Physiological Substrate for Antidromic Reciprocating Tachycardia
Prerequisite Characteristics of the Accessory Pathway and Atrioventricular Conduction System
Douglas L. Packer, MD; John J. Gallagher, MD; and Eric N. Prystowsky, MD

Background. Although the anatomic "substrate" for the occurrence of antidromic reciprocating tachycardia (ART) has been previously examined, the underlying physiological substrate for this unusual arrhythmia in patients with the Wolff-Parkinson-White syndrome has not been thoroughly characterized.

Methods and Results. The electrophysiological properties of the accessory pathway and normal ventriculoatrial conduction system in 30 patients with ART and a single accessory pathway were compared with those observed in a control group of 36 patients without this arrhythmia to elucidate the critical physiological substrate essential for the development and maintenance of ART. Inducible ART had a mean cycle length of 286±31 msec. The average retrograde ventriculoatrial conduction system effective refractory period in ART patients was significantly less than that in the control group (244±32 versus 291±46 msec, p=0.0002). All of the ART patients showed retrograde conduction over the normal conduction system at cycle lengths of ≤360 msec; 23 had 1:1 conduction to ≤300 msec, and 16 showed 1:1 propagation at cycle lengths of ≤260 msec. The shortest cycle length accompanied by 1:1 retrograde propagation over the normal conduction system in patients with ART was also significantly less than that observed in the control group (274±39 versus 347±73 msec, p<0.001). The accessory pathway anterograde ERP in ART patients with 1:1 retrograde conduction over the normal ventriculoatrial conduction system at cycle lengths of ≤360 was significantly less than that seen in comparable control patients (247±23 versus 284±56 msec, p=0.001), and the accessory pathway location was significantly further from the atrioventricular node in 21 patients with ART undergoing surgery than that in 22 operated control patients (3.8±0.8 versus 2.9±0.8 mapping units, p=0.0025) who also had retrograde ventriculoatrial conduction to cycle lengths of ≤360 msec. No significant differences in anterograde atrioventricular conduction system properties, retrograde accessory pathway refractoriness, or shortest ventricular pacing cycle lengths maintaining 1:1 conduction via the accessory pathway were observed between groups.

Conclusions. This quantitative characterization of the properties of conduction and refractoriness of both the accessory pathway and ventriculoatrial conduction system and the relationship between these characteristics and the accessory pathway location in ART patients provides additional insight into the prerequisites for the initiation and maintenance of this rhythm disturbance. (Circulation 1992;85:574–588)

Preexcited reciprocating tachycardia, in which an accessory atrioventricular pathway is used as the anterograde limb of an atrioventricular reentrant tachycardia, has been documented clinically in only 4–5% of patients with the Wolff-Parkinson-White (WPW) syndrome but may be in-
of the ART circuit and demonstrated that the accessory pathway was usually located ≥4 cm from the atrioventricular node for the initiation of ART. Furthermore, no patient with a single posteroseptal accessory pathway had ART, which also indicates a requirement of a critical distance between the accessory pathway and the AVCS.

In contrast, the underlying physiological substrate required for ART has been less thoroughly characterized. Previous reports have stressed the importance of atrial premature complex (APC)—induced unidirectional anterograde block in the atrioventricular node, with resolution of this block before the arrival of the return retrograde impulse into the atrioventricular node for the initiation of ART. The requirement of retrograde His–Purkinje delay generated by an ART-initiating APC has also been demonstrated. However, the contribution of this delay, working in concert with the previously described accessory pathway–atrioventricular node distance, has also been recently explored in a pacing model of preexcitation in patients without the WPW syndrome. However, retrograde ventriculoatrial conduction in the model patients was relatively poor. Comparable data in patients with WPW have been unavailable. Therefore, we carefully examined the electrophysiological properties of the accessory pathway and ventriculoatrial conduction system (VACS) in a large number of patients with ART and compared them with those observed in patients without ART to elucidate further the critical physiological substrate essential for the development and maintenance of ART in patients with WPW.

