Atypical atrioventricular nodal reentrant tachycardia: prevalence, electrophysiologic characteristics, and tachycardia circuit


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Aims

This study aimed at assessing the prevalence, electrophysiologic characteristics, and mechanism of atypical atrioventricular nodal reentrant tachycardia (AVNRT).

Methods and results

We studied 925 consecutive patients with AVNRT. Atrial-His (AH) and His-atrial (HA) intervals were measured during atypical AVNRT (HA > 70 ms), and compared with measurements in 34 patients with typical (slow-fast) AVNRT. Assuming that conduction velocity over the fast pathway is similar in the anterograde and retrograde directions, the AH interval during the fast–slow form should be smaller than the HA during slow–fast. Atypical AVNRT was diagnosed in 59 patients (6.4%), median age 50 years (range 19–79 years), and 37 (59.7%) of them female. Fast–slow AVNRT was diagnosed in 44 patients (74.5%), and slow–slow AVNRT in 9 patients (15.2%). The remaining six patients (10.2%) could not be reliably classified due to inconsistent AH, and HA/AH patterns or variable intervals. Tachycardia induction with anterograde conduction jumps was seen in two patients with the fast–slow, and in three patients with slow–slow or intermediate forms. Atrial-His in the fast–slow group was significantly longer than HA in the slow–fast group, 99.7 ± 40.5 ms vs. 45.8 ± 7.7 ms, P < 0.001. Tachycardia cycle length was longer in fast–slow compared with slow–fast, 379.1 ± 68.5 ms vs. 317.1 ± 42.8 ms, P < 0.001.

Conclusion

Of AVNRT cases, 6.4% are atypical and may display patterns that do not necessarily correspond to the fast–slow or slow–slow conventional types. Atypical fast–slow and typical AVNRT do not appear to utilize the same limb for fast conduction.

Keywords

Atypical atrioventricular nodal tachycardia • Fast-slow tachycardia • Slow–slow tachycardia • Nodal extensions

Introduction

Atypical atrioventricular nodal reentrant tachycardia (AVNRT) has not been extensively studied. Published reports have presented a limited number of cases, and no universal scheme for the definition and classification of forms of atypical AVNRT exists, with various criteria used by different investigators.1 The tachycardia mechanism of atypical, as well as typical, AVNRT also remains elusive.5 The longitudinally dissociated dual atrioventricular (AV) nodal pathways have not been demonstrated histologically, and the exact circuit responsible for the reentrant tachycardia is unknown. Attempts to provide a reasonable hypothesis have been made by reference to contextual considerations, such as the anisotropic conduction properties of the transitional area between the atria and the AV node.3–7 In addition, there has been electrophysiologic evidence that the right and left inferior extensions of the human AV node and the atrio-nodal inputs they facilitate, which have been identified histologically, might provide the anatomic substrate of the slow pathway.8–11 However, data indicating the potential anatomic site of the fast pathway are virtually non-existent. There has been histologic and electrophysiologic evidence of multiple superior atrial inputs to the AV node,12–16 and of variability in the space constant of tissue and poor gap junction.
What’s new?

- Atypical atrioventricular nodal reentrant tachycardia (AVNRT) may display patterns that do not necessarily correspond to the conventional fast–slow or slow–slow paradigms.
- The term ‘fast–slow AVNRT’ is ambiguous and probably should be abandoned in favour of the term ‘atypical AVNRT’ for all subtypes.
- Atypical AVNRT of the fast–slow type and typical AVNRT do not appear to utilize the same pathway for fast conduction.
- Atypical AVNRT may involve the left and right inferior nodal extensions, regardless of the atrial-His/His-atrial relationship.

Methods

Patients

Data from consecutive adult patients with AVNRT undergoing catheter ablation at four centres, Athens Euroclinic, Greece (2007–2014); Beth Israel Deaconess Medical Center, Boston, MA, USA (2009–2013); The Heart Hospital, London, UK (2009–2013); and The Johns Hopkins Hospital, Baltimore, MD, USA (2011–2014), were analysed. All patients were studied in the post-absorptive state, under mild sedation, and after all antiarrhythmic agents had been discontinued for more than 5 days. No patient had received amiodarone for the preceding three months. The study received approval from our institutional review boards.

