Electrophysiologic Features Differentiating the Atypical AV Node-Dependent Long RP Supraventricular Tachycardias

Running title: Ho et al.; Electrophysiology of Long RP Tachycardias

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Abstract:

**Background** - Diagnosing atypical AVN – dependent long RP SVTs can be challenging.

**Methods and Results** - Nineteen patients with 20 SVTs (atypical AVNRT without (n=11)/ with (n=3) a bystander nodo-fascicular (NF) accessory pathway (AP), orthodromic reciprocating tachycardia (ORT) using a decremental atrio-ventricular (PJRT, n=4) or nodo-fascicular (NFRT, n=2)) AP underwent electrophysiologic study. Post-pacing interval (PPI) – tachycardia cycle length (TCL), corrected PPI (cPPI), ΔVA, ΔHA, ΔAH values and responses to His-refractory VPDs were studied. Compared to AVNRT, ORT patients were younger (42 ± 13yrs vs. 54 ± 19yrs, p=0.036) and female (5/6 (83%) vs. 3/14 (21%), p=0.036); TCLs were similar (435ms vs. 429ms, CI=- 47.5 – 35.5). PPI – TCL was shorter for ORT (118ms vs. 176ms, CI= 26.3 – 89.7) but only 50% had PPI – TCL < 115ms while 5/6 (83%) had PPI – TCL < 125ms (sensitivity: 83%, specificity: 100%). Corrected PPI < 110ms, ΔVA < 85ms, and ΔHA < 0ms had equivalent sensitivity (67%) and 100% specificity for ORT. Compared to PJRT, NFRT/AVNRT had longer ΔAH (29ms vs. 10ms, CI= 3.03 – 35.0) or AH(SVT) < AH(NSR). His-refractory VPDs advanced (4/8 (50%), delayed (4/8 (50%)), or terminated (5/8 (63%)) SVT in all AP patients.

**Conclusions** - This unusual SVT requires separate maneuvers to delineate its upper and lower circuit. Standard entrainment criteria are moderately sensitive but highly specific for ORT; and PPI – TCL of 125ms appears better than 115ms. The ΔAH criteria or paradoxically, AH(SVT) < AH(NSR), differentiates NFRT/AVNRT from PJRT. Bystander APs were only identified by His-refractory VPDs.

**Key words:** supraventricular tachycardia, catheter ablation, atrioventricular node, accessory pathway, nodo-fascicular, nodo-ventricular, entrainment, resetting
Introduction

Establishing the diagnosis of an atypical AV node-dependent long RP supraventricular tachycardia (SVT) can be difficult. Standard diagnostic criteria are lacking and often extrapolated from pacing maneuvers applied to the more common short RP SVT. Long RP SVTs involving concealed nodo-fascicular (NF) accessory pathways (AP) are particularly rare with descriptions limited to isolated case reports. Prolonged conduction over the slow pathway (SP) of the AV node or a decremental AP following entrainment from the ventricle can produce A-A-V patterns that might be mistaken for atrial tachycardia (AT). Additionally, slow AP conduction following entrainment of an atypical orthodromic reciprocating tachycardia (ORT) can generate long post-pacing intervals (PPI) that cause misdiagnosis of atrio-ventricular nodal reentrant tachycardia (AVNRT) despite correction for delay in the AV node (cPPI).

This study sought to evaluate the electrophysiologic features and criteria differentiating the four atypical AV node–dependent long RP SVTs: atypical AVNRT, atypical AVNRT with a concealed, bystander NF AP (atypical AVNRT/NF AP), ORT using a concealed, slowly-conducting, decremental atrio-ventricular (AV) AP (also called the permanent form of junctional reciprocating tachycardia or PJRT), and ORT using a concealed NF AP (also called nodo-fascicular reentrant tachycardia or NFRT).

