP was demonstrable in all patients. A VA 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 electrocardiogram during tachycardia**

A 12-lead electrocardiogram was recorded during spontaneous tachycardia in all patients (figure 2). All patients but one (no 7), whose AV conduction was fragile, 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 atrioventricular nodal reentrant tachycardia**

Atrial extrastimulation reproducibly induced the tachycardia in 5 patients, atrial overdrive pacing in 1, and both techniques in 2 patient (table 2). Ventricular overdrive pacing induced the tachycardia in 2 patients, ventricular extrastimulation in 2 and both techniques in 1 patient.

Isoproterenol was needed to induce the tachycardia in a single patient. The arrhythmia induced in the electrophysiologic laboratory was a long RP tachycardia in 7 patients, manifest by a >0.5 HA interval/tachycardia cycle length ratio, except in patients no 7, whose AH and HA intervals were identical. The atrial activation sequence during tachycardia was identical to that over a sup-SP during ventricular pacing in all patients (figure 1). The overdrive pacing criterion was fulfilled in 5 patients. Transient 2nd degree AV 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, AV reentrant tachycardia was excluded by a) fulfillment of the overdrive pacing criterion, or b) development of 2nd degree AV block during ongoing tachycardia, or c) both observations. In the remaining patient, AV reentrant tachycardia was excluded only by differential entrainment pacing. Atrial tachycardia was positively excluded by the observation of a) a V-A-V activation sequence after induction or entrainment of the tachycardia from the right ventricle in 6 patients (figure 3A), b) DAR after induction of the tachycardia from the right ventricle in the other 2 patients, and c) termination of the tachycardia by ventricular pacing without atrial capture in 1 of these 2 other patients (figure 4). In 6 patients, a V-A-V response was elicited upon induction of the tachycardia during ventricular pacing. In 2 of these 6 patients, ventricular entrainment was confirmed by 1:1 retrograde conduction over the superior SP, followed by a V-A-V response. Multiple DAR were induced by extrastimulation, at a wide range of coupling intervals in patient no 6, and a single DAR was induced following overdrive pacing in patient no 1. In patient no 2, a V-A-V activation sequence and a DAR were both observed during an electrophysiologic study (figure 3). The injection of ATP during ongoing tachycardia, in an average 3-mg bolus, gradually increased the atrial cycle length and the HA interval, before terminating the tachycardia, which ended with a ventricular EGM in 7 patients, consistent with a sensitivity to ATP of the superior SP. In patient no 7, whose anterograde conduction over the FP was fragile, the tachycardia ended with an atrial EGM, consistent with a diagnosis of AVNRT.

Catheter ablation and follow-up

The tachycardia was successfully ablated from the non-coronary sinus of Valsalva (NCSV) in 4 patients (figure 5) and from the right-sided perinodal region in 4 patients (figure 6; 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 ± 4 ms and a mean A/V of 1.7 ± 0.7. In 1 of these 4 patients (no 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 patient no 2, whose site of earliest activation was in the NCSV, ablation was successfully performed in the right-sided anteroseptal perinodal region, posterior and superior to the HB recording site, at the earliest atrial EGM, with a mean interval between the local atrial electrogram and P wave onset of -35 ± 24 ms and a mean A/V of 1.3 ± 0.3. A HB electrogram was never detected at the site of successful ablation. The tachycardia was terminated at a mean of 1.5 ± 0.7 sec 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, figure 6B). Retrograde conduction over the superior SP was completely eliminated after successful ablation in all but patient no 3, in whom retrograde conduction over the superior SP was slower after ablation (table 3). After ablation, neither AV 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 slow pathway

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.\textsuperscript{2,3} Nawata et al. reported 3 cases of atypical AVNRT identical to our sup-F/S-AVNRT. However, they were unable to confirm the presence of retrograde conduction over the SP by ventricular stimulation and did not proceed with its ablation.\textsuperscript{3} Otomo et al. described 9 cases of atypical AVNRT with electrophysiological characteristics similar to those of our sup-F/S-AVNRT, in whom successful ablation was achieved in the mid septum in the majority of patients,\textsuperscript{2} which did not fully support the presence of a superior SP. Among 6 patients who underwent intraoperative ice-mapping, Keim et al. observed a single case of typical AVNRT where the SP was anterior to the FP.\textsuperscript{5} 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 radiofrequency delivery 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,\textsuperscript{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 AV 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 AV nodal SP. Moreover, considering a three-dimensional relationship between the site of successful ablation and the HB, the superior SP may consist of AV nodal tissue that extends from the compact AV 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 tachycardia, was detected as the atrial EGM at the site of earliest activation. Interindividual variations in the distance measured between the site of successful ablation and the HB (patients 1 to 4 in table 3) seem to reflect interindividuva variations in the length of the superior SP.

