New Criteria During Right Ventricular Pacing to Determine the Mechanism of Supraventricular Tachycardia

Soufian T. AlMahameed, MD; Alfred E. Buxton, MD; Gregory F. Michaud, MD

Background—Right ventricular pacing (RVP) during supraventricular tachycardia produces progressive QRS fusion before the QRS morphology becomes stable. This transition zone (TZ) may provide useful information for differentiating orthodromic reciprocating tachycardia (ORT) from atrioventricular nodal reentrant tachycardia and atrial tachycardia independent of entrainment success.

Methods and Results—We studied the effect of properly timed RVP on atrial timing during the TZ in 92 patients with supraventricular tachycardia who had RVP within 40 ms of the tachycardia cycle length. The TZ during RVP includes progressively fused QRS complexes and the first paced complex with a stable QRS morphology based on analysis of the 12-lead ECG. We also measured the stimulus-atrial interval from the end of the TZ and with each QRS complex thereafter until pacing was terminated or ventriculo-atrial block occurred. A fixed stimulus-atrial interval was defined as variation <10 ms during RVP. Atrial preexcitation, postexcitation, or supraventricular tachycardia termination with abrupt ventriculo-atrial block was observed within the TZ in 32 of 34 patients with ORT. A fixed stimulus-atrial interval was established within the TZ in 33 of 34 patients with ORT. At least 1 of these 2 responses was observed in all patients with ORT. None of the patients with atrioventricular nodal reentrant tachycardia or atrial tachycardia had atrial timing perturbed or a fixed stimulus-atrial interval established within the TZ.

Conclusions—During RVP within 40 ms of the tachycardia cycle length, ORT is the likely mechanism when atrial timing is perturbed or a fixed stimulus-atrial interval is established within the TZ. (Circ Arrhythm Electrophysiol. 2010;3:578-584.)

Key Words: supraventricular tachycardia ▪ AV node reentry ▪ orthodromic reciprocating tachycardia ▪ accessory pathway ▪ entrainment

Significant diagnostic information may be derived from right ventricular pacing (RVP) that results in entrainment of supraventricular tachycardia (SVT). An “AVV” response after overdrive pacing suggests atrial tachycardia (AT), whereas an “AV” response suggests an atrioventricular (AV) nodal-dependent SVT. When differentiating atypical AV node reentry (AVNRT) from orthodromic reciprocating tachycardia (ORT) using a septal accessory pathway, a stimulus-atrial (SA) minus ventriculo-atrial (VA) interval <85 ms, a corrected postspacing interval (PPI) minus tachycardia cycle length (TCL) of <110 ms and entrainment with stable QRS fusion are highly specific for ORT. However, repeated RVP attempts fail to achieve entrainment in 22% of patients with SVT. Instead, RVP may result in AV dissociation or tachycardia termination, preventing evaluation of the postspacing response. Single ventricular extrastimuli can be scanned throughout the diastolic SVT cycle and their effect on subsequent atrial timing measured to help differentiate among AVNRT, ORT, and AT. This pacing technique does not rely on entrainment success. The diagnosis of ORT is suspected when a His refractory ventricular extrastimulus results in atrial preexcitation and is confirmed when a His refractory ventricular extrastimulus results in atrial postexcitation or abrupt SVT termination without atrial activation. Progressive QRS fusion is usually seen at the beginning of RVP and is the result of collision between the ventricular activation wave front via the AV node and the pacing wave front. Therefore, the significance of fusion QRS complexes is similar to scanned single ventricular extrastimuli delivered when the His bundle is refractory. These fused QRS complexes are identified by a QRS morphology intermediate between native QRS complexes and the paced complex with a stable morphology. The influence of RVP during this “transition zone” (TZ) on various forms of SVT has not been reported.

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Methods

Study Design

We performed a retrospective study of the response of various SVT mechanisms to RVP delivered for the purpose of entrainment.
Patients were included if they had a single SVT mechanism and had RVP within 40 ms less than the TCL. Patients were excluded when SVT exhibited spontaneous oscillation >10 ms within 3 cycles of the onset of RVP (n=16 patients). One patient had repeated ablation for ORT, and only his first procedure was included. Study approval was obtained from the Rhode Island Hospital Institutional Review Board.

