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ORIGINAL RESEARCH

CATHETER ABLATION - SVT

The Value of Programmed Ventricular Extrastimuli From the Right Ventricular Basal Septum During Supraventricular Tachycardia

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ABSTRACT

BACKGROUND The difference between the right ventricular (RV) apical stimulus-atrial electrogram (SA) interval during resetting of supraventricular tachycardia (SVT) versus the ventriculoatrial (VA) interval during SVT (ΔSA-VA_{apex}) is an established technique for discerning SVT mechanisms but is limited by a significant diagnostic overlap.

OBJECTIVES This study hypothesized that the difference between the RV SA interval during resetting of SVTs versus the VA interval during SVTs (ΔSA-VA) would yield a more robust differentiation of atrioventricular nodal re-entrant tachycardia (AVNRT) from atrioventricular reciprocating tachycardia (AVRT) when using the RV basal septal stimulation (ΔSA-VA_{base}) as compared to the RV apical stimulation (ΔSA-VA_{apex}). Moreover, it was predicted that the ΔSA-VA_{base} might distinguish septal from free wall accessory pathways (APs) effectively.

METHODS In this prospective study, 105 patients with AVNRTs (age 48 $\pm$ 20 years, 44% male) and 130 with AVRTs (age 26 $\pm$ 18 years, 54% male) underwent programmed ventricular extrastimuli delivered from both the RV basal septum and RV apex. The ΔSA-VA values were compared between the 2 sites.

RESULTS The ΔSA-VA_{base} was shorter than the ΔSA-VA_{apex} during AVRT (44 $\pm$ 30 ms vs 58 $\pm$ 29 ms; $P < 0.001$), and the opposite occurred during AVNRT (133 $\pm$ 31 ms vs 125 $\pm$ 25 ms; $P = 0.03$). A ΔSA-VA_{base} of $\geqq$85 milliseconds had a sensitivity of 97% and specificity of 96% for identifying AVNRT. Furthermore, a ΔSA-VA_{base} of 45-85 milliseconds identified AVRT with left free wall APs (sensitivity 86%, specificity 95%), 20-45 milliseconds for posterior septal APs (sensitivity 72%, specificity 96%), and <20 milliseconds for right free wall or anterior/mid septal APs (sensitivity 86%, specificity 98%).

CONCLUSIONS The ΔSA-VA_{base} during programmed ventricular extrastimuli produced a robust differentiation between AVNRT and AVRT regardless of the AP location with $\geqq$85 milliseconds as an excellent cutoff point. This straightforward technique further allowed localizing 4 general AP sites. (J Am Coll Cardiol EP 2023;9:219–228) © 2023 by the American College of Cardiology Foundation.

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ABBREVIATIONS AND ACRONYMS

AP = accessory pathway

AVNRT = atrioventricular nodal re-entrant tachycardia

AVRT = atrioventricular reciprocating tachycardia

CI = coupling interval

NPV = negative predictive value

PPV = positive predictive value

ROC = receiver-operating characteristic

RV = right ventricular

SA = stimulus-atrial

SVT = supraventricular tachycardia

TCL = tachycardia cycle length

V2 = programmed ventricular extrastimuli

VA = ventriculoatrial

VOP = ventricular overdrive pacing

The differentiation of atrioventricular nodal re-entrant tachycardia (AVNRT) from atrioventricular reciprocating tachycardia (AVRT) as well as the localization of accessory pathways (APs) are necessary to guide the catheter ablation of supraventricular tachycardia (SVT).[1-13] However, existing techniques may prove challenging: for example: 1) in differentiating atypical forms of AVNRT from AVRT using a septal AP; or 2) localizing AVRT with different septal AP insertion sites. Therefore, a comprehensive diagnostic maneuver that can be applicable to both situations would be potentially of value in challenging cases.

The difference between the ventricular stimulus-atrial electrogram (SA) interval with ventricular entrainment or resetting with premature ventricular complexes and the ventriculoatrial (VA) interval during SVT (ΔSA-VA) is a commonly used and important diagnostic maneuver.[1-8] Although several reports have demonstrated the usefulness of the ΔSA-VA using right ventricular (RV) apical stimulation ($\Delta SA\text{-}VA_{apex}$) to distinguish AVNRT from AVRT, there remains significant overlap in the ΔSA-VA complicating the distinction between these 2 arrhythmias.[1]

Previous studies have shown that positional entrainment (RV basal pacing vs apical pacing) may serve as an ancillary discriminatory maneuver to diagnose challenging SVT cases.[3-5,9] This finding is best explained by the anatomic distance between the pacing site versus the SVT circuit. Theoretically, stimulation from the RV basal septum ($\Delta SA\text{-}VA_{base}$) would be expected to shorten the SA interval in AVRT because atrioventricular pathways insert at the ventricular base. On the other hand, the $\Delta SA\text{-}VA_{base}$ should have the opposite effect on AVNRT because the impulse must first pass from the RV base to the apex to access the right bundle branch. Therefore, we hypothesized that: 1) the $\Delta SA\text{-}VA_{base}$ should yield a more robust differentiation of AVNRT from AVRT than the $\Delta SA\text{-}VA_{apex}$; and 2) the $\Delta SA\text{-}VA_{base}$ might further better differentiate the AP sites than the $\Delta SA\text{-}VA_{apex}$.