Definitions

Atrioventricular nodal reentrant tachycardia was diagnosed by fulfillment of established criteria during detailed atrial and ventricular pacing manoeuvres, and subsequent abolition of the tachycardia by anatomic ablation of the slow-pathway. Typical (slow–fast) AVNRT was defined by an AH/His-atrial ratio > 1, and HA interval <70 ms. Atypical AVNRT was defined by delayed retrograde atrial activation with HA > 70 ms. If the AH was <200 ms 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. Retrograde atrial activation sequence may be variable in all forms of AVNRT, typical or atypical, and was not considered as a criterion for classification of AVNRT types. Similarly, the demonstration of a lower common pathway was not employed as a reliable criterion for differential diagnosis of AVNRT types.

Measurements

Atypical atrioventricular nodal reentrant tachycardia

The AH interval during tachycardia was measured from the latest rapid deflection of the atrial activation to the earliest deflection of the His bundle electrogram (Figure 1). The HA interval during tachycardia was measured from the end of the His bundle activation to the earliest rapid deflection of the atrial activation in the His bundle electrogram (Figure 1). Data are presented in Table 1.

Typical atrioventricular nodal reentrant tachycardia

For comparative purposes, patients with typical, slow-fast AVNRT, and in whom proper identification of the retrograde atrial and His bundle activation during tachycardia could be achieved, were also considered. During typical AVNRT, precise identification of earliest atrial activation may be difficult. Thus, techniques for separation of atrial and ventricular activity were employed as previously described. Identification of atrial electrograms was verified by observations on spontaneous, pharmacologic or pacing-induced termination of tachycardia as well as by producing separation of ventricular and atrial electrograms by atrial or ventricular extrastimuli. Details of our methodology have been discussed and presented elsewhere. Atrial-His and HA intervals were measured as previously described. Data on typical AVNRT represent measurements from consecutive, consenting patients studied with this methodology at Athens Euroclinic (Table 1).

All measurements were performed at a speed of 200 mm/s using an on-line automated caliper system. Measurements were analysed by two different investigators at separate time intervals to assess inter-observer reproducibility. To assess temporal reproducibility, all measurements were made during repeated episodes of tachycardia and right ventricular pacing.

Study rationale: theoretical considerations

During AVNRT, the tachycardia circuit is confined within the AV node territory, and activation of the atrium takes place following activation of the retrograde pathway. Thus, during typical, slow–fast AVNRT, the HA interval represents the time difference between activation of the His bundle and activation of the atrium, this is HA = F_{retro} + A – H, where F_{retro} is the time the impulse goes retrogradely along the fast pathway, A is the time the impulse travels from the AV node to right atrium as recorded by the electrode positioned on the His bundle, and H is the time the impulse travels from the AV node to the His bundle (Figure 2). Similarly, the AH interval represents the time difference between activation of the right atrium as recorded by the electrode positioned on the His bundle, and the next activation of the His bundle, this is AH = S_{antrum} + H – A, where S_{antrum} is the anterograde conduction along the slow pathway (probably one of the inferior nodal extensions), H is the time the impulse travels from the AV node to the His bundle, and A the time the impulse travels from the AV node to right atrium. During atypical, fast–slow AVNRT, HA = S_{antrum} + A – H, where S_{antrum} is the
time the impulse goes retrogradely along the slow pathway, \( A \) is the time the impulse travels from the AV node to the right atrium, and \( H \) is the time the impulse travels from the AV node to the His bundle. \( \text{AH} = F_{\text{ante}} + H - A \), where \( F_{\text{ante}} \) is the anterograde conduction along the ‘fast’ pathway, \( H \) is the time the impulse travels from the AV node to the His bundle, and \( A \) the time the impulse travels from the AV node to right atrium. Atrial–His and HA intervals during slow–slow tachycardia that utilizes two slow pathways (probably the two inferior nodal extensions), are also depicted in Figure 2. Assuming that conduction velocity over the fast pathway is similar in the anterograde and retrograde direction, and that \( A \geq H \) (because His bundle activation always precedes atrial activation, thus \( F + A > H \), and \( F \) by definition should be a minimum quantity), the AH interval during fast–slow tachycardia should be smaller or equal than the HA interval during slow–fast, since \( (H - A) \leq (A - H) \), if these two tachycardia types use the same ‘fast’ pathway component in their circuits. We therefore compared intervals in a series of atypical AVNRT with those derived by patients with typical, slow–fast AVNRT in whom measurements could be accomplished as described.