**Electrophysiological Procedure**

Electrophysiological tests were performed in the fasting state. The patient’s written informed consent was obtained before sedation. Quadripolar electrode catheters were inserted into the femoral vein and positioned in the high right atrium, right ventricular (RV) apex or base, and the anterosetal tricuspid valve (His bundle recording). A decapolar catheter was inserted into the femoral vein and positioned in the coronary sinus (CS). All 12 ECG leads and intracardiac electrograms were recorded and stored on a digital recording system (Prucka Engineering Inc, Houston, Tex, or Bard Labsystem, Pro EP, Billerica, Mass). Bipolar intracardiac electrograms were filtered between 30 and 500 kHz and recorded from the proximal electrode pair of quadripolar catheters and all pairs of decapolar catheters at speeds of 100 to 200 mm/s. Bipolar pacing was performed at twice the diastolic threshold from the distal electrode pair using a programmable stimulator (Bard Micropace). The onset of RVP was timed to begin on the basis of sensing from the RV catheter so the coupling interval between the last sensed RV signal and the first paced beat approximated the pacing cycle length. Improperly timed RVP trains were excluded from analysis.

**SVT Diagnosis**

The diagnosis of typical AVNRT was made when the VA interval in the earliest intracardiac atrial recording was ≤70 ms and 1 or more of the following criteria were satisfied: (1) presence of anterograde functional dual AV nodal pathways; (2) a midline atrial activation sequence during SVT that matched that during RV pacing; (5) AV block coincident with tachycardia termination; (4) a delta atrial–His bundle (AH) interval ≥40 ms between tachycardia and atrial pacing at the tachycardia cycle length during sinus rhythm; and (5) an “AV” response after entrainment with RVP. The diagnosis of atypical AVNRT was made when earliest VA interval was >70 ms and 1 or more of the following criteria were satisfied: (1) concentric atrial activation pattern; (2) an “AV” response after entrainment with RVP and a PPI-TCL corrected >110 ms. The diagnosis of ORT was made when the earliest VA interval was >70 ms and 1 or more of the following criteria were satisfied: (1) atrial timing was advanced and tachycardia reset, atrial timing delayed or tachycardia terminated without depolarizing the atrium associated with a scanned single premature ventricular extrastimulus that occurred when the His bundle was refractory; (2) the VA interval during tachycardia increased by ≥20 ms with the development of ipsilateral bundle-branch block; (3) an “AV” response after entrainment with RVP and a PPI-TCL corrected <110 ms. Atrial tachycardia was diagnosed by the presence of an “AAV” response after RVP, absence of VA linking (ie, variable AH and VA intervals), changes in H-H or V-V intervals that were preceded by changes in A-A intervals, or AV dissociation with rapid RVP at a cycle length between 200 and 250 ms during tachycardia.

**Characteristics of RVP Trains and Definitions**

RVP was attempted from the RV apex or base. Entrainment was confirmed when the atrial cycle length accelerated to the pacing cycle length (PCL) and the tachycardia resumed after pacing was discontinued. Typically, RVP results in overdrive suppression when the atrial cycle length is accelerated to the paced cycle length in patients with focal atrial tachycardia, but we defined this as “entrainment” in this study. We reviewed the surface ECG for all patients included in this study and classified RV basal pacing when an inferior axis was present. RVP trains were included in the analysis regardless of entrainment success if (1) PCL was 10 to 40 ms shorter than the TCL; and (2) the maximum spontaneous oscillation in TCL within 3 cycles before the RVP train was <10 ms. The TZ of RVP was defined as the region that contains paced complexes showing progressive QRS fusion and the first paced complex showing a stable QRS morphology. The end of the TZ, therefore, usually is a fully paced complex, but this complex may represent constant fusion in some patients with ORT. All 12 ECG leads were inspected to determine the beginning and the end of the TZ in all patients (Figure 1). Atrial preexcitation was defined as atrial cycle length shortening by ≥15 ms during RVP. Atrial postexcitation was defined when atrial cycle length increased by ≥15 ms. Termination without atrial depolarization was defined when SVT terminated with abrupt VA block during RVP. The SA interval was measured at the end of the TZ, from the first paced complex showing a stable QRS morphology and for each subsequent QRS complex until pacing terminated or VA block occurred. (Figure 1A and Figure 2A and 2B). A fixed SA interval was defined as varying by <10 ms. For the purpose of measuring SA intervals, an RVP train that resulted in termination of tachycardia was included if (1) no change in atrial activation sequence was noted before SVT termination and (2) there were at least 3 paced QRS complexes with a stable morphology and VA conduction. When fusion beats resulted in termination of tachycardia during RVP train (3 patients, Figure 3), SA interval was measured during other RVP trains in the same patient.