METHODS

This was a multicenter prospective study in which we delivered programmed ventricular extrastimuli (V2) from both the RV basal septum (distal His bundle region) and RV apex during SVT in patients with AVNRT or AVRT until SVT resetting was achieved. We delivered V2 instead of ventricular overdrive pacing (VOD) because the V2 technique is associated with a lower frequency of tachycardia termination as compared to the VOD technique.[1] Participants were excluded if 2 or more tachycardia mechanisms were present, the tachycardia was not sustained, or the tachycardia cycle length (TCL) varied by >30 milliseconds. In addition, patients with atrial tachycardia, antidromic AVRT, and tachycardia using a nodoventricular or nodofascicular AP were excluded from this study. The clinical characteristics, including the age, sex, body mass index, ejection fraction, and presence of cardiac disease were recorded. All studies were performed as part of the clinical evaluation of the patients' arrhythmias. This study was conducted according to the principles of the Declaration of Helsinki, and the study protocol was approved by the local ethics committees. All studies were initially performed with a signed informed consent, and the data were retrospectively collected and completely anonymized.

ELECTROPHYSIOLOGICAL STUDY. Standard techniques were used to diagnose AVNRT and AVRT. All antiarrhythmic drug therapies were discontinued for least 5 half-lives before the procedure. Multipolar electrode catheters were introduced and positioned in the high right atrium, coronary sinus, His-bundle region, and RV. Twelve-lead surface electrocardiograms and intracardiac electrograms were recorded and stored on a Prucka CardioLab (GE Healthcare) recording system. The bipolar intracardiac electrograms were filtered between 30 and 500 Hz as well as recorded at a speed of 100 mm/s. Bipolar pacing was performed at twice the diastolic threshold from the distal electrode pair.

The tachycardia was induced by programmed stimulation from the RV or high right atrium. If SVT was not inducible or not sustained at baseline, isoproterenol was then infused continuously to

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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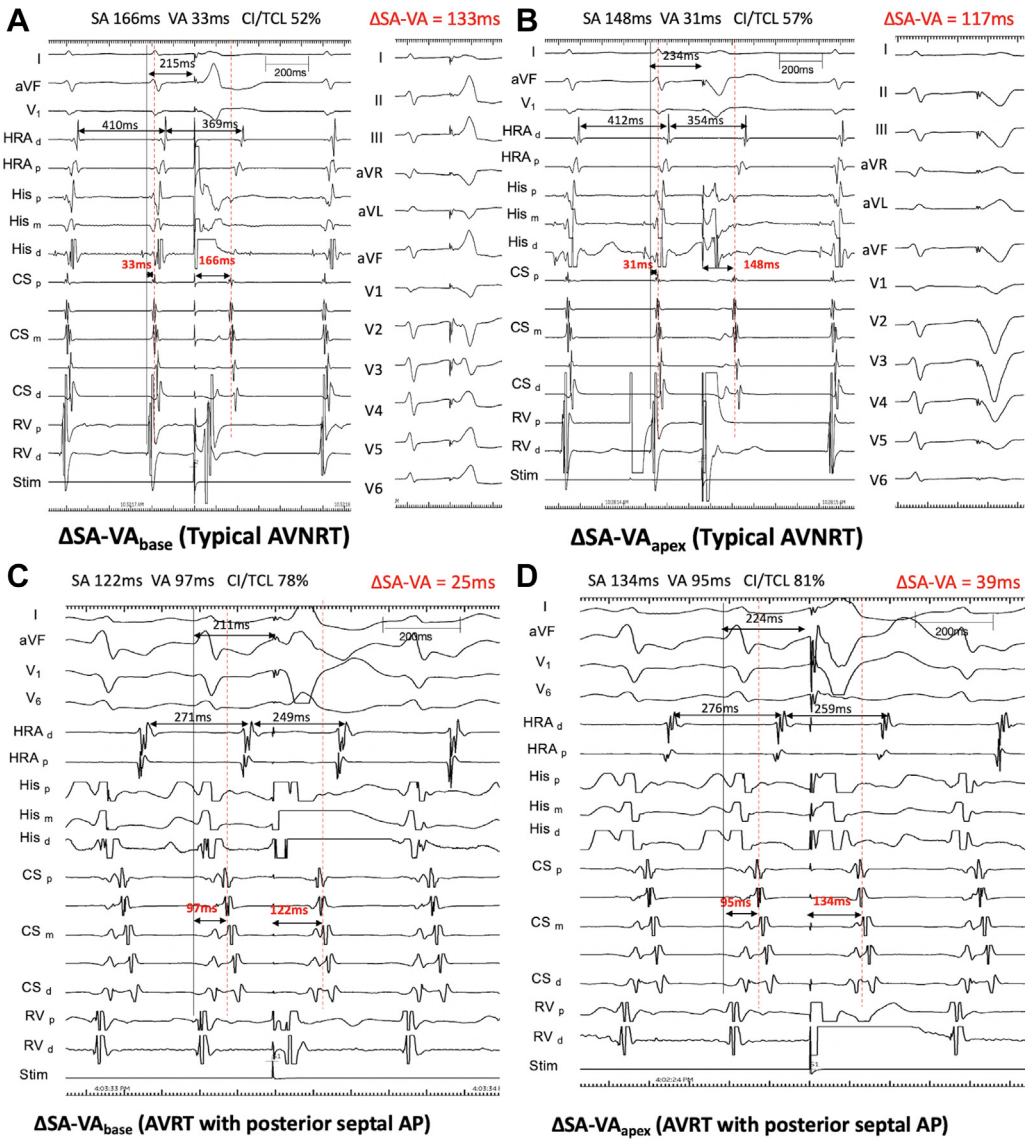
FIGURE 1 **An Example of the ΔSA-VA$_{base}$ and ΔSA-VA$_{apex}$ Using the V2 Technique**



