Establishing the Mechanism of Supraventricular Tachycardia in the Electrophysiology Laboratory

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ABSTRACT. Supraventricular tachycardia (SVT) is a common disorder which is associated with significant morbidity due to recurrent symptoms and multiple hospital visits. With advancements in mapping and ablation technology, most of the patients with SVT are now treated with radiofrequency ablation. However, for the procedure to be highly effective and safe it is imperative that the mechanism of tachycardia be understood accurately. The current paper reviews various tachycardia features and maneuvers that help to correctly establish the mechanism of various narrow complex SVTs.

KEYWORDS. supraventricular tachycardia, atrioventricular, atrial tachycardia.

Introduction

Supraventricular tachycardia (SVT) is a common disorder, affecting 570,000 people each year. The patients are often young, although the disorder can present at any age. While rarely life-threatening, SVT can be the source of significant morbidity, including disabling symptoms and hospital visits. While medications including atrioventricular (AV) nodal blocking agents and other antiarrhythmic drugs are reasonable treatments, radiofrequency ablation (RFA) has revolutionized the management of SVT. The vast majority of SVTs are one of three types of arrhythmia, atrioventricular nodal re-entrant tachycardia or AVNRT (responsible for approximately 65% of cases), atrioventricular reciprocating tachycardia or AVRT (responsible for approximately 30% of cases), and atrial tachycardia or AT (responsible for approximately 5% of cases). Radiofrequency ablation can be effective treatment for SVT with cure rates ranging from >70% for AT to over 95% for AVRT and AVNRT. Before radiofrequency ablation can be performed, however, an electrophysiologic study should be performed to correctly diagnose the mechanism of the SVT. In this review we will discuss different maneuvers that can help determine the SVT mechanism during an electrophysiological study. It is important to recognize that as with most diagnostic tests, no single observation or maneuver discussed below is 100% sensitive or specific. Therefore it is important to obtain data from multiple observations and maneuvers to verify the diagnosis before proceeding with ablation.

Observations during sinus rhythm and during initial electrophysiologic testing

When approaching an unknown SVT, it is important to incorporate all pieces of information in analysis, including surface electrocardiograms (ECGs) and baseline measurements during sinus rhythm. At baseline, is the activation concentric, implying midline activation of the ventricle during sinus rhythm, or eccentric, meaning that the lateral wall of the ventricle is activated prematurely? It is important to keep in mind that eccentric conduction nearly always is associated with an accessory pathway, but midline conduction does not rule out the possibility of a septal accessory pathway or a pathway that conducts only in a retrograde manner. Pre-excitation observed during sinus rhythm has a positive predictive value of 86% for AVRT as the mechanism of the tachycardia. During the baseline electrophysiologic study it is also...
important to evaluate the antegrade and retrograde conduction patterns (concentric versus eccentric) during atrial and ventricular pacing. It is possible that a patient may demonstrate concentric antegrade conduction during sinus rhythm, but develops eccentric conduction with rapid atrial pacing as the refractory period of the AV node is reached and the accessory pathway becomes the sole antegrade conduction system. During ventricular pacing it is important to assess for eccentric conduction, as it is common for accessory pathways to conduct only in a retrograde fashion. During atrial and ventricular pacing it is also important to look for decremental conduction. While there are rare reports that accessory pathways can demonstrate the property of decremental conduction, observing it nearly always implies AV nodal conduction and effectively rules out a competitive accessory pathway. A complete lack of retrograde conduction also rules out the possibility of AVRT as the mechanism of the tachycardia.4

Observations during tachycardia

Once the tachycardia is initiated in the electrophysiology laboratory, or if the patient presents to the laboratory in tachycardia, certain tachycardia characteristics may provide important clues to the mechanism of the tachycardia even before attempting diagnostic maneuvers.

Rate: There is substantial overlap in the rates of tachycardias and different mechanisms but an SVT cycle length of over 500 ms (<120 bpm) has a relatively high positive predictive value for AVNRT (approximately 83%).4

VA interval: The VA interval is similar to the RP interval measured on a surface ECG. A septal VA interval of <70 ms was found in 47% of patients with SVT and had a 99% PPV for AVNRT. Generally AVRT and AT have VA intervals of ~70 ms. Exceptions are present, including the situation in which a patient with an AT has substantial PR prolongation leading to a relatively short VA interval4 (Figure 1).