**Statistical Analysis**

Continuous variables are expressed as mean±SD. Continuous variables were analyzed using 1-way ANOVA. Nominal variables were compared with the Fisher exact test. A probability value ≤0.05 was considered statistically significant. Statistical analysis was performed using SAS 9.1.

**Results**

**Baseline Characteristics**

A total of 92 patients with a history of paroxysmal SVT who underwent radiofrequency ablation at Rhode Island Hospital between January 2006 and April 2009 were included in the study. There were 34 patients with ORT, 28 patients with typical AVNRT, 8 patients with atypical AVNRT, and 22 patients with AT. Accessory pathway locations were left free wall in 23 (68%), septal in 7 (20%), and right free wall in 4 (12%) patients. Baseline patient characteristics are shown in Table 1. RV basal pacing was present in only 6 of 34 patients with ORT, 3 of 28 patients with typical AVNRT, and 1 of 8 patients with atypical AVNRT. Because so few patients had RV basal pacing, statistical comparison of apical versus basal RV pacing was not conducted.

**Frequency of Entrainment and Fusion QRS Complexes With Ventricular Pacing**

**ORT Group**

There were 34 patients with a total of 76 RVP trains in the ORT group, of which 41 (54%) resulted in successful entrainment. Twenty-one of 34 (62%) patients had successful entrainment with any RVP train. Fusion QRS complexes were seen in 66 of 76 (87%) RVP trains and in 31 of 34 (91%) patients during any RVP train.

**Typical AVNRT Group**

There were 28 patients with a total of 68 RVP trains in the typical AVNRT group, of which 51 (75%) resulted in successful entrainment. Twenty-five of 28 (89%) patients had successful entrainment with any RVP train. Ventricular
fusion QRS complexes were seen in 62 of 68 (91%) RVP trains and in 27 of 28 (96%) patients with any RVP train.

Atypical AVNRT Group
There were a total of 8 patients with a total of 15 RVP trains in the atypical AVNRT group, of which 13 (86%) resulted in successful entrainment. All 8 patients had entrainment with any RVP train. Ventricular fusion QRS complexes were seen in 12 of 15 (80%) RVP trains and in 6 of 8 (75%) patients with any RVP train.

AT Group
There were a total of 22 patients in the AT group. Eighteen of 22 (82%) patients had AV dissociation during RVP. AT was the only mechanism of tachycardia in which this was observed during the study. All 4 patients without AV dissociation had successful “entrainment” with at least 1 RVP train. There were a total of 10 RVP trains in this subgroup, and 9 resulted in successful “entrainment.” Ventricular fusion QRS complexes were seen in 9 of 10 RVP trains and in 3 of 4 patients with any RVP train. An AAV response was observed in all 4 patients with “entrainment.”

Atrial Timing Perturbation in the TZ
Patients with typical AVNRT, atypical AVNRT, and AT did not show atrial timing perturbation in the TZ of RVP. Atrial activation was advanced 1 beat after the TZ in 2 patients with AVNRT but occurred most commonly 2 or 3 beats later (Figure 2A). In the ORT group, 32 of 34 (94%) patients demonstrated perturbation of atrial timing during the TZ (Figures 1A and 2B). Fusion QRS complexes during RVP perturbed subsequent atrial timing in 25 of 34 (74%) patients. Fusion QRS complexes resulted in atrial preexcitation in 19 of 34 (56%) patients, atrial postexcitation in 3 of 34 (9%) patients and SVT termination without depolarizing the atrium in 3 of 34 (9%) patients (Figure 3). Relative to accessory pathway location, atrial timing was perturbed with fusion QRS complexes in 16 of 23 (69%) patients with a left free wall accessory pathway, 6 of 7 (87%) patients with a septal AV pathway, and 3 of 4 (75%) patients with a right lateral pathway. No effect on atrial timing was seen with fusion QRS complexes in 9 of 34 (26%) ORT patients. Seven of these 9 patients (5 left free wall, 1 septal, and 1 right lateral AV pathway), however, had advancement of atrial activation ≥15 ms at the last beat of the TZ, that is, the first paced beat with stable QRS morphology. The 2 remaining ORT patients had left free wall AV pathways and had advancement of atrial activation of 10 ms at the last beat of the TZ, which we considered too small a difference to be measured reliably.
Characteristics of the TZs