**Diagnosis of atrioventricular nodal reentrant tachycardia**

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 with 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. VA 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 AV reentry using an AP. While it is not specific, this phenomenon is characteristic of the sup-F/S-AVNRT, and may be attributable to a functional VA conduction block below the AV nodal reentrant circuit (the lower common pathway) during ongoing tachycardia. The cause of a high rate of VA block during tachycardia at the level of the lower common pathway is uncertain, though its length, which varies among forms of AVNRT, may be related to its conduction properties.

**Similarity with adenosine-sensitive atrial tachycardia originating from the perinodal region**

The clinical entity of ATP-sensitive atrial tachycardia originating from near the HB is widely recognized. Intraatrial reentry, the circuit of which includes ATP-sensitive perinodal
atrial tissue, has been proposed as the mechanism of that arrhythmia. 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, 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: 1) A-V-A-V activation sequence after ventricular induction of the tachycardia due to DAR, and 2) VA dissociation produced by ventricular overdrive stimulation during the tachycardia, due to VA 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-AVNRT. However, this hypothesis may be difficult to verify because, if VA block is consistently observed during ongoing tachycardia, ventricular stimulation cannot entrain 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-AVNRT versus an ATP-sensitive atrial tachycardia.

**Catheter ablation of the superior type of F/S AVNRT**

Our study showed that the sup-F/S-AVNRT 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 from the right-sided perinodal region, without impairing AV 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 AVNRT. However, this was a small, retrospective study, which limited its enrolment to successful ablations. Therefore, the overall safety and efficacy of this therapy, and the proper selection of right-sided perinodal
ablation sites versus 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.28

Conclusions

The sup-F/S-AVNRT is a distinct clinical entity, which involves a superior SP located above Koch’s triangle as the retrograde limb, and which can be eliminated by radiofrequency ablation. We recommend, in patients with confirmed F/S AVNRT, to systematically map the retrograde activation of the SP before attempting its ablation.

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Conflict of Interest Disclosures: None.

References:


Clinical Perspective

The usual arrhythmogenic substrate of atrioventricular (AV) nodal reentrant tachycardia (NRT) is a slow pathway (SP) that can be ablated in the posterior or mid septum. We identified 8 patients with a rare, superior (sup-) type of fast-slow (F/S-) AVNRT incorporating a sup-SP located near the His bundle (HB). Programmed ventricular stimulation induced retrograde conduction over the sup-SP with an earliest atrial activation near the HB, a mean shortest stimulus-atrial interval of 378 ± 119 ms and decremental properties in all patients. sup-F/S-AVNRT was characterized by 1) a long RP interval, 2) a retrograde atrial activation sequence during tachycardia identical to that during retrograde conduction over a sup-SP during ventricular pacing, 3) ventriculoatrial dissociation during ventricular overdrive pacing of the tachycardia or AV block occurring during tachycardia, excluding AV reentrant tachycardia, 4) termination of the tachycardia by adenosine triphosphate and 5) a V-A-V activation sequence immediately after ventricular induction or entrainment of the tachycardia, including dual atrial responses. Elimination or modification of retrograde conduction over the sup-SP by ablation 1) near the right perinodal region, or 2) from the non-coronary cusp of Valsalva, eliminated and confirmed the diagnosis of AVNRT in 4 patients each. sup-F/S-AVNRT is a distinct supraventricular tachycardia, incorporating a SP above Koch’s triangle as the retrograde limb, which 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.
Table 1. AV nodal function before and after ablation.

<table>
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<th>Patient no</th>
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<td></td>
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<td>HV</td>
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Means: 74±7 104±33 42±10 376±52 313±45 99±33 39±6 335±33 244±29

A/G DAVNP = anterograde dual AV nodal physiology; ERPFP = anterograde effective refractory period of fast pathway; 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; WBCL = maximum pacing cycle inducing Wenckebach type AH block.
Table 2. Electrophysiological characteristics of superior slow pathway and atrioventricular nodal reentrant tachycardia.