Methods

Nineteen patients with 20 symptomatic atypical long RP SVTs underwent diagnostic electrophysiologic study. After informed consent, femoral vein access was achieved percutaneously and multipolar catheters were positioned in the high right atrium, His bundle region, right ventricle and coronary sinus. Programmed stimulation and burst pacing were
delivered from the ventricle and atrium to evaluate retrograde and antegrade conduction, respectively and induce SVT. If SVT was non-inducible in the baseline state, isoproterenol was infused and the stimulation protocol was repeated. After tachycardia induction, scanning ventricular premature depolarizations (VPDs) were delivered during the diastolic period of tachycardia and SVT was entrained from the ventricle at a pacing cycle length (PCL) 10-50ms shorter than tachycardia cycle length (TCL). Para-Hisian pacing and/or entrainment was performed selectively to confirm diagnoses. Radiofrequency ablation was performed in all patients by targeting either the SP or AP after activation mapping or SP using the standard approach during sinus rhythm.

**Definition of terms:**

Post-pacing interval (PPI) - time between the pacing stimulus to the 1st return RV electrogram following entrainment of tachycardia from the ventricle

\[ \text{Corrected PPI (cPPI)} = (\text{PPI} - \text{TCL}) - (\text{AH}_{(1\text{st return AH following entrainment from ventricle})} - \text{AH}_{(SVT)}) \]

\[ \Delta VA = \text{VA}_{(entainment from ventricle)} - \text{VA}_{(SVT)} \]

\[ \Delta HA = \text{HA}_{(entainment from ventricle)} - \text{HA}_{(SVT)} \]

\[ \Delta AH = \text{AH}_{(atrial pacing/entrainment at/near TCL)} - \text{AH}_{(SVT)} \]

or

\[ \text{AH}_{(NSR)} - \text{AH}_{(SVT)} \] if paradoxically, \( \text{AH}_{(SVT)} < \text{AH}_{(NSR)} \)

**Diagnostic criteria for SVT:**

All tachycardias had a long RP (RP > PR) interval with earliest atrial activation near the ostium of the coronary sinus. Atrial tachycardia was excluded by the following criteria: 1) spontaneous termination with atrio-ventricular (AV) block, 2) termination of tachycardia by VPDs that failed to reach the atrium (VA block), 3) A-A-V response to entrainment from the ventricle. The following criteria established a diagnosis of:
Atypical AVNRT: 1) non-obligatory 1:1 AV relationship (persistence of tachycardia despite retrograde block to the atrium or antegrade block to the ventricle), 2) failure of bundle branch block (BBB) to affect tachycardia, 3) failure of His-refractory VPDs to affect tachycardia, 4) inability to entrain tachycardia from the ventricle with orthodromic capture of the His bundle, 5) PPI – TCL ≥ 115ms (or cPPI ≥ 110ms), 6) ΔVA ≥ 85ms, 7) ΔHA > 0ms, 8) ΔAH > 40ms (or paradoxically, AH(SVT) < AH(NSR)).

PJRT: 1) obligatory 1:1 AV relationship , 2) VA/TCL prolongation with development of BBB, 3) His-refractory VPDs reset (advance or delay) the atrium or terminate tachycardia with VA block, 4) ability to entrain tachycardia from the ventricle with orthodromic capture of the His bundle, 5) PPI – TCL < 115ms (or cPPI < 110ms), 6) ΔVA < 85ms, 7) ΔHA < 0ms, 8) ΔAH < 20ms.

NFRT: 1) non-obligatory 1:1 AV relationship (persistence of tachycardia with retrograde block to the atrium but not antegrade block to the ventricle), 2) VA/TCL prolongation with development of BBB, 3) His-refractory VPDs reset or terminate tachycardia with VA block, 4) ability to entrain tachycardia from the ventricle with orthodromic capture of the His bundle, 5) PPI – TCL < 115ms (or cPPI < 110ms), 6) ΔVA < 85ms, 7) ΔHA < 0ms, 8) ΔAH > 40ms (or paradoxically, AH(SVT) < AH(NSR)).

Atypical AVNRT/NF AP: 1) non-obligatory 1:1 AV relationship (persistence of tachycardia despite retrograde block to the atrium or antegrade block to the ventricle), 2) failure of BBB to affect tachycardia, 3) His-refractory VPDs reset the atrium or terminate tachycardia with VA block, 4) ability to entrain tachycardia from the ventricle with orthodromic capture of the His bundle, 5) PPI – TCL ≥ 115ms (or cPPI ≥ 110ms), 6) ΔVA ≥ 85ms, 7) ΔHA > 0ms, 8) ΔAH > 40ms (or paradoxically, AH(SVT) < AH(NSR)).
Statistics

Continuous data are expressed as mean ± SD or 95% CI. Categorical data are presented as frequency and percentage. The Student t test and comparison of proportions were used to compare differences between groups. P values ≤ 0.05 were considered significant.

