Catheter Ablation of Atypical Atrioventricular Nodal Reentrant Tachycardia

Running Title: Katritsis et al.; Atypical AVNRT Ablation

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Journal Subject Terms: Arrhythmias
Abstract

Background—Due to its low prevalence, data on atypical atrioventricular nodal reentrant tachycardia (AVNRT) are scarce, and the optimal ablation method is not established. Our study aimed at assessing the efficacy and safety of conventional slow pathway ablation, as applied for typical cases, in atypical AVNRT.

Methods—We studied 2079 patients with AVNRT subjected to slow pathway ablation. In 113 patients, mean age 48.5±18.1 years, 68 female, atypical AVNRT or co-existent atypical and typical AVNRT without other concomitant arrhythmia was diagnosed. Ablation data and outcomes were compared to a group of age- and sex-matched control patients with typical AVNRT.

Results—Fluoroscopy and radiofrequency current delivery times were not different in the atypical and typical groups, 20.3±12.2 vs 20.8±12.9 min (P=0.730) and 5.9±5.0 vs 5.5±4.5 min (P=0.650), respectively. Slow pathway ablation was accomplished from the right septum in 110 patients, and from the left septum in 3 patients, in the atypical group. There was no need for additional ablation lesions at other anatomical sites, and no cases of AV block were encountered. Recurrence rates of the arrhythmia were 5.6% in the atypical (6/108 patients), and 1.8% in the typical (2/111 patients) groups, in the next 3 months following ablation (P=0.167).

Conclusions—Conventional ablation at the anatomical area of the slow pathway is the therapy of choice for symptomatic AVNRT, regardless of whether the typical or atypical form is present.

Key-words: arrhythmia; ablation; atrioventricular node; atypical, reentrant, tachycardia
Clinical Perspective

What is new?

- Atypical AVNRT, regardless of the type, can be successfully ablated by targeting the anatomic area of the slow pathway.
- When a right septal approach is not successful, the anatomic area of the slow pathway can be ablated from the left septum.
- Higher septal lesions that may increase the risk of inadvertent AV block or lesions within the coronary sinus os are not necessary for the ablation of atypical AVNRT.
- It seems that the same slow pathway participates in both typical and atypical AVNRT.

What are the clinical implications?

- Catheter ablation at the anatomical area of the slow pathway from the right or left septum is the treatment of choice for atypical AVNRT.
- This approach is not associated with an increased risk of inadvertent AV block.
- The recurrence rate following ablation of atypical AVNRT may not be significantly higher than that seen following ablation of typical AVNRT.
Introduction

Atrioventricular nodal re-entrant tachycardia (AVNRT), represents the most common regular arrhythmia encountered in clinical practice.\textsuperscript{1,2} A recent randomized trial, as well as observational studies, have provided evidence that catheter ablation is the treatment of choice for symptomatic patients, by substantially reducing hospitalization,\textsuperscript{3} improving quality of life,\textsuperscript{4-9} and reducing costs.\textsuperscript{9-11}

Approximately 6.4% of patients with AVNRT present with delayed retrograde atrial activation, and these arrhythmias are characterized as atypical variants.\textsuperscript{12} Due to its low prevalence, data on atypical AVNRT, as opposed to its typical counterpart, are scarce. Atypical AVNRT has been identified as a predictor of lower ablation success rate,\textsuperscript{13} and the optimal method of catheter ablation is not established. Although conventional slow pathway ablation has been reported safe and effective for atypical AVNRT,\textsuperscript{14,15} in most published series ablation was guided by identifying the slow pathway via consideration of retrograde atrial activation or other techniques.\textsuperscript{16-22} These approaches, however, may result in energy delivery in the mid or superior septum; \textsuperscript{18,19,21,22} i.e. sites that are potentially associated with an increased risk of inadvertent atrioventricular block.\textsuperscript{20,23}

We hypothesized that since the slow pathway appears to be a component of the AVNRT circuit regardless of typical or atypical characteristics,\textsuperscript{12,24,25} conventional slow pathway ablation as applied for typical AVNRT, should be equally safe and effective in atypical cases. We have, therefore, studied an extensive series of atypical AVNRT cases treated with anatomical slow pathway ablation, and compared them to sex and age-matched patients who underwent ablation for typical AVNRT.
Methods