The differentiation of right ventricular (RV) stimulus-atrial electrogram (SA) interval and the atrioventricular nodal re-entrant tachycardia (AVNRT) from atrioventricular reciprocating tachycardia (AVRT) when using **(A)** the RV basal septal stimulation (ΔSA-VA$_{base}$) and **(B)** the RV apical stimulation (ΔSA-VA$_{apex}$) in a patient with typical AVNRT. The 12 lead ECGs are representative of RV basal versus apical stimulation, respectively. The stimulated (Stim) ventricular beat advances the next atrial potential on the high right atrial (HRA) catheter. The VA interval is measured from the onset of the surface QRS complex to the rapid deflection of the atrial electrogram during supraventricular tachycardia on the same electrode recording used for the measurement of the SA interval. The ΔSA-VA is calculated by subtracting the VA interval during the supraventricular tachycardia from the SA interval obtained by the V2 technique. The ratio of the shortest coupling interval (CI) that entered into the tachycardia and tachycardia cycle length (TCL) is also calculated. Note that in this case, the ΔSA-VA$_{base}$ is longer than the ΔSA-VA$_{apex}$ supported by the longer SA interval during RV basal pacing. The **(C)** ΔSA-VA$_{base}$ and **(D)** ΔSA-VA$_{apex}$ in a patient with AVRT using a posterior septal accessory pathway (AP). Note that the ΔSA-VA$_{base}$ is shorter than the ΔSA-VA$_{apex}$ supported by the shorter SA interval during RV basal pacing. CS = coronary sinus; d, m, p = distal, mid, and proximal electrode pairs of the relevant catheter.

accelerate the sinus rate by >20% or to a sinus rate of >100 beats/min, and programmed stimulation was repeated. Atrial tachycardia was excluded by the presence of a ventricular-atrial-ventricular response after RV entrainment of the SVT. The diagnosis of AVNRT versus AVRT as well as the AP location were established according to conventional electrophysiological criteria and successful ablation. Patients were

TABLE 1 Baseline Characteristics

	AVRT (n = 130)	AVNRT (n = 105)	*P* Value
Age, y	26 ± 18	48 ± 20	<0.001
Male	69 (54)	45 (44)	0.08
Left ventricular ejection fraction, %	60 ± 9	60 ± 7	0.68
Structural heart disease	7 (6)	9 (9)	0.36
Hypertension	9 (7)	27 (26)	<0.001
Diabetes	2 (2)	8 (8)	0.05
Chronic heart failure	3 (2)	6 (6)	0.31
Typical AVNRT (slow-fast)	–	91 (87)	
Atypical AVNRT (slow-slow/fast-slow)	–	14 (13)	
AVRT with right free wall AP	19 (15)	–	
AVRT with septal AP	38 (29)	–	
AVRT with left free wall AP	69 (53)	–	
AVRT with slowly conducting AP	4 (3)	–	
Tachycardia			
TCL, ms	330 ± 62	380 ± 75	<0.001
VA interval during tachycardia, ms	108 ± 42	51 ± 73	<0.001
QRS interval, ms	80 ± 16	78 ± 17	0.41

Values are mean ± SD or n (%). Dashes indicate that data did not exist.

AP = accessory pathway; AVNRT = atrioventricular nodal re-entrant tachycardia; AVRT = atrioventricular re-entrant tachycardia; TCL = tachycardia cycle length; VA = ventriculoatrial.

defined as having a slowly conducting AP if the ratio of the VA interval to the TCL was ≧40%, and a non-decremental AP was defined as VA:TCL ratio of <40% with a minimal change in the VA interval with a V2.[10]

MEASUREMENTS AND DEFINITIONS OF THE ΔSA-VA. The V2 technique was performed by delivering a single RV extrastimulus starting from a coupling interval (CI) of 10 milliseconds shorter than the TCL. The CI was decreased in 10-millisecond steps until resetting occurred, the tachycardia terminated, or the ventricular effective refractory period was reached. Entry into the tachycardia circuit was confirmed when the atrial depolarization following the V2 was advanced or delayed by at least 10 milliseconds without termination of the tachycardia (Figure 1). The V2 with the shortest CI was used as the primary value for the measurement.[1] The ratio of the shortest CI that entered into the tachycardia and TCL was also calculated. Stimulation from the RV basal septum was performed from the distal His region using the His-bundle catheter or by withdrawing the RV catheter to the base. RV basal pacing was confirmed by QRS complex morphology, which demonstrated positive deflections in leads V_4-V_6 as well as an inferior axis (Figure 1A). Recordings with His-bundle capture as previously described were excluded from the study.[5,11,12] In contrast, stimulation from the RV apex was confirmed by a QRS complex morphology exhibiting negative deflections in leads V_4-V_6 with a superior axis (Figure 1B).