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<th>Patient no</th>
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<th>ISO</th>
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<th>AH, ms</th>
<th>HA, ms</th>
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<td>+/-/-</td>
<td>-</td>
<td>2.5</td>
</tr>
<tr>
<td>7</td>
<td>VOP, VPS</td>
<td>-</td>
<td>450</td>
<td>240</td>
<td>430</td>
<td>+</td>
<td>APS</td>
<td>-</td>
<td>380</td>
<td>200</td>
<td>180</td>
<td>+</td>
<td>+/-/+</td>
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</tr>
<tr>
<td>8</td>
<td>VOP, VPS</td>
<td>-</td>
<td>496</td>
<td>NM</td>
<td>520</td>
<td>+</td>
<td>AOP, VOP, VPS</td>
<td>-</td>
<td>541</td>
<td>96</td>
<td>445</td>
<td>-</td>
<td>+/-/-</td>
<td>-</td>
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</tbody>
</table>

Means ± SD: 378±119 312±58 558±93 419±53 145±46 274±81 3±1

AOP = atrial overdrive pacing, APS = atrial premature stimulation, AV block = AV block during ongoing tachycardia; ATP = lowest dose of ATP to terminate the tachycardia; DP = decremental properties; ISO = isoproterenol infusion; Min S-A = minimum spike-atrial interval; Min VP = shortest ventricular pacing cycle length causing 1:1 VA conduction; NM = not measured; R/G ERP s-SP = retrograde effective refractory period of superior slow pathway; Term w/o A = termination of tachycardia by ventricular pacing without atrial capture; V-A-V (I) = V-A-V activation sequence after ventricular induction pacing; V-A-V (E) = V-A-V activation sequence after ventricular entrainment; VOP = ventricular overdrive pacing; VPS = ventricular extrastimulation; sVPS = ventricular extrastimulation after simultaneous atrial and ventricular pacing.
Table 3. Ablation of superior slow pathway and follow-up observations.

<table>
<thead>
<tr>
<th>Patient no</th>
<th>Successful site</th>
<th>Distance, mm</th>
<th>A-P, ms</th>
<th>A, mV</th>
<th>V, mV</th>
<th>A/V</th>
<th>EJC</th>
<th>Time before termination, sec</th>
<th>Induction</th>
<th>R/G s-SP</th>
<th>Follow-up, months</th>
<th>Rec</th>
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<tr>
<td>1</td>
<td>NCSV</td>
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<td>-29</td>
<td>0.21</td>
<td>0.19</td>
<td>1.1</td>
<td>+</td>
<td>1.2</td>
<td>-</td>
<td>-</td>
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<tr>
<td>2</td>
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<td>-12</td>
<td>0.54</td>
<td>0.34</td>
<td>1.6</td>
<td>+</td>
<td>1.3</td>
<td>-</td>
<td>-</td>
<td>18</td>
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<tr>
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<td>0.09</td>
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<td>-</td>
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<td>-</td>
<td>+</td>
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<td>-</td>
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<td>NM;</td>
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<td>-</td>
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<tr>
<td>7</td>
<td>Rt. PN</td>
<td>NM;</td>
<td>-10</td>
<td>0.18</td>
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<td>1.0</td>
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<td>27</td>
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</tr>
<tr>
<td>8</td>
<td>Rt. PN</td>
<td>NM;</td>
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<td>0.66</td>
<td>0.64</td>
<td>1.0</td>
<td>+</td>
<td>NM;</td>
<td>32</td>
<td>-</td>
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<tr>
<td>Means ± SD</td>
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<td>-30±18</td>
<td>0.3±0.2</td>
<td>0.2±0.2</td>
<td>1.5±0.6</td>
<td>+</td>
<td>1.5±0.7</td>
<td>38±32</td>
<td></td>
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</tbody>
</table>

A and V = amplitude of atrial and ventricular EGM; A-P = earliness of local atrial EGM relative to the onset of P wave; A/V = atrial / ventricular EGM ratio; Distance = distance measured between ablation site and His-bundle; EJC = ectopic junctional complex; NCSV = non-coronary sinus; NM = not measured; Rt. PN = right-sided perinodal region; Rec = recurrence; R/G s-SP = retrograde conduction over superior slow pathway.
Figure Legends:

Figure 1. Retrograde conduction over the superior SP followed by anterograde conduction over the fast pathway at a 350-ms S1-S2 extrastimulus from the right ventricular apex (RVA) after a simultaneous overdrive pacing train at an S1-S1 basic cycle length of 500 ms from the high right atrium (HRA) and RVA (A) and the intracardiac EGM during the tachycardia (B) in patient no 6. The dashed vertical lines marks the earliest atrial activation on the HB channel. Note that atrial activation sequence with the earliest site of HB during retrograde conduction over the superior SP is identical to that during the tachycardia. I, II, V1 and V6 = surface electrocardiogram; HBE1–2 and 9-10 = distal to proximal HB region; CS9–10 to 1–2 = proximal to distal coronary sinus (CS) recording.