Patients

Participating centers keep detailed databases of their patients subjected to catheter ablation and these databases were searched for eligible patients. Data from adult patients with symptomatic AVNRT, with at least one episode every 2 months, undergoing catheter ablation at Beth Israel Deaconess Medical Center, Boston, MA, USA (2009-2015); Rhode Island Hospital, Providence, RI, USA (1999-2001); Athens Euroclinic, Greece (2007-2015; the Heart Hospital, London, UK (2009-2014); the Johns Hopkins Hospital, Baltimore, MD, USA (2011-2014); the University of Michigan Health System, Ann Arbor, MI, USA (2009-2015); and the Brigham and Women’s Hospital, Boston, MA, USA (2008-2015), were analyzed. All patients with a diagnosis of atypical AVNRT subjected to catheter ablation were identified, and compared to age- and sex-matched control patients with typical AVNRT. Controls were matched to atypical cases at each participating center. We used an optimal matching algorithm to match each atypical AVNRT case to a typical AVNRT case of the same age and sex, at each institution. When more than one potential eligible controls were identified within an institution (same gender and age as the atypical case), we selected the typical AVNRT control with procedure date closest to the atypical AVNRT case. We did not specify a priori a matching caliper width for age, because we anticipated that exact or nearly exact matches could be identified for each atypical AVNRT case. Patients displaying characteristics of co-existent typical and atypical AVNRT were included in the study, but patients with other arrhythmias such as atrial flutter or fibrillation that required additional ablation were excluded. Patients who had been subjected to ablation for AVNRT in the past were also excluded. Electrograms and ablation characteristics of patients were studied by authors at participating institutes and, if needed, verified by the first and last author of this report.
All patients were studied in the post-absorptive state, under mild sedation, and after all antiarrhythmic agents had been discontinued for more than 5 half-lives. No patient had received amiodarone for the preceding three months. The study received approval from our institutional review boards.

Definitions

AVNRT was diagnosed by fulfillment of established criteria during detailed atrial and ventricular pacing maneuvers.\textsuperscript{26} Typical (slow-fast) AVNRT was defined by an atrial-His/His-atrial ratio (AH/HA) >1, and HA interval \( \leq 70 \) ms. Atypical AVNRT was defined by delayed retrograde atrial activation with HA>70 ms. If the AH was <200 msec and the AH<HA, the atypical form was characterized as fast-slow. If AH>200 ms and AH>HA, the atypical form was considered slow-slow. Tachycardias with a prolonged AH interval >200 ms but AH<HA, or with AH<200 ms and AH>HA, or with variable intervals during the same or different episodes, were classified as indeterminate. Details of our methodology for measurements of intervals during tachycardia have been described elsewhere.\textsuperscript{12,25}

Mapping and Ablation

Anatomical slow pathway ablation was performed at each center according to standard techniques.\textsuperscript{27-29} In brief, a conventional 4-mm ablation catheter was positioned at the inferior (posterior) part of the tricuspid annulus until an A/V ratio of <1 was recorded, and the atrial electrogram was delayed relatively to the atrial electrogram recorded at the His bundle. Care was taken to keep the ablation catheter below the ostium of the coronary sinus (CS) as visualized in the right anterior oblique (RAO) projection; mapping was not performed at the mid or anterior septum (Figure 1). When multicomponent signals or separate, low-amplitude potentials were obtained, radiofrequency (RF) current, 20-40W aimed at a temperature of 60\(^\circ\)C, was delivered.
for up to 30 sec until a junctional rhythm with 1:1 retrograde ventriculo-atrial (VA) conduction was elicited. If VA conduction was not seen, RF delivery was immediately stopped. Once junctional rhythm with VA conduction was recorded, energy delivery was continued for 10-30 sec or until cessation of the junctional rhythm. Following ablation, arrhythmia induction with the use of isoproterenol was attempted. End-points for ablation success were demonstration of RF-induced junctional rhythm conducted to the atria, and non-inducibility of AVNRT with programmed stimulation during isoproterenol challenge.