Results

The 20 atypical long RP SVTs in 19 patients included pure atypical AVNRT (n = 11), atypical AVNRT/NF AP (n = 3), PJRT (n = 4), and NFRT (n = 2). One patient had both NFRT and atypical AVNRT/NF AP. All APs were concealed and none demonstrated antegrade conduction. Illustrative cases shown are shown in Figures 1-4. Compared to atypical AVNRT, patients with ORT (PJRT/NFRT) were younger, more often female but TCLs were similar (Table 1).

Entrainment from ventricle

A-A-V patterns were common and occurred more frequently with atypical AVNRT than ORT (79% vs. 17%, p = 0.036). They were pseudo A-A-V patterns in 9/12 (atypical AVNRT (n = 8), PJRT (n = 1)) and true A-A-V responses in the remaining 3 (atypical AVNRT with (n = 2) and without (n = 1) NF AP). Although the PPI – TCL was shorter for ORT (118 ms vs. 176 ms, CI = 26.3 – 89.7), half had a PPI – TCL ≥ 115 ms (sensitivity (SN): 50%, specificity (SP): 100%, positive predictive value (PPV): 100%). In contrast, PPI – TCL < 125 ms occurred in 5/6 ORT and 0/14 AVNRT (SN: 83%, SP: 100%, PPV: 100%) (Figure 5). The cPPI was also shorter for ORT (115 ms vs. 170 ms, CI = 21.7 – 88.3), but 2/6 ORT had a value ≥ 110 ms, both associated with antidromic capture of the His bundle (SN: 67%, SP: 100%). The ΔVA was smaller for ORT (101 ms vs. 160 ms, CI = 25.9 – 42.2) but 2/6 ORT had a value ≥ 85 ms (SN: 67%, SP: 100%). His bundle electrograms were identifiable during entrainment in 17/20 (85%) SVTs and
were captured orthodromically in ORT (n = 4) and antidromically in 13 (AVNRT (n = 11), ORT (n = 2)). The ΔHA was smaller for ORT (-1ms vs. 72ms, CI = 35.6 – 110) but 2/6 ORT had a value > 0ms (SN: 67%, SP: 100%).

**His-refractory VPDs**

His-refractory VPDs reset or terminated tachycardia in all 8 patients with an AP; and was the only maneuver to identify a concealed, bystander NF AP during atypical AVNRT. They advanced the atrium in 4 APs (AV AP (n = 3), NF AP (n = 1)), delayed it in 4 (NF AP (n = 3), AV AP (n = 1)), and terminated SVT with VA block in 5 (NF AP (n = 3), AV AP (n = 2)). All 4 APs exhibiting paradoxical delay was associated with PPI – TCL > 125ms.

**Other criteria**

Compared to PJRT, the ΔAH was longer for NFRT/atypical AVNRT (29ms vs. 10ms, CI = 3.03 – 35.0); and 3 SVTs (atypical AVNRT/NF AP (n = 2), NFRT (n = 1)) had an AH interval paradoxically shorter than that during sinus rhythm. Para-Hisian pacing was unhelpful in 14/19 (74%) patients because either FP conduction always preempted slower SP/AP conduction during pacing (n = 12) or consistent 1:1 conduction over the SP/AP could not be achieved despite pacing at the slowest rate allowable by sinus rhythm (n = 2). Para-Hisian entrainment was successfully performed in only 2 patients and confirmed the established diagnosis.

**Nodal pathways**

The proximal insertion of all four nodal APs was the SP of the AV node. In one patient, it was the left atrio-nodal extension of the SP requiring ablation along the posteroseptal mitral annulus. The distal insertion of the nodal APs was fascicular (para-Hisian pacing (n = 1), para-Hisian entrainment (n = 1), Figure 4)), ventricular (manifest QRS fusion during entrainment (n = 1), Figure 2), and indeterminate (n = 1).
Ablation

The successful ablation site for all patients with atypical AVNRT with and without a bystander NF AP was the SP of the AV node along the postero-septum of the right atrium and includes the patient with both atypical AVNRT/NF AP and NFRT. The other patient with NFRT had an AP inserting into the left atrio-nodal extension of the SP and required ablation along the postero-septal mitral annulus. All patients with PJRT had successful AP ablation along the postero-septum of the tricuspid annulus near the ostium of the coronary sinus identified by activation mapping during tachycardia.