The SA interval was measured from the RV stimulus to the rapid deflection of the earliest reset atrial electrogram on the high right atrium or coronary sinus recording, whichever was more clearly seen during the tachycardia. The VA interval was measured from the onset of the surface QRS complex to the rapid deflection of the bipolar atrial electrogram during SVT on the same electrode recording used for the measurement of the SA interval. The ΔSA-VA was calculated by subtracting the VA interval during the SVT from the SA interval obtained by the V2 technique (Figure 1).

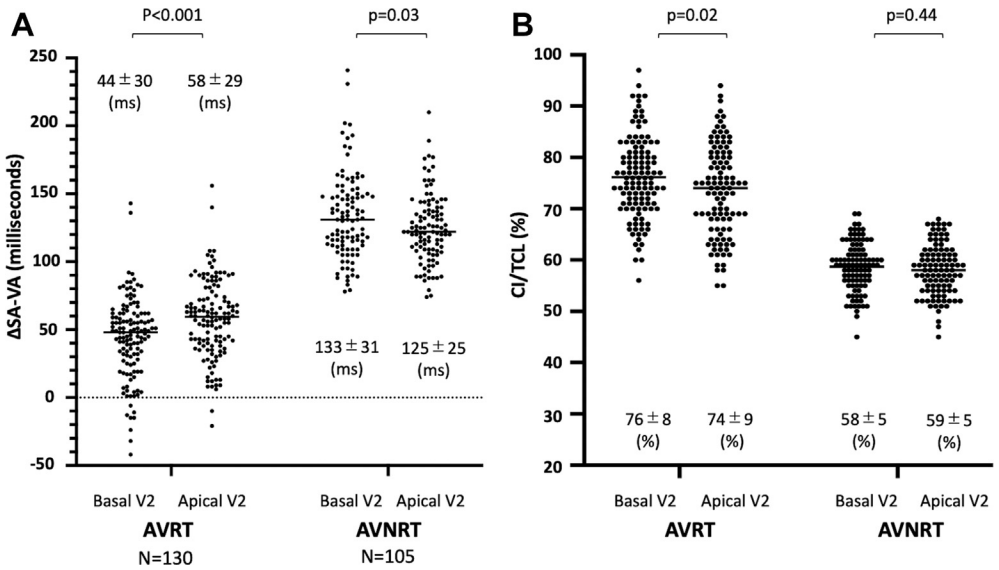
STATISTICAL ANALYSIS. For the statistical analysis, all analyses were performed using SPSS software (version 23.0, SPSS Inc). The results are presented as the mean ± SD or proportion (percentage). Student *t*-tests were used to compare normally distributed continuous variables. The nominal variables were compared using chi-square tests or Fisher exact tests (when an expected value was <5). The receiver-operating characteristics (ROC) curve was calculated to evaluate the performance of the optimal cutoff values for differentiating the SVT mechanism. The sensitivity, specificity, positive predictive values (PPV), and negative predictive values (NPV) were calculated, respectively, based on each group and threshold values. A value of $P < 0.05$ was considered statistically significant.

RESULTS

CLINICAL CHARACTERISTICS. The V2 technique was successfully performed from both the RV basal septum and RV apex in 235 patients including 130 with AVRT (55%) and 105 with AVNRT (45%). The clinical characteristics of each group are shown in Table 1. The participants with AVNRT were significantly older, had a higher prevalence of hypertension, and had longer TCL than those with AVRT did.

THE ΔSA-VA (V2) FROM THE RV BASAL SEPTUM AND RV APEX. The difference in the ΔSA-VA and CI:TCL ratio from the RV basal septum and RV apex is shown in Figure 2. For those with AVRT, the mean ΔSA-VA_{base} during V2 was significantly shorter than the ΔSA-VA_{apex} (44 ± 30 ms vs 58 ± 29 ms; $P < 0.001$), whereas the ΔSA-VA_{base} was longer than the ΔSA-VA_{apex} among those with AVNRT (133 ± 31 ms versus 125 ± 25 ms; $P = 0.03$) (Figure 2A). Among 130 patients with AVRT, 118 (91%) exhibited a decrease of ≧5 milliseconds in the ΔSA-VA_{base} as compared to that of the ΔSA-VA_{apex}, however, 11 (8%) had equivalent (<5 ms), and 1 (1%) increased values. In contrast, among those with AVNRT, 73 patients (70%)

FIGURE 2 The ΔSA-VA (V2) and CI/TCL Ratio in the Patients With AVRT and AVNRT When Comparing RV Basal Septal Pacing and RV Apical Pacing



(A) The ΔSA-VA_{base} and ΔSA-VA_{apex} among those with AVRT and AVNRT. **(B)** The CI:TCL ratio. Note that a better spilt can be seen in the ΔSA-VA_{base} because of the shorter values in those with AVRT and longer values in those with AVNRT as compared to that of the ΔSA-VA_{apex}. V2 = programmed ventricular extrastimuli; other abbreviations as in Figure 1.

exhibited an increase of ≧5 milliseconds in the ΔSA-VA_{base} as compared to that of the ΔSA-VA_{apex}, however, 24 (23%) had equivalent (<5 ms) and 8 (8%) decreased values. Furthermore, as expected, smaller values of the CI:TCL ratio were found in those with AVNRT than in those with AVRT (Figure 2B).