If RF-induction of junctional rhythm or non-inducibility of tachycardia could not be accomplished from the right side of the septum, mapping at the corresponding part of the left septum was undertaken. In some centers, additional lesions higher in the septum or in the roof of the proximal coronary sinus were delivered before proceeding to left septal ablation. A mapping electrode was retrogradely introduced through the noncoronary cusp of the aortic valve to record a left-sided His bundle electrogram as described elsewhere. Since positioning of the left septal catheter retrogradely through the non-coronary cusp inevitably results in mapping of the anterior part of the septum, a trans-septal approach was used for introduction of the left-sided ablation catheter and additional mapping of the posterior part of the septum (Figure 2). Mapping of the inferior (posterior) part of the mitral annulus, below the left-sided His was performed with the same principles as on the right septum. At the successful ablation site the atrial electrogram is closer to this recorded by the CS rather than the His electrode. Thus, inadvertent AV block is avoided following ablation either from the right or the left septum. Following successful ablation, patients were discharged from hospital within 24 hours on aspirin and no antiarrhythmic drugs. All patients were followed-up for an at least 3-month interval, and repeated ablation was performed in case of recurrence of symptoms during that time. Patients
with a diagnosis of AVNRT at repeat ablation, or patients who presented with a documented tachycardia similar to the clinical one before ablation, were registered as failures.

**Statistical analysis**

Continuous, normally distributed variables are presented as mean ± standard deviation. Data normality was analysed using the Kolmogorov-Smirnov test. In all cases the examined variables followed the normal distribution and the Student’s *t*-test was used to analyse differences between two groups, and the one-way ANOVA test to analyse differences between more than two groups. Categorical data are expressed as frequencies (percentages) and were compared using Fisher's exact test. All reported p-values were based on two-sided tests and were compared to a significance level of 5%. Statistical calculations were performed on SPSS for Windows version 21 (IBM Corporation, Armonk, New York). A post hoc power analysis was performed to estimate the achieved statistical power for the comparison of arrhythmia recurrence in the typical and the atypical group using G*Power version 3.1.9.2 (Heinrich-Heine-Universität Düsseldorf, Düsseldorf, Germany). Considering an alpha level of 0.05, and according to the sample size used in the study and the observed difference, the present study achieved a power of 0.25. A total sample size of 815 patients would be needed to achieve a power of 0.80 (Online appendix, Figure 1). In exploratory analysis, the critical recurrence rate in the atypical group that the study would be able to detect with a power of 0.80, was estimated to be 12.3% (Online appendix, Figure 2).
Results

Patients

In total, 2079 patients with AVNRT were studied at Beth Israel Deaconess Medical Center (Boston, MA, USA), and Rhode Island Hospital (Providence, RI, USA) (n=287); Athens Euroclinic, Greece (n=307); the Heart Hospital, London, UK (n=179); the Johns Hopkins Hospital, Baltimore, MD, USA (n=271); the University of Michigan Health System, Ann Arbor, MI, USA (n=621); and the Brigham and Women’s Hospital, Boston, MA, USA (n=414). Using the criteria mentioned above, 113 patients (5.4%) had atypical AVNRT or co-existent atypical and typical AVNRT and no other concomitant arrhythmia. The mean age of patients with atypical AVNRT was 48.5±18.1 years (range 14 to79), and 68 patients (60%) were female. The control group consisted of 113 patients with typical AVNRT, age- (48.6±17.3 years) and sex-matched to the atypical AVNRT patients, on a one-to-one basis.

Electrophysiologic characteristics

Eighty-five out of the 113 patients (75.2%) had fast-slow AVNRT according to both the AH<HA and AH<200 ms, and 14 patients (12.4%) slow-slow AVNRT. The remaining 14 patients (12.4%) could not be reliably classified due to inconsistent AH and HA/AH patterns or variable intervals. There were no significant differences in age and gender among the different atypical AVNRT forms (P = 0.592, and P = 0.323, respectively). Twenty patients (8.9%) had co-existent typical and atypical AVNRT during the electrophysiology study. Details of this group have been presented elsewhere. Atypical AVNRT was induced by ventricular pacing or extrasystoles in 27 patients (Figure 3), or spontaneously during isoprenaline infusion in 11 patients. In the remainder atypical AVNRT was induced by high right atrial (HRA) or CS pacing (Figures 4 and 5), and in 2 of them following an anterograde conduction jump ≥50 ms (Figure 6). In 7 out of 98
patients with atypical AVNRT, earliest retrograde atrial activation was recorded by the His bundle electrode. Four of these patients were classified as fast-slow, 2 as slow-slow, and one as indeterminate. Electrophysiological characteristics during tachycardia are shown in Table 1.