Figure 2. Magnified P waves recorded in lead I, II and V1 during tachycardia (upper to lower tracings) in patients no 1 - 3, 5, 6 and 8. The P wave is negative in lead II in patient no 1 and 8, and biphasic in the other patients. A long RP tachycardia was present in all but patient no 7.

Figure 3. Induction of tachycardia with an initiating “V-A-V” (A) and “V-A-A-V” (B) activation sequence in patient no 2. A: After the first and second of triple RV apical stimulation at an S-S cycle length of 320 ms, retrograde conduction over FP is visible, with the earliest site of atrial activation in the HB region and a gradual increase in the S-atrial interval. When the third S caused a marked increase in the VA interval, retrograde conduction over the superior 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 RV apical stimulation at an S-S cycle length of 290 ms, no retrograde conduction to the atria was observed after the first S, while the first and second atrial EGM 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 EGM of 296 ms, shorter than the tachycardia cycle length of 363 ms. The other abbreviations are as in figure 1.

**Figure 4.** Termination of tachycardia at an atrial cycle length of 410 ms by RV overdrive pacing at a cycle length of 380 ms in patient no 1. A long VA interval and an earliest site of atrial activation in the HB region were observed in response to the first 2 ventricular stimuli, consistent with retrograde conduction over the superior SP. After a slight rate-dependent increase in VA conduction over the superior SP between the 1st and 2nd paced cycle, the 3rd and 4th stimuli are blocked. In response to the 5th ventricular stimulus, a short VA interval and the earliest site of retrograde atrial activation in the HB region (HBE 1-2) were observed, consistent with retrograde conduction over a FP, followed by 2:1 VA conduction over that pathway. The numbers between atrial EGM at the HRA indicate the cycle length in ms. The perpendicular dotted lines indicate the onset of the earliest atrial EGM at HBE1-2 during retrograde conduction over the superior SP and FP, respectively. The other abbreviations are as in figure 1.

**Figure 5.** Intracardiac recordings during tachycardia immediately before and after successful radiofrequency delivery (A) and fluoroscopic views showing the position of catheters (B and C) and CARTO activation maps of the right atrium and non-coronary aortic cusp (NCSV) during the tachycardia (D and E) in the right and left anterior oblique views, respectively, in patient no 4, whose successful ablation was at the NCSV. A: The atrial EGM at ABL1-2 precedes the onset of the atrial EGM at the HBE by 4 ms. The tachycardia ended 1.3 sec after the onset of radiofrequency delivery followed by no junctional rhythm. B and C: The white arrows point to the tip of the ablation catheter at the site of successful ablation; HRA = high
right atrium; CS = coronary sinus catheter; HBE = His bundle electrogram; RVA = right ventricular apical catheter. D and E: The distance between the site of successful ablation (red tag and unfilled arrows) and the nearest HB (indicated by yellow tags) measured 4.7 mm. Other abbreviations as in figure 1.

**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 non-coronary aortic cusp during the tachycardia (E and F) in the right and left oblique projections, respectively, in patient no 2, whose successful ablation was in the right-sided perinodal region. A: The atrial EGM at the distal pole of the ablation catheter (ABL1-2) precedes the onset of the P wave by 12 ms. B: The tachycardia ended 1.3 sec after the radiofrequency delivery (RF on) followed by the development of several ectopic junctional cycles (asterisks) with the earliest activation in the HB region (HBE) before returning to sinus rhythm (SR). E and F: The distance between the successful site (red tag and filled arrows) and the nearest HB (yellow tags) measured 10.7 mm. Other abbreviations as in figure 7.
Figure 1
Figure 2
Atypical Fast-Slow Atrioventricular Nodal Reentrant Tachycardia Incorporating a "Superior" Slow Pathway: A Distinct Supraventricular Tachyarrhythmia

Yoshiaki Kaneko, Shigeto Naito, Kaoru Okishige, Itsuro Morishima, Takeshi Tobiume, Tadashi Nakajima, Tadanobu Irie, Masaki Ota, Takanori Saito, Osamu Igawa, Ritsushi Kato, Kazuo Matsumoto, Fumio Suzuki and Masahiko Kurabayashi

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