An ANOVA was used to compare the number of beats in the TZ. The results of this test ($F=2.55$, $P=0.06$) were similar to the results of an ANOVA using a rank transformation of the number of beats in the TZ ($F=3.18$, $P=0.03$). A Tukey post hoc test indicated that the mean number of beats in the TZ was significantly higher in the AVNRT group (4.4) than in the ORT group (3.6; $P<0.05$). No other comparisons between 2 groups were significant.

Fixed SA Interval Established Within the TZ

Fixe d SA Interval in the AVNRT and AT Groups

A fixed SA interval was never established in the TZ in patients with AVNRT or AT (Figure 2A). Termination of SVT with any RVP train was seen in 12 of 28 patients with typical AVNRT, 2 of 8 patients with atypical AVNRT, and none of 22 patients with AT (Table 1).

Fixed SA Interval in the ORT Group

The SA interval became fixed within the TZ in 33 of 34 (97%) patients with ORT (Figures 1A and 2B). One patient with a decrementally conducting right free wall accessory pathway had an increasing SA interval after the TZ.

Combined Criteria of Atrial Perturbation or Fixed SA Interval Within the TZ

All 34 patients with ORT showed either a fixed SA interval or atrial timing perturbation within the TZ when pacing the RV within 40 ms of the tachycardia cycle length (Table 2). Similar to scanned ventricular extrastimuli, these findings are independent of entrainment success. Dissimilar to scanned ventricular extrastimuli, these criteria are not as dependent on accessory pathway location relative to RV pacing site. In most patients with AT, AV dissociation was present during RVP.

Discussion

Main Study Findings

The main study finding was that all patients with ORT had either atrial timing perturbed (94%) or a fixed SA interval (97%) established within the TZ when pacing the RV within 40 ms of the tachycardia cycle length (Table 2). Similar to scanned ventricular extrastimuli, these findings are independent of entrainment success. Dissimilar to scanned ventricular extrastimuli, these criteria are not as dependent on accessory pathway location relative to RV pacing site. In most patients with AT, AV dissociation was present during RVP.
Atrial Timing Perturbation During the TZ of RVP

AVNRT and ORT use the AV node as an integral part of the reentrant circuit. Brugada et al.\(^1\) studied transient entrainment and interruption of AVNRT with RVP trains and concluded that the ventricle is not part of the AVNRT reentrant circuit. In the present study, the TZ of RVP had no effect on atrial timing in patients with AVNRT because paced complexes delivered when the His bundle was refractory did not have access to the AV node. In our study, no bystander APs were present to confound this finding. Our results are consistent with Brugada’s discovery that the ventricle is not part of the reentrant circuit in AVNRT. In contrast, ORT uses an accessory pathway as the retrograde limb of the reentrant circuit and both atrial and ventricular tissue are obligatory components of the tachycardia circuit. Zipes et al.\(^1\) showed that in the presence of an accessory pathway, a single right ventricular extrastimulus administered during SVT at a time when the His bundle is refractory often resulted in preexcitation of the atrium. Ross et al.\(^1\) investigated the effect of His refractory right ventricular extrastimuli during SVT in 99 patients. They found that atrial timing was advanced during SVT in 89% of patients with right-sided AP, 85% of patients with septal AP, and 11% of patients with left-sided APs. Knight et al.\(^1\) studied the diagnostic value of different pacing maneuvers during paroxysmal SVT. Perturbation of atrial timing was seen with right ventricular extrastimuli delivered when the His bundle was refractory in 25% of patients with ORT.\(^9\) Miles et al.\(^1\) developed a preexcitation index to quantify the degree of prematurity required for an RV extrastimulus to result in atrial preexcitation ≥10 ms in patients with ORT. Of 20 patients with left free wall accessory pathways, none had preexcitation when ventricular extrastimuli were delivered within 45 ms of the TCL, 5 within 45 to 75 ms, and the rest >75 ms.\(^1\) The present study shows that tachycardia behavior in the TZ of RVP yields similar information to scanned single His refractory ventricular extrastimuli in patients with ORT. We found that atrial timing was perturbed in 74% of patients with ORT coincident with fusion QRS complexes. The incidence of perturbation of atrial timing by fusion QRS complexes during RVP was