Termination of tachycardia with AV block

Termination of the tachycardia with the development of AV block (spontaneous or induced by vagal maneuvers

Figure 1: Short VA interval <70 ms in a patient with typical atioventricular nodal re-entrant tachycardia (AVNRT) (slow–fast). The VA interval is a pseudo-interval in case of a typical (slow–fast) AVNRT and is a result of near simultaneous activation of atrium and ventricle. Short VA interval <70 ms virtually rules out the AVRT as the mechanism of the tachycardia. A short VA, however can also be seen in junctional tachycardia and a septal atrial tachycardia.
or AV nodal blocking medications) favors the diagnosis of the AV nodal-dependent tachycardias, AVNRT or AVRT. This finding has a PPV value of 66% for AVNRT, 33% for AVRT, and almost 0% for AT.4 While it is theoretically possible for an AT to terminate simultaneous to AV block, it would be significantly coincidental, thereby decreasing the possibility.

**Atrial activation during tachycardia**

An eccentric atrial activation favors the diagnosis of AVRT and AT (Figure 2). This has a positive predictive value of 76% and 24% for AVRT and AT, respectively. However, eccentric activation cannot rule out AVNRT as the mechanism of tachycardia. Similarly proximal to distal atrial activation in coronary sinus catheter can be seen in the septal accessory pathway (Figure 3). It has been reported that patients with left-sided inputs to the AV node can have distal to proximal atrial activation in coronary sinus catheters.6 In this situation two observations during tachycardia that may favor the diagnosis of AVNRT are VA interval (<70 ms) and the earliest retrograde activation in the His catheter.

**Initiation dependent on a critical AH or VH interval**

A sudden increase in the AH or VH interval with the delivery of a slightly decrementing single extrastimulus implies the presence of dual AV nodal pathways and is strongly suggestive of AVNRT as the mechanism of the SVT with a PPV of 91%. Because of the difficulty in seeing a His signal during ventricular pacing, the sudden AH prolongation, known as a “jump,” observed with delivery of slowly decrementing single atrial extrastimuli, is the more typically helpful maneuver. By definition, an AH jump is defined as increase in the AH interval of more than 50 ms following a 10-ms decrement in the paced coupling interval between the last two atrial paced beats when compared with the final coupling interval in the previous atrial drivetrain and extrastimuli.4

**Continuation of tachycardia during AV block**

If the tachycardia continues during development of VA block, AVRT as the mechanism of the tachycardia is excluded because of the need for ventricular participation. While on first blush it would seem that AVNRT should also

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**Figure 2:** Eccentric activation during tachycardia in a patient with left free wall pathway. Note the earliest retrograde atrial activation seen at CS 1 and 2.

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Figure 3: Proximal to distal atrial activation during tachycardia. Note the earliest atrial activation is at CS 7 and 8. Also note the long VA time of 209 ms. This patient had a posteroseptal accessory pathway.

Figure 4: Demonstration of Coumel’s Law. The tracing shows the effect of left bundle branch block on the tachycardia cycle length (TCL) and VA interval. Note during left bundle branch block the VA increases from 99 ms to 126 ms. The TCL increased from 249 ms to 376 ms during the transient left bundle branch block. This patient had a left sided accessory pathway. It is possible that the TCL could remain unchanged in this situation so it is recommended that the VA interval be used when possible.
Figure 5: A His-synchronous premature ventricular contraction (PVC) advances the next atrial signal without terminating the tachycardia. Note atrial activation remains unchanged and the tachycardia is reset. This proves that the pathway is not only present but critical to the tachycardia circuit.

Figure 6: A His-refractory premature ventricular contraction (PVC) terminates the tachycardia without conducting to the atrium. The PVC blocks in the accessory pathway and this maneuver not only proves that the pathway is present but also is critical to tachycardia circuit.
Figure 7: Pre-excitation index Pi₁ is measured by subtracting the longest coupling interval of the premature ventricular contraction (PVC) (V1 V2) from the tachycardia cycle length (TCL). The TCL in this case is 410 ms and V1 V2 is 346. Pi₁ = 410 – 346 = 64 ms. The Pi₂ = V1V2/TCL = 346/410 = 0.8. Pi₁ of 64 ms is consistent with a left free wall accessory pathway.

Figure 8: Ventricular overdrive pacing in atrial tachycardia. Not the post-pacing response is VAAV. One can also observe that atrial activation is different during ventricular pacing than during tachycardia.
be excluded, the complexity of AV node and containment in the atrium with the presence of a lower common pathway allows this to be possible. This occurrence highly favors the diagnosis of AVNRT and atrial tachycardia with a PPV value of 60% and 40%, respectively.4

Development of bundle branch block

It is not unusual to observe aberration during SVT. The rapidity of the conduction can lead to functional block in one of the bundles. Development of left bundle branch block (BBB) favors the diagnosis of AVRT with a positive predictive value of 92%. An increase in the VA interval of more than 20 ms during development of BBB has a positive predictive value of nearly 100% for AVRT and also helps with the localization of the accessory pathway.4 In the setting of AVRT, sudden aberration with prolongation in the VA time localizes the involved accessory pathway to the side on which the functional block is occurring (Coumel’s Law)7 (Figure 4).