Discussion

Compared to atypical AVNRT, patients with ORT (NFRT/ PJRT) were younger and predominantly female although the demographics may be skewed by the small study population. These long RP tachycardias respond differently than their short RP counterparts to pacing maneuvers; and the rare tachycardia associated with a NF AP can be misdiagnosed as PJRT if the upper circuit is not analyzed. Therefore, separate pacing maneuvers in the atrium and ventricle are required to delineate the upper and lower circuit, particularly when a 1:1 AV relationship exists (Figure 6, Table 2).

Entrainment from ventricle

While A-A-V responses are generally considered diagnostic of AT, A-A-V patterns were common in our series, particularly for atypical AVNRT with its longer paced VA interval. Pseudo A-A-V patterns occur when decremental conduction over the SP or AP produced long VA intervals that exceed the PCL so that the 1st atrial electrogram after entrainment is actually driven by the penultimate pacing stimulus. True A-A-V responses were the result of dual
retrograde responses ("double fire") with simultaneous conduction over the FP and NF AP or SP occurring only with atypical AVNRT with and without a concealed, bystander NF AP, respectively. This is different from the A-A-V response of AT which results from retrograde conduction over the AV node followed by the 1st return beat of AT after pacing. A mechanism to explain dual retrograde ("A-A-V") responses during atypical AVNRT is the presence of a large excitable gap with collision between antidromic and orthodromic wavefronts in the SP (retrograde limb) of the circuit. The last (n) paced antidromic wavefront conducts completely over the FP to the atrium (1st A) and then collides with the previous (n -1) orthodromic wavefront in the SP. The last (n) paced orthodromic wavefront has no antidromic wavefront with which to collide, conducts over the SP to activate the atrium (2nd A) before conducting antegradeley over the FP to the ventricle. With the more common single retrograde ("A-V-A") responses, the collision point between antidromic and orthodromic wavefronts is in the FP (antegrade limb). An alternative mechanism is tachycardia termination and subsequent re-initiation. With onset of ventricular pacing, retrograde block occurs in the SP effectively terminating tachycardia and conducts exclusively over the FP. When pacing stops, retrograde conduction occurs over both the FP and SP, the latter re-initiating tachycardia.

Conventional SVT criteria during entrainment from the ventricle establish the lower portion of the tachycardia circuit as macroreentrant involving the His-Purkinje system/ventricle (PJRT/NFRT) or not (AVNRT). A PPI – TCL < 115 was specific for ORT but conduction delay over the AP caused PPI – TCL > 115ms in half ORT yielding misdiagnosis of AVNRT as has been observed in other series. A higher cut-off value of 125ms increased the sensitivity for ORT by 33% while maintaining 100% specificity. The cPPI correctly adjusted the long PPI during ORT when both retrograde AP and antegrade AV node decrement occurred – the latter
from antidromic capture of the His bundle and retrograde concealment into the AV node. However, when substantial delay occurred over the AP, the cPPI could not correct the long PPI and even paradoxically prolonged it because the 1st return AH became shorter than during SVT. Slow, decremental AP conduction also affected the sensitivity of the ∆VA and ∆HA criteria for ORT but maintained their high specificity. Therefore, any standard criteria positive for ORT (PPI – TCL < 115ms, cPPI < 110ms, ∆VA < 85ms, ∆HA < 0ms) was diagnostic of ORT despite discordance among each other which occurred 50% of the time.