Figure 3 shows the ΔSA-VA_{base} and ΔSA-VA_{apex} sorted by the SVT mechanisms. There were clear trends in the ΔSA-VA_{base} for a separation between AVNRT (both typical and atypical forms) and AVRT regardless of the AP location. Of note, the ΔSA-VA_{base} showed minimal overlap between those with typical AVNRT and AVRT using left free wall APs, whereas the ΔSA-VA_{apex} exhibited significant overlap between the 2 groups. In addition, when focusing on the ΔSA-VA_{base}, the right free wall APs and anterior/mid septal APs had a smaller ΔSA-VA_{base} than the posterior septal Aps did, and the posterior septal APs had a smaller ΔSA-VA_{base} than those with left free wall Aps did.

DISCRIMINATIVE PERFORMANCE OF THE ΔSA-VA_{base}.

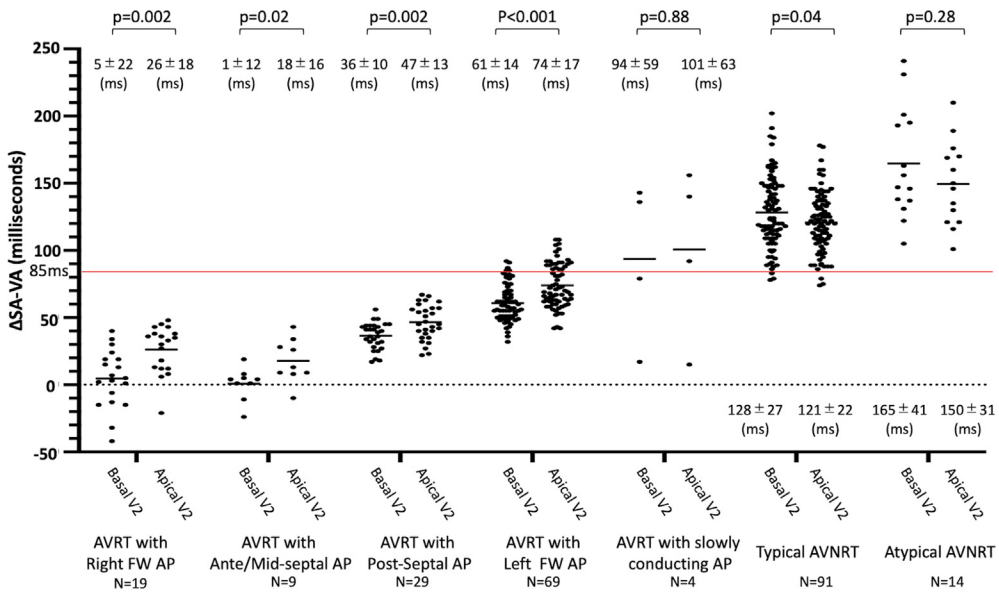
Figure 4 shows the ROC curve analyses for studying the discriminative performance of the ΔSA-VA_{base} (V2) to identify AVNRT. The area under the ROC curve for the RV basal V2 was 0.988 (95% CI: 0.974-1.000; $P < 0.001$), and the best discriminative cutoff value for the ΔSA-VA_{base} was ≧85 milliseconds for differentiating AVNRT from AVRT regardless of the AP location (sensitivity 97%, specificity 96%, PPV of 95%, and NPV of 98%).

Figure 5 summarizes the discriminative performance of the ΔSA-VA_{base} to anatomically localize the AP. A subgroup of 4 patients with AVRT that used a slowly conducting AP were excluded because of a marked overlap with those with AVNRT. The ΔSA-VA_{base} from 45 to 85 milliseconds had a sensitivity of 86%, specificity of 95%, PPV of 88%, and NPV of 94% for localizing left free wall APs, ΔSA-VA_{base} from 20 to 45 milliseconds had a sensitivity of 72%, specificity of 96%, PPV of 70%, and NPV of 96% for localizing pathways in the posterior septal regions, and ΔSA-VA_{base} <20 milliseconds had a sensitivity of 86%, specificity of 98%, PPV of 89%, and NPV of 98% for localizing right free wall or anterior/mid septal AP regions.

DISCUSSION

To the best of our knowledge, this prospective study is the first to evaluate the role of ΔSA-VA_{base} using the RV basal programmed stimulation to discern SVT mechanisms. We found that a ΔSA-VA_{base} ≧85

FIGURE 3 The ΔSA-VA$_{base}$ and ΔSA-VA$_{apex}$ Sorted by the Mechanisms and Pathway Location

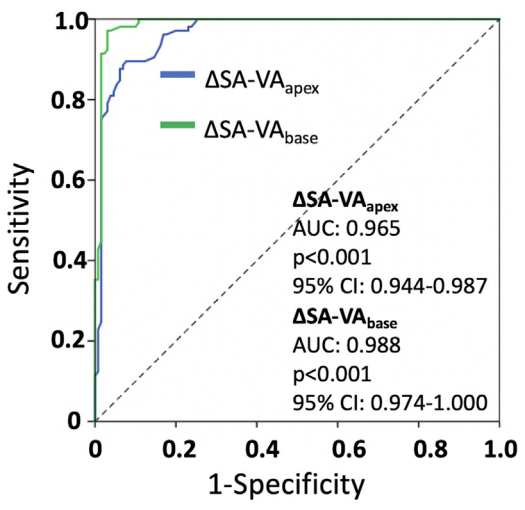


In those with AVRT, the ΔSA-VA$_{base}$ exhibits significantly shorter values than the ΔSA-VA$_{apex}$ for all AP locations except for AVRT with a slowly conducting AP. Note that the ΔSA-VA$_{base}$ shows a better split between those with typical AVNRT and AVRT using left free wall (FW) APs, whereas the ΔSA-VA$_{apex}$ exhibits significant overlap between the 2 groups. The **red line** drawn at 85 milliseconds represents the discriminant point between atypical AVNRT and AVRT using septal APs according to previously published criteria. FW = free wall; other abbreviations as in Figures 1 and 2.