### Table 1. Baseline Characteristics of Study Subjects

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>ORT</th>
<th>AVNRT</th>
<th>AVNRT*</th>
<th>AT</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>34</td>
<td>28</td>
<td>8</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>41 ± 16</td>
<td>50 ± 17</td>
<td>64 ± 13</td>
<td>63 ± 12</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Men, %</td>
<td>68</td>
<td>91</td>
<td>96</td>
<td>75</td>
<td>0.076</td>
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<tr>
<td>TCL, ms</td>
<td>357 ± 74</td>
<td>356 ± 65</td>
<td>442 ± 96</td>
<td>429 ± 112</td>
<td>0.002</td>
</tr>
<tr>
<td>Entrainment, † %</td>
<td>62</td>
<td>89</td>
<td>100</td>
<td>18</td>
<td>0.0001</td>
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<tr>
<td>AV dissociation, %</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>82</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Termination with RVP, † %</td>
<td>48</td>
<td>43</td>
<td>13</td>
<td>0</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Note that patients with ORT were younger and predominantly men, in contrast to the other groups. AV dissociation with RVP was seen only in patients with AT. *Atypical AVNRT.

### Table 2. Responses to RVP Trains Within 40 ms of the Tachycardia Cycle Length in All Types of SVT

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>ORT</th>
<th>AVNRT</th>
<th>AVNRT*</th>
<th>AT</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>34</td>
<td>28</td>
<td>8</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>Fusion complexes, %</td>
<td>91</td>
<td>96</td>
<td>75</td>
<td>75</td>
<td></td>
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<tr>
<td>Atrium affected in TZ, %</td>
<td>94</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Atrium advanced</td>
<td>176</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Atrium delayed</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Abrupt termination‡</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Fixed SA, %</td>
<td>97</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Note that fusion complexes during Tz were very common in all 4 groups. Note that only patients with ORT had either atrial timing perturbed (84%) or a fixed SA interval (87%) established within the Tz with pacing. *Atypical AVNRT.

†First atrial advancement ≥15 ms coincided with fusion QRS complexes in 56% and with first paced QRS with stable morphology in 20% of patients with ORT.

‡Abrupt termination without depolarizing the atrium.
higher in our sample of patients with left free wall APs (69%) than previously reported by Knight, Ross, or Miles with single His refractory ventricular extrastimuli. From our data, we believe consecutive fusion QRS complexes advance the retrograde limb of the ORT circuit more effectively than a single extrastimulus. One plausible explanation is that scanned premature ventricular contractions necessarily produce significant long-short sequences that are more likely to produce significant intraventricular conduction delay, which offsets their prematurity. Furthermore, continuous activation from the paced site may “beat back” the wave front from the intrinsic tachycardia.

Another interesting discovery in this study is that atrial timing is perturbed in 94% of ORT patients within the TZ, which includes the first paced complex with a stable QRS morphology. The 2 patients with minimal atrial preexcitation of 10 ms within the TZ also had a fixed SA interval established within the TZ, suggesting that when the paced cycle length is only slightly faster than the TCL, detection of atrial perturbation may be difficult. This finding argues for pacing between 20 and 40 ms faster than the TCL.

Fixed SA Interval Established in the TZ of RVP

None of the patients with AVNRT or AT had a fixed SA interval established within the TZ of RVP. All but 1 of the 34 patients with ORT had a fixed SA interval established within the TZ. The fixed SA interval reflects a stable decrease of the atrial cycle length to the paced cycle length. The 1 patient with ORT who failed to meet this criterion had a decrementally conducting right sided accessory pathway responsible for an increasing SA interval after the TZ but had the diagnosis of ORT confirmed by atrial preexcitation within the TZ.

Why Is the First Paced Complex With a Stable QRS Morphology Important to Identify?