**His-refractory VPDs**

His-refractory VPDs that reset (advance or delay) or terminate tachycardia indicate the presence of an AP but not necessarily its participation in tachycardia. They can reset or terminate atypical AVNRT in the presence of a concealed, bystander NF AP inserting into the retrograde SP.\(^1,3\) In such a case, the VPD conducts over the NF AP ahead of the AVNRT wavefront and penetrates its excitable gap in the SP after the lower turnaround point of the circuit. Its antidromic wavefront collides with tachycardia while its orthodromic wavefront encounters either relative or absolute distal SP refractoriness delaying or terminating tachycardia, respectively. His-refractory VPDs identified an AP in all patients with ORT and was the only pacing maneuver to diagnose a concealed, bystander NF AP in 3 patients with atypical AVNRT by delaying the atrium and/or terminating tachycardia with VA block. While entrainment of atypical AVNRT/NF AP from the ventricle with orthodromic capture of the His bundle is theoretically possible, it was not observed. His-refractory VPDs also determined the degree of decremental conduction over each AP. Severe AP decrement paradoxically delayed the atrium because the degree of VPD prematurity was offset by ≥ degree of AP conduction delay (fully compensatory).\(^18\) Mild AP decrement advanced the atrium because the degree of VPD
prematurity was offset by < degree of AP conduction delay (partially compensatory).
Paradoxically delay might identify patients who have long PPIs after entrainment independent of tachycardia mechanism.

Other criteria
The ΔAH criteria differentiates tachycardia circuits whose upper portion is partially extranodal (PJRT) or completely intranodal (NFRT/atypical AVNRT). During PJRT, the AH interval is a true interval reflecting sequential activation of the atrium and His bundle over the AV node and similar to the AH interval when pacing at the TCL. In contrast, during NFRT and atypical AVNRT, the AH interval is a pseudo-interval reflecting simultaneous activation of the atrium and His bundle and is, therefore, shorter than the AH interval when pacing at the TCL. The ΔAH was longer for the nodal tachycardias (NFRT/atypical AVNRT) compared to PJRT and the AH interval was paradoxically shorter for atypical AVNRT/NF AP (n = 2) and NFRT (n = 1) than during sinus rhythm. A major limitation of ΔAH criteria, however, is the sensitivity of the AV node to rapid fluctuations in autonomic tone so that comparison of AH intervals between tachycardia and pacing should be done close in time allowing for minimal change in the autonomic state of the patient. For atypical AV node – dependent long RP tachycardias, para-Hisian pacing was generally not useful because 1) retrograde FP conduction consistently preempted SP/AP conduction, 2) SP/AP often exhibited retrograde Wenckebach conduction despite ventricular pacing at the slowest cycle length allowable by the sinus rate and 3) an “AV nodal” response is not diagnostic of pure AV nodal conduction but can also be observed with a nodo-fascicular AP.

Nodal pathways
The proximal insertion of all four nodal APs was determined to be the SP of the AV node by the
ability of His-refractory VPDs to perturb the retrograde limb of the circuit during atypical AVNRT and NFRT. A SP insertion can also be identified by the ability of His-refractory VPDs to reset or terminate typical AVNRT in the antegrade limb. Various maneuvers can determine the distal insertion site of concealed nodal APs. An “accessory pathway” response to para-Hisian pacing/entrainment identifies a nodo-ventricular AP because retrograde conduction is dependent upon myocardial capture. An “AV nodal” response indicates a nodo-fascicular AP because retrograde conduction is dependent upon His-RB capture. Limited data suggest that manifest fusion during RV entrainment of ORT using a nodal AP is specific to a nodo-ventricular fiber. Because the circuit for NFRT is contained within the specialized conduction system, ventricular fusion cannot occur with paced complexes that penetrate the excitable gap and entrain tachycardia (analogous to AVNRT). While this is true when collision between antidromic and orthodromic wavefronts occurs in the AV node or His bundle, it is not when the collision point is in the right bundle distal to the bifurcation of the His bundle and proximal to the take-off of the nodo-fascicular AP. In this case, the His bundle-left bundle-ventricular axis is orthodromically activated and can fuse with paced complexes from the right ventricle.