milliseconds more effectively distinguished AVNRT from AVRT (regardless of the AP location) as compared to the ΔSA-VA$_{apex}$ because of a marked shortening of the ΔSA-VA in those with AVRT and the opposite effect in those with AVNRT. In addition, the ΔSA-VA$_{base}$ further allowed for a rapid localization of 4 general AP regions (right free wall or anterior/mid septal, posterior septal, and left free wall APs) with a high sensitivity and specificity.

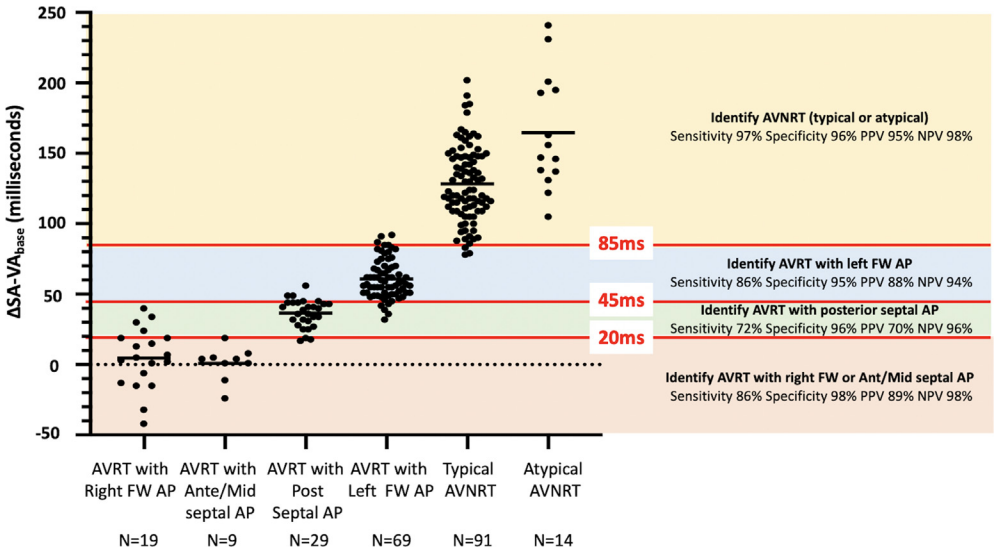
Previous studies have used the difference between RV apical and basal pacing to serve as an ancillary discriminatory maneuver to differentiate AVNRT from AVRT.[3-5,9] For example, Martínez-Alday et al[9] showed that the difference of SA interval between apical and basal pacing during sinus rhythm would be positive when using posterior septal APs and negative in AV nodal pathways. In addition, Khan et al[4] and Segal et al[3] extended this observation to evaluate the difference in the entrainment response from the RV basal pacing in comparison with the RV apical pacing. They demonstrated that the positive values between ΔSA-VA$_{base}$ and ΔSA-VA$_{apex}$ would effectively identify AVNRT. These findings were compatible with our findings. However, we think our study was able to further add 3 important findings.

FIGURE 4 ROC Curve Analyses to Discriminate AVNRT From AVRT Using the ΔSA-VA (V2)



A higher value of the area under the receiver-operating characteristic (ROC) curve is noted for ΔSA-VA$_{base}$ than for ΔSA-VA$_{apex}$. AUC = area under the curve; other abbreviations as in Figures 1 and 2.

FIGURE 5 **Discriminative Performance for the Use of the ΔSA-VA$_{base}$ to Localize the APs**