**Ablation characteristics**

All but 3 patients underwent conventional, right-sided ablation at the anatomical area of the slow pathway as described. This was successful without the need of additional lesions at other sites. In 3 patients a left-sided approach was successfully accomplished following unsuccessful right-sided slow pathway ablation, as well as ablation lesions at higher septal sites, and, in one of them, following unsuccessful additional lesions in the roof of the proximal coronary sinus.

Recorded electrograms by the left-sided ablation catheter at the successful site displayed the same temporal relationships as those usually recorded by a right ablation catheter (Figure 1). In one patient, both typical and atypical AVNRT was inducible following unsuccessful right septal ablation, whereas in the remainder atypical only AVNRT was induced following unsuccessful right septal ablation. Left septal ablation was needed for only one of the 7 patients in whom earliest retrograde atrial activation was recorded by the His electrode. Fluoroscopy and RF times are presented in Table 2. A junctional rhythm during RF ablation was noted in 110 (97%) and 111 (98%) patients in the atypical and typical groups, respectively (P=1.000). No cases of AV block were encountered in the atypical AVNRT group. Three patients of the typical AVNRT controls had transient AV block or PR prolongation that resolved by the next day following the procedure (P=0.247).

**Follow-up**

Three-month follow-up was complete in 108 patients of the atypical group (96%), and recurrence of symptomatic AVNRT was detected in 6 (5.6%) patients. Five of these patients had fast-slow
type, and one a slow-slow tachycardia. One patient had been subjected to both right and left septal ablation. Repeat ablation was performed in 3 patients. In two of them right septal ablation at the area of the slow pathway was unsuccessful, and a left-sided procedure, as previously described, was necessary. Three patients refused repeated ablation and were treated with medication. In the typical group, two patients were lost to follow-up, and recurrence was seen in 2 (1.8%) patients (P=0.167, for the percentages of recurrences in atypical vs typical AVNRT ablation). No case of AV block was noted during follow-up in either the typical or the atypical group.

**Discussion**

Our study represents the largest series of atypical AVNRT cases published. Results indicate that conventional slow pathway ablation is generally a safe and effective method of ablation for patients with AVNRT, regardless of the type. No cases of AV block were encountered, and success and recurrence rates were similar for typical and atypical AVNRT ablation. We did not find atypical AVNRT to be associated with a significantly higher rate of ablation failure, as previously reported,\(^5\) and no significant difference in recurrence rates was detected between the two groups, but the modest statistical power achieved in our study does not preclude the possibility of a type II error. Anatomical slow pathway ablation, as described in our study, can be accomplished without the need for additional pacing and other manoeuvres,\(^{33,34}\) and offers a safe therapeutic approach for the management of these patients. In our extensive series, detailed mapping and/or ablation attempts at the CS os or higher in the septal area were not necessary for the elimination of this arrhythmia.