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**Table 1** Tachycardia types and conduction intervals

<table>
<thead>
<tr>
<th>AVNRT type</th>
<th>( n )</th>
<th>Age (-years)</th>
<th>Sex (F/M)</th>
<th>CL (ms)</th>
<th>AH (His) activation (ms)</th>
<th>HA (His) activation (ms)</th>
<th>HA (pCS) activation (ms)</th>
<th>Earliest retrograde atrial activation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fast–slow</td>
<td>44</td>
<td>50.7 ± 19.3</td>
<td>23/21</td>
<td>379.1 ± 68.5</td>
<td>99.7 ± 40.5</td>
<td>251.7 ± 76.4</td>
<td>251.5 ± 77.2</td>
<td>pCS (59%)</td>
</tr>
<tr>
<td>Slow–slow</td>
<td>9</td>
<td>45.6 ± 20.0</td>
<td>7/2</td>
<td>476.4 ± 137.9</td>
<td>286.0 ± 83.2</td>
<td>163.3 ± 60.5</td>
<td>171.0 ± 60.9</td>
<td>His (67%)</td>
</tr>
<tr>
<td>Intermediate</td>
<td>6</td>
<td>52.5 ± 13.7</td>
<td>4/2</td>
<td>395.8 ± 105.5</td>
<td>197.0 ± 31.1</td>
<td>176.0 ± 72.9</td>
<td>173.8 ± 66.7</td>
<td>His/pCS (50%)</td>
</tr>
<tr>
<td>Slow–fast</td>
<td>34</td>
<td>39.0 ± 7.5</td>
<td>22/12</td>
<td>331.1 ± 46.8</td>
<td>270.4 ± 44.3</td>
<td>45.8 ± 7.7</td>
<td>51.4 ± 7.5</td>
<td>His (85%)</td>
</tr>
</tbody>
</table>

Fast–slow type: \( \text{AH} < 200 \text{ ms and AH} < \text{HA} \); slow–slow type: \( \text{AH} > 200 \text{ ms and AH} > \text{HA} \); intermediate: all other patterns. CL, tachycardia cycle length; AH tachy, atrial to His interval during tachycardia; HA tachy, His to right atrium interval during tachycardia.
Statistical analysis

Data normality was analysed using the Kolmogorov–Smirnov test. In all cases the examined variables followed the normal distribution and Student’s t-test was used to analyse difference between two groups and the one-way analysis of variance test to analyse differences between more than two groups. All reported P values were based on two-sided tests and were compared with a significant level of 5%. All statistical calculations were performed on SPSS for Windows version 13.0 (SPSS, Inc.).

Results

Prevalence

In total, 925 consecutive patients with AVNRT were studied in Athens Euroclinic, Greece (n = 287), Beth Israel Deaconess Medical Center, Boston, MA, USA (n = 188), the Heart Hospital, London, UK (n = 179), and the Johns Hopkins Hospital, Baltimore, MD, USA (n = 271). Using the criteria mentioned above, 59 patients (6.4%) had atypical AVNRT. Of these, 44 patients (74.5%) had fast–slow AVNRT according to both the AH < HA and AH < 200 ms, and 9 patients (15.2%) had slow–slow AVNRT. The remaining six patients (10.2%) could not be reliably classified due to inconsistent AH and HA/AH patterns or variable intervals. Patient characteristics and conduction intervals during tachycardia are shown in Table 1. Median age of all patients was 50 years (range 19–79 years), and 37 patients (59.7%) were female. Patient age and tachycardia cycle lengths in the atypical AVNRT group were significantly higher than in the slow–fast group. 50.1 ± 18.7 vs. 39.0 ± 7.5 years (P = 0.002), and 395.7 ± 91.0 vs. 331.1 ± 46.8 ms (P < 0.001), respectively. There was no significant difference in age between atypical AVNRT groups (P = 0.721).