Limitations
The number of patients in our collection is relatively small and our data should be evaluated in more patients. Furthermore, one patient contributed two SVTs which violates the requirement for independent observations. However, it is to our knowledge the only series comparing both bystander NF tachycardias and NFRT providing useful information about these rarely-described tachycardias. Accurate diagnosis requires evaluating all available clues from the EP study (e.g. effect of BBB) as differentiating NFRT from atypical AVNRT/ NF AP using entrainment alone can be difficult and potentially misleading in certain situations. If the refractory period of a
bystander NF AP is sufficiently short to support 1:1 conduction during entrainment and conduction over the NF AP is faster than over the His-Purkinje system, the pathway for entrainment of atypical AVNRT/NF AP and NFRT are the same and the PPI can be short. Conversely, severe decremental conduction over a NF AP might generate long PPIs during entrainment of NFRT that resemble atypical AVNRT. Entrainment results therefore, should be corroborated with the other important findings of the study. Entrainment is not possible for patients with only non-sustained tachycardia or whose tachycardia repeatedly terminates with pacing. In such situations, evaluating the response of tachycardia at the beginning of ventricular overdrive pacing can help differentiate ORT from AVNRT but does not distinguish NFRT from PJRT or AVNRT/NF AP from ORT.\textsuperscript{21,22} Atrial extrastimulation and overdrive pacing were not systematically performed during tachycardia to exclude junctional tachycardia (JT) or assess VA linking.\textsuperscript{23-25} However, because focal JT associated with retrograde conduction over the SP is extremely rare and none of our tachycardias exhibited nonreentrant behavior (e.g. warm-up/cool-down phenomena, initiation after a spontaneous junctional complex), we are confident of our diagnoses. Additionally, the value of VA linking in long RP tachycardias is unclear because VA intervals can vary significantly during atypical ORT and AVNRT due to decremental conduction over the AP and SP, respectively. Rather, AT was excluded by classical electrophysiologic criteria.

**Conclusion**

Diagnosing the atypical AV node – dependent long RP SVT requires separate pacing maneuvers to delineate the upper and lower limbs of the circuit. Long PPIs are common and a PPI – TCL < 125ms appears better than 115ms for differentiating ORT (PJRT/NFRT) from atypical AVNRT.
Other entrainment criteria (cPPI < 110ms, ΔVA < 85ms, ΔHA < 0ms) are only modestly sensitive but 100% specific for ORT. Differentiating nodal tachycardias (NFRT/atypical AVNRT) from PJRT can be established by ΔAH criteria or the paradoxical finding of AH(SVT) < AH(NSR). His-refractory VPDs was the only maneuver to identify a bystander, concealed NF AP during atypical AVNRT.

**Conflict of Interest Disclosures:** None

**References:**


Table 1. Electrophysiologic criteria differentiating the four atypical AV node–dependent long RP SVTs

<table>
<thead>
<tr>
<th></th>
<th>Atypical AVNRT (w/wo NF AP) (n = 14)</th>
<th>PJRT/ NFRT (n = 6)</th>
<th>p value /95% CI</th>
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</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>54 ± 19</td>
<td>42 ± 13</td>
<td>p= 0.036</td>
</tr>
<tr>
<td>Female</td>
<td>3/14 (21%)</td>
<td>5/6 (83%)</td>
<td>p= 0.036</td>
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<tr>
<td>TCL</td>
<td>429ms</td>
<td>435ms</td>
<td>Cl= - 47.5 – 35.5</td>
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<tr>
<td>A-A-V pattern</td>
<td>11/14 (79%)</td>
<td>1/6 (17%)</td>
<td>p= 0.036</td>
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<td>PPI – TCL</td>
<td>176ms</td>
<td>118ms</td>
<td>Cl= 26.3 – 89.7</td>
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<td>PPI – TCL &lt; 115</td>
<td>0/14 (0%)</td>
<td>3/6 (50%)</td>
<td>p= 0.029</td>
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<td>PPI – TCL &lt; 125</td>
<td>0/14 (0%)</td>
<td>5/6 (83%)</td>
<td>p= 0.001</td>
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<tr>
<td>cPPI</td>
<td>170 ± 34ms</td>
<td>115 ± 69</td>
<td>Cl= 21.7 – 88.3</td>
</tr>
<tr>
<td>cPPI &lt; 110</td>
<td>0/13 (0%)*</td>
<td>4/6 (67%)</td>
<td>p= 0.007</td>
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<tr>
<td>ΔVA</td>
<td>160 ± 34ms</td>
<td>101 ± 76ms</td>
<td>Cl= 25.9 – 42.2</td>
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<td>ΔVA &lt; 85</td>
<td>0/14 (0%)</td>
<td>4/6 (67%)</td>
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<td>ΔHA</td>
<td>72 ± 40ms</td>
<td>-1 ± 58ms</td>
<td>Cl= 35.6 – 110</td>
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<tr>
<td>ΔHA &lt; 0</td>
<td>0/11 (0%)</td>
<td>4/6 (67%)</td>
<td>p= 0.012</td>
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<tr>
<td>ΔAH</td>
<td>29 ± 19ms</td>
<td>10 ± 17ms</td>
<td>Cl= 3.03 – 35.0</td>
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* cPPI could not be calculated in 1 patient because a His bundle deflection was not observed following entrainment