Maneuvers during tachycardia

While the previous observations and pacing maneuvers may suggest the mechanism of SVT, it is unlikely that a definitive diagnosis can be made without performing some maneuvers during tachycardia.

Delivery of His-synchronous premature ventricular contractions

Extrasystole, whether spontaneous or induced, can often help identify the mechanism of arrhythmia. A commonly used maneuver is to deliver a His-synchronous premature ventricular contraction (PVC), delivered on time or within 40 ms of the His potential. Once this PVC is delivered, careful measurements should be made to assess whether the subsequent atrial signal has been advanced. Often this may only be a variation of several milliseconds, so great care must be observed in measurement. If the subsequent atrial signal arrives earlier than expected, an accessory pathway is present (Figure 5). If the tachycardia

Figure 9: Ventricular entrainment of a typical atrioventricular nodal re-entrant tachycardia (AVNRT). Note a short septal VA interval of 16 ms and a typical VAHV or a VAVA post-pacing response. Post-pacing interval—TCL > 115 ms consistent with a typical AVNRT. All these features are consistent with a diagnosis of a typical AVNRT.

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terminates during this maneuver without conducting to the atrium, an accessory pathway is present and is a necessary part of the arrhythmia circuit and not just a possible bystander accessory pathway (Figure 6). If no change in the subsequent atrial signal is observed, it does not rule out the possibility of an accessory pathway.8

Pre-excitation index

A PVC delivered during the tachycardia (but not in a His-synchronous fashion) can potentially affect the tachycardia either by pre-exciting, post-exciting, or terminating it and can be used to calculate a measurement known as the pre-excitation index (PI). In the previous section we discussed the role of His-refractory PVCs. A single PVC delivered much earlier can potentially penetrate the circuit of not just AVRT but also AVNRT. The degree of prematurity of the PVC that can advance the subsequent atrial signal can be used to identify AVNRT or localize the accessory pathway in AVRT.9 Miles et al.9 has previously reported on two methods of calculating the PI. PI1 is the difference between tachycardia cycle length (TCL) and the longest coupling interval of the delivered PVC that is capable of advancing the next atrial electrogram (Figure 7):

\[ PI_1 = \text{TCL} - \text{longest coupling interval that pre-excites the atrium (V1V2)} \]

PI2 is the difference in the coupling interval that advances the next atrial electrogram divided by the TCL:

\[ PI_2 = \frac{(V1 - V2)}{\text{TCL}} \]

In using this maneuver, it is important that the atrial activation sequence remains unchanged. Because of the proximity of the RV catheter to the tachycardia circuit in orthodromic reciprocating tachycardia (ORT) it is much easier to pre-excite the atrium than AVNRT, where the circuit is away from the RV catheter. A PI1 of >100 is consistent with the diagnosis of AVNRT. In case of ORT using a septal pathway, PI is usually <45 ms, and ORT involving a left free wall pathway PI is usually >75 ms. The mean PI2 were 0.75 for left free wall pathway, 0.88 for posteroseptal pathway, 0.95 for anteroseptal pathway, and 0.75 for AVNRT. Thus the PI1 measurement appears to better differentiate location and mechanism of the tachycardia and should be preferentially used over PI2 (Figure 7).

Figure 10: Pseudo-VAAV response to ventricular entrainment in a patient with an atypical (fast–slow) AVNRT. In this case last entrained atrial beat (#2) should be the first post-ventricular atrial beat.
Retrograde right bundle branch block
Retrograde right BBB is commonly seen during ventricular extrastimuli. By definition a retrograde BBB is defined as an increase in VH interval of >50 ms. In a study by Srivathsan et al., 22 out of 105 patients had evidence of induced retrograde right BBB. The average VH interval increase with induction of right BBB was 53.7 ms for patients with AVRT and 54.4 ms for patients with AVNRT (p=NS). The average VA interval increase with induction of right BBB was 13.6 ms with AVRT and 70.1 ms with AVNRT (p<0.001). All patients with a greater VH than VA interval change had AVRT, and those with a smaller had AVNRT.