Mode of induction and earliest atrial retrograde activation

Tachycardia induction during atrial pacing with antegrade conduction jumps was seen in two patients with the fast–slow form, and in three patients with slow–slow or intermediate forms and variable AH/HA intervals (Figure 3). Variable AH/HA intervals were seen in six patients (Figure 4, left panel). During tachycardia, induction by ventricular pacing discrete retrograde jumps were difficult to define, mainly due to inability to record reliably a retrograde His bundle electrogram. Seven patients (11.9%) had also typical AVNRT induced, either spontaneously or following autonomic manipulation with atropine or isoproterenol (Figure 4, right panel, and Figures 5 and 6). One patient had transient 2 : 1 infrahisian block during tachycardia. Earliest atrial retrograde activation was variable, but most often recorded at the proximal coronary sinus electrogram (57%), especially in the fast–slow form (Table 1).

Conduction intervals

The AH interval in the fast–slow group was significantly longer than HA in the slow–fast group, 99.7 ± 40.5 vs. 45.8 ± 7.7 ms, P < 0.001, and tachycardia cycle length was also longer (379.1 ± 68.5 vs. 331.1 ± 46.8, P < 0.001), respectively. The HA interval during fast–slow was not different that the AH during slow–fast (251.7 ± 76.4 vs. 270.4 ± 44.3, P = 0.2), respectively. In Figure 4 (right panel), the HA during slow–fast (55 ms) is completely different from both the AH and HA during atypical AVNRT. The AH in slow–slow is shorter than the AH during slow–fast (260 vs. 340 ms), with the difference probably due to shorter tachycardia cycle length as well as fast retrograde activation that results in delayed conduction.
in the slow pathway or the AV nodal–His interval. In Figure 5 the HA during slow–fast (left panel) is shorter (45 ms) than the AH during fast–slow (67 ms, right panel).

**Discussion**

There are four main findings of our study. First, the prevalence of atypical AVNRT is lower than previously reported. Second, atypical AVNRT may display patterns that do not necessarily correspond to the fast–slow or slow–slow conventional types. Third, earliest retrograde activation during atypical AVNRT is variable and not always eccentric. Fourth, atypical AVNRT of the fast–slow type, and typical AVNRT most probably do not utilize the same limb for fast pathway conduction.

In our series, AVNRT with prolonged HA intervals was identified in \(\approx 6\%\) of all AVNRT cases. We believe that the reason for a higher prevalence reported by other groups is the characterization as slow–slow atypical AVNRT what are actually typical AVNRT cases, mainly due to eccentric retrograde activation or identification of a lower
**Figure 5** Induction of typical AVNRT by atrial pacing (**left panel**), and atypical AVNRT by ventricular pacing (**right panel**) in the same patient.

**Figure 6** (A) Typical AVNRT with 2:1 supraventricular conduction block. Following ventricular ectopic beat (arrow), there is restoration of 1:1 conduction. The tachycardia cycle length is stable and the same (262 ms) in both atrial and ventricular electrograms. (B) Same patient. Dissociation of atria (atrial fibrillation) and ventricles during AVNRT (stable His and V cycle length of 294 ms). (C) Same patient. Spontaneous initiation of atypical AVNRT.
common pathway.¹ Our results demonstrate that the electrophysio-
logic characteristics of the ‘fast’ component of the tachycardia circuit
in the fast→slow form are not similar to those of the fast conduction
pathway during typical, slow→fast AVNRT. Our measurements are in
keeping with data provided by other investigators who have reported
on atypical AVNRT. Atrial-His and HA intervals during fast→slow
tachycardia were 135±32 and 250±89 ms,²¹ 107±29 ms and
222±67 ms,²³ 109±31 ms and 261±69 ms,²⁴ 77±21 ms and
321±61, 112±8 ms and 230±30 ms, 120±38 ms and 282±
120 ms,²⁵ respectively. For typical AVNRT, reported AH and HA
intervals during tachycardia were 364±71 and 45±15 ms, re-
spectively,²¹ although reliable identification of the atrial electrogram
without specific maneuvers as described, may not be possible in
typical AVNRT where atrial and ventricular activation may be sim-
ultaneous. Induction of fast→slow AVNRT with an AV conduction jump
indicates involvement of a ‘slow’ pathway in anterograde conduction,
and measured intervals suggest that ‘fast’ conduction properties
during fast→slow tachycardia resemble those displayed during retro-
grade conduction of slow→slow AVNRT. It is therefore probable that
re-entry may involve the left and right inferior extensions in all forms
of atypical AVNRT, including the fast-slow variety, as proposed by
Heidbuchel and Jackman²² and Lockwood et al.²² Depending on
the relative lengths and conduction properties of the left and right
inferior extensions, AH and HA relationship may vary, and this
explains the indeterminate forms that cannot be classified according
to conventional criteria, as well as the disagreement about classifica-
tion and definitions of forms of atypical AVNRT. For example,
fast→slow AVNRT has been defined by means of an AH interval
<185 ms²² or AH < 200 ms,²³ or by means of the AH/HA pattern
only,²³–²⁵ by different investigators.