Table 2. Differential diagnosis of a long RP tachycardia reset (advanced or delayed) or terminated (with VA block) by His-refractory VPDs.

<table>
<thead>
<tr>
<th></th>
<th>Atypical AVNRT + bystander NF</th>
<th>NFRT</th>
<th>PJRT</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPI – TCL</td>
<td>&gt;125ms</td>
<td>&lt;125ms</td>
<td>&lt;125ms</td>
</tr>
<tr>
<td>ΔAH</td>
<td>&gt;40ms or AH_{SVT} &lt; AH_{NSR}</td>
<td>&gt;40ms or AH_{SVT} &lt; AH_{NSR}</td>
<td>&lt;20ms</td>
</tr>
</tbody>
</table>
Figure Legends:

Figure 1. ORT using a decremental atrio-ventricular AP (PJRT). Top: A His-refractory VPD paradoxically delays tachycardia by 19ms (fully compensatory). Bottom: Entrainment from the ventricle with antidromic capture of the His bundle showing a pseudo A-A-V pattern. All criteria (PPI (253ms), cPPI (251ms), ΔVA (233ms), ΔHA (124ms)) yield a false diagnosis of atypical AVNRT.

Figure 2. ORT using a concealed nodo-ventricular AP (NVRT). Top: A spontaneous His-refractory VPD terminates tachycardia with VA block. Paradoxically, AH_{SVT} < AH_{NSR} which excludes PJRT. Bottom: Entrainment from the ventricle with orthodromic capture of the His bundle showing an A-V-A response, PPI – TCL = 100ms, ΔVA = 38ms, and ΔHA = -11ms.

Figure 3. Atypical AVNRT with a concealed, bystander nodo-fascicular AP. Top: A His-refractory VPD paradoxically delays tachycardia by 41ms (fully compensatory). The AH_{SVT} is very short (38ms) (AH_{NSR} = 54ms). Bottom: Entrainment from the ventricle with antidromic capture of the His bundle showing a true A-A-V response (retrograde FP and SP/NF AP) and long PPI – TCL = 135ms, ΔVA = 135ms, and ΔHA = 39ms.

Figure 4. Atypical AVNRT with a concealed, bystander nodo-fascicular AP. Top: A His-refractory VPD terminates tachycardia with VA block. Paradoxically, AH_{SVT} < AH_{NSR} which excludes PJRT. Middle: Entrainment from the ventricle yields a true A-A-V response and a long PPI – TCL = 232ms. Note that the atrium is not advanced until the 3rd complex despite

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retrograde capture of the His bundle indicating that the His bundle is not part of the circuit further excluding ORT. Bottom: Para-Hisian entrainment. Loss of His bundle capture (last paced complex) causes a 55ms increase in the VA at the anteroseptum (indicating FP conduction) which then exposes conduction over a slowly-conducting NF AP caused by the penultimate pacing stimulus (arrows). Note the change in the proximal CS electrogram and equivalent increase in the VA at the posteroseptum that is only possible with a NF (not NV or AV) AP.

**Figure 5.** PPI –TCL values for atypical AVNRT (with/without a concealed, bystander NF AP) and ORT (PJRT/NFRT).

**Figure 6.** Diagram illustrating the reentrant circuits for the four atypical AV node – dependent SVTs. The upper and lower portions of each circuit differ and require separate pacing maneuvers for diagnosis. * A concealed NF AP bypasses the His bundle and allows access to the AVNRT circuit from the ventricle. HPS = His-Purkinje system
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