These results are in keeping with our previous observations that atypical AVNRT can be induced by both atrial and ventricular extra-stimulation, and, rarely, may also be induced
following an anterograde conduction jump. The coexistence of both typical and atypical types in the same patient, as well as the fact that spontaneous conversions from one type to another may occur, also argue in favor of the concept that atypical and typical AVNRT may share a common electrophysiological substrate. Although atypical AVNRT of the fast-slow type, and typical AVNRT do not utilize the same limb for fast pathway conduction, in anatomical models the arrhythmia circuit utilizes the same “slow” component that is most likely located in the area of the posterior nodal extensions. Indeed, although there is much uncertainty about the nature of the fast pathway, the evidence supporting the inferior nodal extensions as the anatomic substrate of the slow pathway is strong. It was initially proposed that “fast-slow” AVNRT utilises a posterior extension as the “slow” pathway, while the “fast” component comprised the fast pathway of the typical form in the opposite direction, but this was refuted by subsequent evidence. It seems more likely that all subforms of atypical AVNRT utilize the right and left inferior extensions as the substrate of both components of their circuit (Figure 7). In this context, both typical and atypical AVNRT, can be ablated by targeting the area of the slow pathway either from the right or the left side. However, we have not been able to identify any electrophysiological features that may predict the need for a left septal slow pathway ablation, either for atypical or typical AVNRT. This is also the case for our patients with recurrences following an apparently successful procedure.

Study limitations

The main limitation of our study is the low statistical power achieved for comparison of recurrences in the typical and atypical AVNRT groups. Although we report the largest series of atypical AVNRT cases, due to the low recurrence rates in the typical and atypical groups, a larger sample size would be needed to establish equal effectiveness of slow pathway ablation in
typical and atypical AVNRT. However, even if there is a statistically significant difference, it is likely of small magnitude and may not be as meaningful from a clinical standpoint, especially considering the rarity of the recurrences. Second, our results have been derived by high volume EP laboratories, and very experienced operators. Thus, whether our data can be generalized to lower volume centers, is not known. Third, this is a retrospective collection of cases performed by several operators in 7 different centers that participated. Still, all successful ablations were performed by targeting the anatomic area of the slow pathway either from the right or the left septum. Finally, a 3-month only follow-up was available for all patients, but in our experience, AVNRT recurrences are very unlikely after the 3 first months post-ablation.

Conclusions

Atypical AVNRT can be safely and effectively treated by conventional slow pathway ablation. Detailed mapping during tachycardia for ablation purposes, and targeting the higher septum or the coronary sinus ostium, do not appear necessary for the ablation therapy of the vast majority of AVNRT, whether typical or atypical.

Disclosures

None
References


14. Yeh SJ, Wang CC, Wen MS, Lin FC, Chen IC, Wu D. Radiofrequency ablation therapy in


Table 1. Conduction intervals during typical and atypical AVNRT.

<table>
<thead>
<tr>
<th></th>
<th>Atypical AVNRT n=113</th>
<th>Typical AVNRT n=113</th>
<th>P</th>
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<tbody>
<tr>
<td>CL (ms)</td>
<td>367.0±91.5</td>
<td>365.9±67.5</td>
<td>0.927</td>
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<tr>
<td>AH (ms)</td>
<td>134.2±73.6</td>
<td>300.4±69.2</td>
<td>&lt; 0.0001</td>
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<tr>
<td>HA (His) (ms)</td>
<td>230.9±78.9</td>
<td>49.5±15.4</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>HA (pCS) (ms)</td>
<td>226.3±80.4</td>
<td>59.8±16.1</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>ERAA (His/CS)</td>
<td>7/91*</td>
<td>95/8**</td>
<td></td>
</tr>
</tbody>
</table>

*: no CS measurements in 15 patients, **: no CS measurements in 10 patients.
CL: tachycardia cycle length; AH: atrial to His interval during tachycardia; HA (His): His to atrium interval on the His recording electrode; HA (pCS): His to atrium interval on the proximal coronary sinus recording electrode; ERAA: earliest retrograde atrial activation.

Table 2. Ablation characteristics.

<table>
<thead>
<tr>
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<th>Atypical AVNRT n=113</th>
<th>Typical AVNRT n=113</th>
<th>P</th>
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</thead>
<tbody>
<tr>
<td>Fluoroscopy time (min)</td>
<td>20.1±12.2</td>
<td>20.8±12.9</td>
<td>0.730</td>
</tr>
<tr>
<td>RF delivery time (min)</td>
<td>5.9±5.0</td>
<td>5.5±4.5</td>
<td>0.650</td>
</tr>
<tr>
<td>Junctional rhythm during RF (pts)</td>
<td>110</td>
<td>111</td>
<td>1.000</td>
</tr>
<tr>
<td>AV block (pts)</td>
<td>0</td>
<td>3 (transient)</td>
<td>0.247</td>
</tr>
</tbody>
</table>