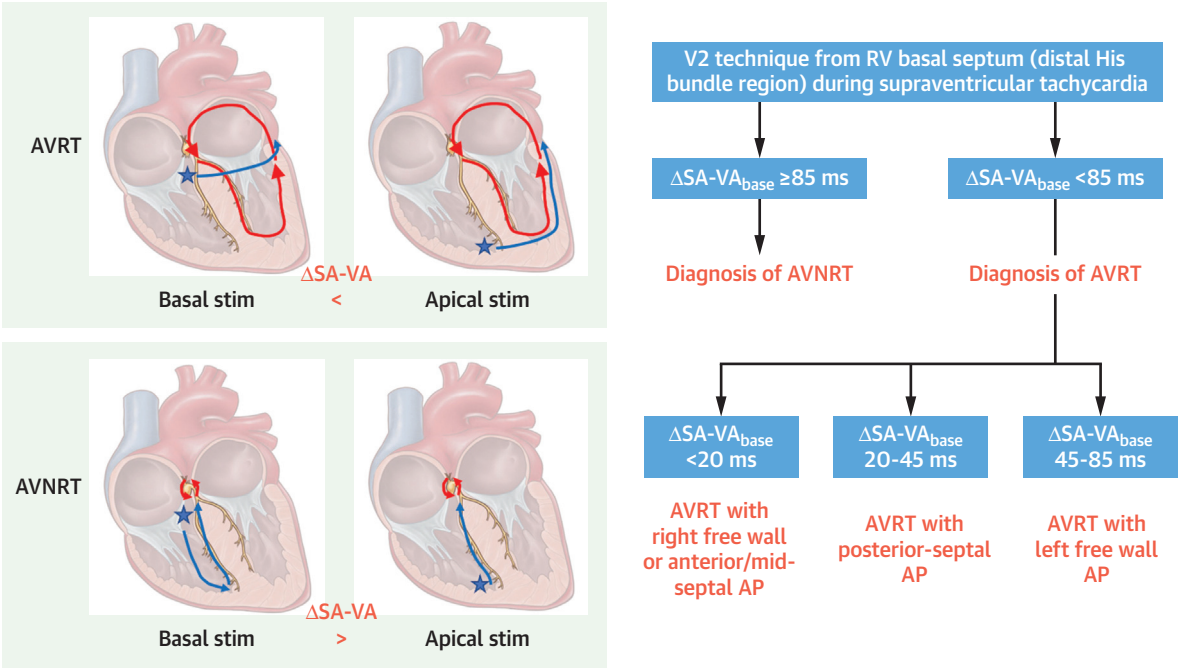
(Left) The ΔSA-VA$_{base}$ is shown according to the supraventricular tachycardia mechanism, and **(right)** the discriminative performance for each group showing the sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV), respectively, is shown. The ΔSA-VA$_{base}$ with a **dashed line** at 85 milliseconds represents the discriminant point between AVNRT and AVRT. The ΔSA-VA$_{base}$ with a **dashed line** at 45 milliseconds represents the discriminant point between AVRT using a left FW AP and posterior septal AP. The ΔSA-VA$_{base}$ with a **dashed line** at 20 milliseconds represents the discriminant point between AVRT using a posterior septal AP and right FW AP or anterior/mid septal AP. Abbreviations as in Figures 1 and 3.

First, our study was the largest series of prospectively looking at the positional difference with the use of ΔSA-VA values. Therefore, we were able to provide clear evidence that basal pacing had more robust discriminative performance than apical pacing. Moreover, previous studies emphasized the importance of measuring the SA intervals from both apex and base to calculate the difference in the differentiation.[3-5] In contrast, our conclusion did not require both basal and apical values to identify AVNRT.

Second, this was the first study using V2 technique instead of ventricular entrainment using ventricular overdrive pacing. The V2 technique that we used in the present study has a lesser chance of SVT termination than VOD does.[1,13] Moreover, a previous report[14] demonstrated that resetting with single or double premature stimuli provides a more accurate assessment of the characteristics of the unaltered tachycardia circuit than that with overdrive pacing.

Finally, the biggest novel finding in the present study was that the ΔSA-VA$_{base}$ (V2) allowed the identification of 4 general AP locations. There has been no report providing a comprehensive technique that allows not only to distinguish AVNRT and AVRT but also to localize such number of general AP sites. Previously, several previous studies have addressed efforts to localize APs by using ventricular entrainment pacing or resetting with programmed ventricular stimulation during AVRT.[1,7,15,16] Weiss et al[16] reported that the ΔSA-VA after a reset with an RV extrastimulus was longer in those with left free wall APs than in those with septal Aps; however, they did not show a clear cutoff value.[13] Miles et al[13] compared the longest coupling interval of an RV extrastimulus that advanced the atrial activation during AVRT to the SVT cycle length (pre-excitation index).[15] They found that the pre-excitation index was useful to distinguish septal APs from left free wall APs, but there was overlap between the 2 groups in 23%. González-Torrecilla et al[7] used the ΔSA-VA$_{apex}$ during entrainment to identify AVRT through a septal versus free-wall AP. They found that a ΔSA-VA$_{apex}$ >50 milliseconds allowed differentiating these 2 AP sites with a sensitivity of 88% and specificity of 83%; however, they did not distinguish the septal versus right free wall region. In the present study, the ΔSA-VA$_{base}$ (V2) allowed for identifying 4 broad AP regions with high sensitivity and specificity. One reason that might explain this result is the distance and conduction time from each AP region to the RV basal septal stimulation site. The distal His-bundle region (RV basal septum) is located in the center of both the left

CENTRAL ILLUSTRATION Significance of the ΔSA-VA_{base}



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(Left) The potential mechanism for the advantage of stimulation (stim) from the right ventricular (RV) basal septum. The ΔSA-VAbase in those with AVRT would be shorter than the ΔSA-VAapex because the SA interval is dependent on the proximity of the stimulating site to the ventricular insertion of the AP. In contrast, the ΔSA-VAbase in those with AVNRT would be longer than the ΔSA-VAapex, because the impulse must first pass from the RV base to the apex to access the right bundle. ΔSA-VA = The difference between the ventricular SA interval with ventricular resetting with premature ventricular complexes and the VA interval during supraventricular tachycardia. **(Right)** Algorithm for the use of the ΔSA-VA_{base} programmed ventricular extrastimuli (V2) during supraventricular tachycardia. AP = accessory pathway; AVNRT = atrioventricular nodal re-entrant tachycardia; AVRT = atrioventricular reciprocating tachycardia; RV = right ventricular; SA = stimulus-atrial electrogram; stim = stimulation; V2 = programmed ventricular extrastimuli; VA = ventriculoatrial.

and right basal regions. Therefore, the 4 general AP areas might represent a better balanced distribution of the distance and conduction time from the stimulation sites that would allow for a clear diagnosis of the pathway location.