During ventricular pacing at a rate slightly faster than the TCL, there is a “catch-up” phase during which the pacing train fuses with antegrade ventricular activation until a stable QRS morphology is observed. In patients with ORT, one of the most important findings in our study is that 74% of the patients with ORT have perturbation of atrial timing during progressive fusion, but an additional 20% have atrial advancement at the last beat of the TZ, coincident with the first paced complex showing a stable QRS morphology. This beat may represent the beginning of constant fusion, which would be observed most often in patients with ORT using a septal AP. This represents a stable balance between the paced wave front driving retrograde activation and ventricular activation fusing with antegrade conduction. Alternatively, this beat may appear identical to a fully paced QRS complex, which suggests that AV nodal conduction no longer contributes to ventricular activation. This might be observed in patients with ORT in whom the pacing train results in SVT termination or in whom the AP and pacing sites are remote, for example, patients with left free wall APs and RVP sites. Based on our data, we theorize that the first paced complex identical to the fully paced QRS morphology should drive retrograde activation over an AP regardless of its location, because the antegrade wave front has been completely suppressed by pacing. For patients with AVNRT, we theorize that the tachycardia circuit would rarely be influenced by the first fully paced QRS complex because the retrograde wave front has not penetrated sufficiently beyond the His bundle. In our study, this did not occur until at least the second, but most often the third or fourth paced complex beyond the TZ. Once the retrograde wave front has penetrated beyond the His bundle, it is not clear whether electrophysiological or anatomic properties of the AVNRT circuit influence how late resetting occurs. It is important to remember that all patients included in this study had RV pacing timed properly to produce a TZ and had a paced cycle length within 40 ms of the tachycardia cycle length.

Combined Criteria of Atrial Perturbation or Fixed SA Interval

Neither criterion identified all 34 patients with ORT. In the present study, we observed 1 patient with a decrementally conducting right free wall accessory pathway and an increasing SA interval beyond the TZ of RVP. In this patient, however, atrial timing was advanced within the TZ. Also, if the paced cycle length is within 10 ms of the TCL, atrial timing may be advanced minimally. In this study, we observed atrial advancement of only 10 ms within the TZ in 2 patients; however, a fixed SA interval was also present, allowing detection of ORT. Therefore, all patients with ORT had either a fixed SA interval <10 ms or atrial perturbation ≥15 ms within the TZ of RVP (Table 2).

Potential Pitfalls

This study is retrospective, and the criteria have not been evaluated prospectively. Only cases in which conventional criteria were adequate for diagnosis were included and ambiguous cases were excluded. These criteria may not be useful in patients with SVT showing significant cycle length oscillation, who were excluded from further evaluation. There are possible exceptions to these criteria, such as SVT with a bystander accessory pathway. Another limitation is the small number of subjects in the septal ORT and atypical AVNRT groups, which are often the most elusive diagnoses. Additionally, RVP sites were not uniform, and the choice of ventricular pacing site may influence these results. Identification of the TZ of pacing is based on a qualitative assessment by the operator.

Conclusion

During RVP at a cycle length within 40 ms of the TCL, atrial timing is perturbed in patients with ORT within a TZ that includes QRS complexes showing progressive fusion and the first paced complex with a stable QRS morphology. Unlike atrial perturbation identified with a scanned, single ventricular extrastimulus, these criteria apply to accessory pathways remote from the pacing location. Perturbation of atrial timing ≥15 ms or a fixed SA interval measured from last beat of the TZ was seen in all of the patients with ORT and in none of the patients with AVNRT or AT. These criteria do not depend on entrainment success. Therefore, careful analysis of the beginning of RVP is recommended and may aid in tachycardia diagnosis.
Acknowledgments
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Disclosures
None.

References

CLINICAL PERSPECTIVE
Rapid accurate distinction of atrioventricular nodal reentry from orthodromic atrioventricular reentry tachycardia is critical to successful catheter ablation for these supraventricular tachycardias (SVT). Right ventricular pacing that entrains SVT is often used to make this distinction but is not helpful when pacing fails to entrain or terminates SVT. We developed and tested 2 new criteria for distinguishing these SVTs using right ventricular pacing trains that do not require entrainment. From right ventricular pacing trains, the transition zone is defined from the first QRS complex that is fusion between SVT and pacing, to the first paced complex that has a stable QRS morphology (either completely paced or constantly fused). During the transition zone advance of the stimulus-atrial interval or a fixed stimulus-atrial indicates orthodromic atrioventricular reentry. In 92 patients with SVT of various mechanisms, these criteria showed excellent diagnostic accuracy and could be applied regardless of whether pacing terminated SVT.
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