Entrainment from the ventricle during tachycardia
During entrainment from the right ventricle (RV) pacing is performed at a cycle length slightly faster (10–40 ms) than the tachycardia. This allows for the penetration of the excitable gap of the re-entrant circuit and acceleration of the atrium to the pacing cycle length. Before interpreting post-entrainment responses it is very important to pay close attention to following: 1) the pacing captures the RV; 2) the atrium is accelerated to the pacing cycle length; 3) the tachycardia does not terminate subsequently and resets following termination of the pacing to the original cycle length. Entrainment of AVNRT is usually concealed as the fusion between the orthodromic wavefront of the nth beat and antidromic wavefront of the n+1 pacing beat occurring within the node. In the case of ORT, depending on whether the fusion occurs above or below the His bundle, the His bundle can be activated by the retrograde antidromic wavefront or antegrade orthodromic wavefront of the pacing beat. When the His bundle is orthodromically activated fusion always occurs in the ventricle and is usually manifested by a fused QRS morphology. Other features of orthodromic His capture include similar His morphology during entrainment and SVT, AH interval during entrainment greater or equal to the first AH of the SVT.
Post-pacing response
Following entrainment the post-pacing response can provide useful information. Typically following entrainment, either a VAAV response or a VAVA response will be seen. In a VAAV response, which is consistent with a diagnosis of AT, the last paced ventricular beat (V) goes up the AV node and suppresses the atrial automatic focus with an early atrial electrogram (A). The atrial event following last paced V can not descend down the AV node as it will be still refractory. If the tachycardia continues the next beat will initiate an atrial depolarization (A) which conducts to the ventricle (V) yielding the VAAV response (Figure 8).11

In a VAVA response (Figure 9) which is consistent with either AVNRT or AVRT, the last ventricular paced beat (V) travels up to the atrium (A), either through the AV node or accessory pathway depending on whether the SVT is AVNRT or AVRT.11 Subsequently the ventricle and then the atrium are again activated by continuation through the re-entrant loop (VA). Note that in some situations a pseudo-VAAV response can occur especially when the retrograde conduction is slow either in an atypical AVNRT or a retrogradely conducting slow accessory pathway. In this situation recognizing the last entrained atrial beat is important as that is the first post-ventricular atrial beat (Figure 10).

Post-pacing interval
The post-pacing interval can also help differentiate the arrhythmia mechanism. During AVRT, the post-pacing interval following ventricular entrainment will generally be similar to the TCL given that the ventricle is close to the arrhythmia circuit. In contrast, the post-pacing interval in AVNRT will often be substantially longer than the TCL because of the distance of the RV apex from the AV nodal circuit. Empiric studies have found that AVNRT will typically have a post-pacing interval of greater than 115 ms over the TCL while AVRT will be less than 115 ms over the TCL. Note that this finding remains true in atypical AVNRT and is useful in separating a long RP atypical AVNRT from AVRT.12

During pacing, decremental conduction can occur in the AV node, which can falsely increase the post-pacing interval. In this situation a corrected post-pacing interval (PPI–AH) of >110 ms is consistent with an AVNRT13 (Figure 8).
While performing ventricular entrainment, the difference in the measurement of the stimulation to atrial electrogram and the VA time during SVT can also be used to differentiate typical or atypical AVNRT from AVRT. A difference greater than 85 ms supports the diagnosis of AVNRT as the mechanism of tachycardia (Figure 11).12

Delta HA
Ho et al.14 showed a positive delta HA interval, which is calculated by subtracting HA interval during tachycardia from the HA interval during entrainment from the RV has a sensitivity, specificity, and positive predictive value of 100% in differentiating AVNRT from ORT. A value of more than 0 always occurs in AVNRT. The reason for this is that atria and the ventricles are activated nearly simultaneously during AVNRT and in series during entrainment from the RV. The opposite is true about AVRT, in which atria and the ventricle are activated in series during tachycardia and in parallel during entrainment from the RV. It is often times difficult to appreciate the retrograde His especially when pacing from the right ventricular apex. This maneuver is helpful only when a retrograde His is clearly seen and may even require pacing from the right ventricular base.14

Assessing AV nodal dependence
Supraventricular tachycardias almost always demonstrate some variability in cycle length, particularly near the onset and termination of the arrhythmias. During these periods carefully measure the cycle length, HH intervals, and AA intervals. Evidence of the HH variation leading to subsequent AA variation requires that the tachycardia is dependent on the AV node and rules out the possibility of an atrial tachycardia.15

Maneuvers for special circumstances
Parahisian pacing
This maneuver, performed in sinus rhythm, helps determine whether midline conduction is occurring