In our preliminary study, we found a wide overlap in the ΔSA-VA_{apex} between those with AVNRT and AVRT using left APs.[1] Because the RV apex is remote from a left-sided AP, the SA interval may overlap with that found in AVNRT. The SA interval in those with AVRT was dependent on the proximity of the stimulating site to the ventricular insertion of the AP and consequently was shorter when stimulating from a basal septal site as opposed to an apical site. In contrast, the SA interval from RV basal septum would be longer than that from RV apex over the AV node, because the impulse must first pass from the RV base to the apex to access the right bundle (**Central Illustration**). The ΔSA-VA_{base} exhibited a marked shortening particularly in those with left-sided APs, supporting the advantages of RV basal stimulation in the differentiation of AVNRT from AVRT. Furthermore, based on a previous study, ΔSA-VA_{apex} of ≧85 milliseconds proved useful for differentiating atypical AVNRT versus AVRT with septal AP.[2] Our findings extended those observations to include separation of all forms of AVNRT from AVRT.

CLINICAL IMPLICATIONS. The differentiation of AVNRT from AVRT and the localization of APs are both necessary aspects of an electrophysiological study to guide catheter ablation. However, standard criteria for making these distinctions have limitations. The basal SA-VA may serve as a comprehensive discriminatory maneuver that allows us not only to distinguish AVNRT and AVRT regardless of the pathway location, but also to identify 4 general pathway locations (**Central Illustration**). Moreover, this straightforward technique does not require any knowledge of the location of the earliest atrial activation during SVT. Therefore, this may be of particular value,

for example, in the labs with limited access to catheters because basal SA-VA can be measured by using 2 right-sided diagnostic catheters. This may be also valuable in cases when access to the coronary sinus is difficult or not feasible. In addition, previous studies proposed the use of para-Hisian pacing during SVT to distinguish AVNRT from AVRT.[11,12] However, persistent His-bundle capture during SVT is technically challenging. In contrast, our technique requires only capture of the right basal ventricular myocardium and serves as an easier maneuver to perform in clinical practice. Finally, localizing the detailed septal APs (anterior, mid, and posterior) can be challenging because of the anatomical complexity. In those cases, the ΔSA-VA$_{base}$ may help guide the initial pathway localization.

STUDY LIMITATIONS. Some limitations must be taken into account when interpreting this study. First, there were patients who failed to show tachycardia resetting by basal V2 technique. In the present study, 26.8% of adults and 36.5% of pediatric patients with AVNRT failed to show SVT resetting. We attributed this finding to the more rapid SVT seen in children as well as the increased difficulty in circuit penetration when pacing from the base. In contrast, only 3.5% of adults and 9.6% of pediatric patients with AVRT exhibited failure to reset the SVT and of note all failures occurred in those with left free wall APs. That is not a serious diagnostic issue because the left free wall pathways can be readily distinguished by the eccentric activation patterns. Moreover, even in the instances where V2 fails to penetrate the circuit, one can try to obtain the ΔSA-VA$_{base}$ with ventricular overdrive pacing because entrainment is a result of repetitive reset. Second, there were only a small number of patients with decremental APs and some values of ΔSA-VA$_{base}$ overlapped with those of AVNRT. Therefore, our observations do not extend to those with decremental APs. Third, it is conceivable that a similar analysis involving a large patient group would yield different diagnostic cutoff values as well as performances. However, we found that the results of ΔSA-VA$_{apex}$ during V2 in the present study were quite compatible with those obtained from different samples using the same technique,[1] suggesting that the nature of the sample in the present study was not particularly biased in light of the existing cases. Finally, our analysis allowed for localizing the AP to 4 broad anatomic areas, but more data are required to further refine this technique for determining a more precise AP location.

CONCLUSIONS

This prospective study provided clear evidence that the ΔSA-VA delivered from the RV base has more advantages than that from the RV apex: first, the ΔSA-VA$_{base}$ during V2 produced a robust differentiation between AVNRT and AVRT regardless of the AP location with $\geqq$85 milliseconds as an excellent cutoff point; second, the ΔSA-VA$_{base}$ allowed for a rapid identification of 4 general AP locations (right free wall, ante-mid septal, posterior septal, and left free wall regions) with a high sensitivity and specificity. This straightforward technique may serve as a comprehensive discriminatory maneuver to diagnose SVTs and be of value particularly in labs with limited access to catheters or in situations with limited access to the coronary sinus.

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: The differentiation of AVNRT from AVRT as well as the localization of APs are necessary to guide catheter ablation of SVT. RV basal V2 would enhance the use of the ΔSA-VA for distinguishing AVNRT from AVRT regardless of the AP location. Moreover, the use of the basal V2 allows for a rapid identification of the 4 general AP locations. This straightforward technique may serve as a comprehensive discriminatory maneuver to diagnose SVTs and be of value particularly in labs with limited access to catheters or in situations with limited access to the coronary sinus.

TRANSLATIONAL OUTLOOK: Further studies are required to define the value of ΔSA-VA$_{base}$ to those with decremental APs. In addition, more data are required to further refine this technique for determining a more precise AP location.

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