RF: radiofrequency.
Figure Legends

**Figure 1.** Electrograms at the site of successful ablation. Electrograms at the site of successful ablation recorded from the right (left panel) and left (right panel) septal ablation catheters. HRA: high right atrium; R His: His bundle recorded by the right septal electrode; L His: His bundle recorded by the left septal electrode; R Abl: electrograms at the successful ablation site recorded by the right septal ablation catheter; L Abl: electrograms at the successful ablation site recorded by the left septal ablation catheter; CS: coronary sinus; I, aVF, V1, V6: 12-lead ECG leads.

**Figure 2.** Slow pathway mapping. Right- and left-sided mapping of the slow pathway in the RAO and LAO projections. The left-sided ablation electrode has been introduced through a trans-septal approach in order to map the infero-posterior septum. RAO; right anterior oblique projection; LAO; left anterior oblique projection; L Abl: left septal ablation catheter; R Abl: right septal ablation catheter; His: His bundle; CS: coronary sinus, RV: right ventricle; R His: His bundle recording from the right septum; L His: His bundle recording from the left septum.

**Figure 3.** Induction of atypical AVNRT by ventricular pacing. A few of our atypical cases had induction by 1 to 2 response from the A, with first A blocking in the lower node and going up a slow pathway, then down another slow pathway (the two inferior nodal inputs?) to initiate atypical AVNRT. An alternative explanation might be simultaneous retrograde activation of both a fast and a slow pathway, with the latter initiating the tachycardia. However, this is rather unlikely considering the much prolonged VA time for activation of the second A following V
pacing compared to the VA time during tachycardia. HRA: high right atrium; His: His bundle;
CS: coronary sinus; RV: right ventricle; I, II, III, V1, V6: 12-lead ECG leads.

**Figure 4.** Induction of atypical AVNRT by atrial pacing. Induction of atypical, “slow-slow”
AVNRT by atrial pacing (same patient as in Figure 3).
Abbreviations as in Figure 3.

**Figure 5.** Induction of atypical AVNRT by atrial pacing.
Induction of the “fast-slow” form of atypical AVNRT by atrial pacing.
Abbreviations as in Figure 3.

**Figure 6.** Induction of atypical AVNRT following anterograde conduction jump. Induction of
atypical AVNRT following an anterograde conduction jump during proximal CS pacing. In the
left panel, a S1S2 of 300 ms produces an AH=240 ms. In the right panel, a S1S2 of 290 ms results
in anterograde conduction jump (AH=300 ms) and induces tachycardia. HRA: high right atrium;
His: His bundle; CS: coronary sinus; RV: right ventricle; I, II, III, V1, V6: 12-lead ECG leads.
Figure 7. Proposed circuit of AVNRT. During typical AVNRT (slow-fast), right- or left-sided circuits may occur with antegrade conduction through the inferior inputs and retrograde conduction through the superior inputs (S) or the anisotropic atrionodal transitional area (AAT). In atypical AVNRT conduction occurs anterogradely through one of the inferior inputs, left (LI) or right (RI) and retrogradely through the other one. Depending on the orientation of the circuit we may record the so-called fast-slow, slow-slow or inteterminate types.


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**SUPPLEMENTAL MATERIAL**

**Figure 1.** Statistical power as a function of study sample.

Two-tailed, proportion $p_1=0.018$, proportion $p_2=0.055$, $\alpha$ error probability=0.05, Sample size group 1=111, Sample size group 2=108, Power (1-$\beta$ error probability) =0.2525575, Actual $\alpha=0.0340042$. 
Figure 2. Critical recurrence rate in the atypical group as a function of total study sample size.

Sensitivity analysis: required effect size, two-tailed, effect direction \( p_1 \geq p_2 \), Proportion \( p_2 = 0.018 \), \( \alpha \) error probability=0.05, Power (1-\( \beta \) error probability) =0.80, Sample size group 1 =108, Sample size group 2=111, Estimated Proportion \( p_1 = 0.1233857 \), Actual power=0.8952546, Actual \( \alpha = 0.009600674 \).