Figure 13: Parahisian pacing in a patient with a septal accessory pathway. Note same stimulation to earliest atrial activation time with His capture (a) and without His (b) capture. During His capture QRS complex is narrow and it is wide when there is no His capture. This response to parahisian pacing is called extranodal respons.
through the AV node or a septal pathway. During this maneuver pacing is performed either from the His catheter or preferably through a separate catheter placed close to the His bundle. Pacing is started at a high output to capture the deeply seated and insulated His directly and the surrounding myocardial tissue. The pacing output is gradually decreased until His capture is lost. When the His is captured directly the resulting QRS will be narrow and when His capture is lost the QRS will widen into a bundle branch block pattern. The time from the stimulation artifact to the subsequent atrial signal is measured during His capture and during the loss of His capture. The expectation is that in the setting of no accessory pathway, loss of His capture will result in a widening of the QRS complex and a simultaneous increase in the stimulation-atrial time (Figure 12). In contrast, the presence of a septal accessory pathway will result in an identical stimulation-atrial time both with and without His capture (Figure 13). During this maneuver, it is very important to make sure that there is no capture of local A as this could cloud the results. The presence of a very short stimulation to proximal coronary sinus A (<60 ms) and stimulation to high right A (<70 ms) is suggestive of direct capture of the atrium from the pacing catheter. A stimulation atrial EGM time of greater than 90 ms in the proximal coronary sinus and 100 ms in high right atrium argues strongly against direct atrial capture.

**Differential ventricular pacing**

Differential pacing at the base and the apex of the ventricle can also help identify the presence of an accessory pathway. During this maneuver pacing is sequentially performed at the base and the apex of the ventricle. A stimulation to atrial EGM time is measured (Figure 14). Without the presence of an accessory pathway, the stimulation to atrial time is shorter when pacing is performed at the apex compared with base. However, in the case of an ipsilateral accessory pathway, the stimulation to atrial time is the same during pacing at the apex or base of the RV.

**Resetting zone**

One of the difficulties with ventricular overdrive pacing or entrainment is the possible termination of the tachycardia following pacing. In this situation it is often difficult to assess the post-pacing response. A new
A diagnostic maneuver looking at the transition zone during ventricular pacing may help in these situations. In the transition zone the QRS complex changes morphology and becomes stable over few beats. In a study by Dandamudi et al.\textsuperscript{19} the number of beats required to accelerate the tachycardia to the pacing rate was calculated. In AVNRT the mean number of pacing beats (having a stable paced morphology) required to reset the tachycardia was 3.7 $\pm$ 1.1 while in AVRT it was 1.0 $\pm$ 0. The authors used a cut off of $>1$, which had a 100% positive and a negative predictive value for AVNRT over AVRT. This maneuver is based on a simple principle that it is easier to entrain and reset AVRT during ventricular entrainment as the ventricular pacing site is close to the tachycardia circuit when compared with AVNRT. Also the AVRT circuit is larger than AVNRT circuit with a large excitable gap (Figure 15).

Differentiating junctional tachycardia from AVNRT

Following ablation or modification of the slow pathway of the AV node for the treatment of AVNRT, it is not unusual to see an accelerated junctional rhythm. Given the appearance, it is sometimes difficult to distinguish this from a slower AVNRT, implying that the ablation was unsuccessful and further ablation may be necessary. In these situations, a His-refractory APC that pre-excites the next His terminates the tachycardia or post-excites the next His is consistent with the AVNRT. However if it does not affect the next His the diagnosis of junctional tachycardia is likely.\textsuperscript{20} An earlier APC that terminates the tachycardia is consistent with the AVNRT. However, if an earlier APC brings the next His early and the tachycardia continues it is likely a junctional focus.\textsuperscript{20} Overdrive atrial pacing resulting in an AHA response or a pseudo-AHHA response is consistent with an AVNRT rather than junctional tachycardia, in which case the post-pacing response will be AHHA.\textsuperscript{21} Srivathsan et al.\textsuperscript{22} calculated the HA interval during tachycardia and subtracted it from the HA interval during pacing from the RV at TCL. They found a delta HA interval $>0$ ms had a sensitivity of 89%, specificity of 83%, positive predictive value of 84%, and negative predictive value of 88% for junctional tachycardia.\textsuperscript{22}
Summary

Supraventricular tachycardia is a common problem. Often the diagnosis and treatment is straightforward. However, given the variability that is often seen, it is important that electrophysiologists have a wide array of diagnostic maneuvers available to help in the occasionally difficult case.

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