Different HA times during slow→fast and fast→slow AVNRT could
be explained by different H intervals in the two types of tachycardia.
A long H interval during slow→fast AVNRT (Figure 2) could explain
the short HA and long AH intervals, whereas a short H interval
during fast→slow AVNRT could explain the long HA and relatively
short AH interval. However, the demonstration of both types of
tachycardia in the same patient, argues against such an explanation.

Data on ventricular pacing at the tachycardia cycle length and the
derived ‘lower common pathway’ were not used in our study. During
ventricular pacing, the HA time is HA = H_{retro} + AVN + A, where
H_{retro} is the time the impulse travels retrogradely from the His
bundle to the AV node, AVN is conduction through the compact
AV node and either of the AV nodal pathways, and A the time the
impulse travels from the exit of the AV node to right atrium. Thus,
it is fundamentally different that the HA time with which it is com-
pared with derive the lower common pathway. Conduction during
ventricular pacing may also not be relevant to activation sequence,
and intervals, during AVNRT. We have previously shown that the
breakthrough of atrial activation is discordant from that observed
during ventricular pacing in up to 43% of patients with AVNRT.⁸
Similarly, the difficulty in using earliest atrial activation sites to guide
classification of AVNRT is well documented,¹¹ and the pattern
of earliest activation was not considered to be of diagnostic significance
in our study.

In Figure 7 we propose a hypothetical model of the AVNRT tachy-
cardia circuits based on the concept of nodal extensions. Both
described superior atrial inputs to the AV node and the anisotropic,
transitional atrionodal area could serve as the fast limb of typical
AVNRT. In atypical AVNRT, re-entry involves the inferior nodal
extensions and this is probably why this tachycardia displays different
electrophysiologic behaviour than typical AVNRT. The AH/HA relation-
ship depends on the length and conduction properties of the
right and left (usually shorter) extensions. The AH interval also
may be variable and any threshold used for characterization of the
arrhythmia type is arbitrary.

Clinical implications
Atrioventricular nodal reentrant tachycardia with prolonged retro-
grade atrial activation (HA > 70 ms) represents an atypical form re-
gardless of the site of earliest retrograde atrial activation. Further
classification into fast→slow or slow→slow types has no validity.
Attempts at establishing the presence of a lower common pathway
are also of no practical significance, and may result in unnecessarily
prolonged procedures. Since atypical AVNRT most likely utilizes
the two inferior nodal extensions for its circuit, ablation should be
directed only towards the anatomic position of the slow pathway. If
right septal attempts are unsuccessful, the left septal side should be
tried.²⁸ Ablation targeting earliest atrial activation sites during
atypical AVNRT or the fast pathway, is not justified.

Study limitations
Our study has several limitations. The theoretical models of the three
types of AVNRT accept similar A and H times, an A interval longer
than H, and similar anterograde and retrograde conduction velocities
of the fast and slow AV nodal pathways, but these assumptions cannot
be deduced from our data. Retrograde atrial activation, in particular,
may not take similar paths in all forms of AVNRT, thus disputing the
significance of our calculations. However, induction via a jump, and consistently longer tachycardia cycles in fast→slow, do suggest a different circuit of this tachycardia, rather than an ‘inverse’ slow→fast form.

Conclusions
Acknowledging these limitations, we conclude that atypical AVNRT may display patterns that do not necessarily correspond to the conventional fast→slow or slow→fast paradigms. Atypical AVNRT of the fast→slow type and typical AVNRT do not utilize the same pathway for fast conduction. The term ‘fast→slow AVNRT’ is ambiguous and probably should be abandoned in favour of the term ‘atypical AVNRT’ for all subtypes. Atypical AVNRT probably may involve the left and right inferior nodal extensions, regardless of the AH/HA relationship.

Conflict of interest: none declared.

References