Methods

Patient Population

Of 416 patients with the WPW syndrome and single accessory pathways who had undergone evaluation between 1970 and 1988, 30 (7.2%) demonstrated ART during electrophysiological evaluation and form the basis of the present study. The clinical characteristics, arrhythmia history, and pathway locations are given in Table 1. Of the 30 patients, seven (23%) had a history of clinical preexcited tachycardia, and 22 were included in a prior report of the anatomic substrate of preexcited reciprocating tachycardia. Each patient had a single accessory pathway, with locations as shown in Table 1. No patient with inducible ART had a single posteroseptal accessory pathway. Of the 30 patients with this form of tachycardia, 21 (68%) underwent surgery, during which the accessory pathway location was confirmed by epicardial mapping and the accessory pathway was successfully divided. Of the left free wall pathways, all were located at least 4 cm from the coronary sinus orifice as determined at surgery or calculated at the initial electrophysiology study from the earliest site of retrograde atrial activation indicating the accessory pathway location and both the characteristic cavitary atrial electrogram and typical fluoroscopic position indicating the coronary sinus orifice. Given the known distance of the atrioventricular node to the coronary sinus orifice, these pathways were located at least 5 cm from the region of the atrioventricular node. The right free wall pathways were at least 4 cm from the interatrial septum.

The electrophysiological characteristics of the patients with ART were compared with those observed in a control group of 36 patients chosen from a series of consecutive patients with single accessory pathways, intact anterograde accessory pathway function, and no inducible ART selected from the middle of the total WPW series. A comparison of these patients with a larger series of 312 patients with WPW and single accessory pathways revealed no differences in accessory pathway, reciprocating tachycardia, or atrial fibrillation characteristics. Their clinical characteristics and accessory pathway locations are also shown in Table 1. Patients with single posteroseptal accessory pathways were excluded from the control population because of the absence of similar pathways in patients with ART. Twenty-eight of the control patients (78%) also underwent surgery with complete epicardial mapping. The distribution of left and left posterolateral free wall pathways was similar to that observed in the ART patients, with the exception of one patient with an accessory pathway approximately 4 cm from the atrioventricular node.

By design, all patients in both groups had manifest preexcitation during normal sinus rhythm at the time of initial evaluation.

Study Design

Each patient underwent complete electrophysiological evaluation using standard techniques to determine the location of the accessory pathway, its anatomic relation to the atrioventricular node, and

<table>
<thead>
<tr>
<th>TABLE 1. Clinical and Accessory Pathway Characteristics</th>
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<tbody>
<tr>
<td>Patient characteristic</td>
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</tr>
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<td>Anteroventricle</td>
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<td>Multiple</td>
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ORT, orthodromic reciprocating tachycardia; ART, antidromic reciprocating tachycardia; AF, atrial fibrillation; VF, ventricular fibrillation.

*By design.
the characteristics of the accessory pathway and the AVCS.12-15 Catheters with 1-cm interelectrode spacing were positioned in the coronary sinus, in the right ventricular apex, across the tricuspid valve to record the His bundle electrogram, and in the right atrium. The presence of an accessory pathway was confirmed and its location was determined using observations from the preexcited QRS morphology during sinus rhythm,1 differential atrial pacing,16 and atrial fibrillation,17 and from the response to single and double ventricular premature complexes (VPCs) introduced into orthodromic reciprocating tachycardia (ORT).13,18 In addition, mapping of the right and left atrioventricular grooves during right ventricular pacing and both ORT and ART in the study population and ORT and right ventricular pacing in the control group was performed using a modified Brockenbrough mapping catheter for the right atrioventricular groove and a serial coronary sinus pull-back maneuver for evaluation of the left atrioventricular groove.12

**Antidromic Tachycardia Characteristics**

In the study population, ART was initiated, and the reproducibility of the mode of initiation was determined. The diagnosis of ART was made as previously described2,19 and was based on the presence of 1) a QRS configuration during tachycardia consistent with maximal preexcitation indicating anterograde conduction via the accessory pathway, 2) reproduction of this QRS morphology with atrial pacing at an identical rate as in ART, 3) the presence of a concentric or septal sequence of retrograde atrial activation with the earliest site of atrial depolarization recorded on the His bundle electrogram as seen in Figure 1, 4) persistent 1:1 relation between atrial and ventricular activities during tachycardia, 5) incremental atrial and ventricular pacing and refractory period determination demonstrating that the properties of each limb of the tachycardia circuit were capable of sustaining ART at the observed tachycardia cycle length, 6) exclusion of ventricular
tachycardia, atrial flutter, and atrial reentrant tachycardia, 7) exclusion of tachycardias with left bundle branch block, left-axis deviation with conduction in the anterograde direction via a nodofascicular or atrofascicular pathway, 8) observed changes in the ART cycle length after comparable changes in the ventricle–His (VH) interval, indicating that the retrograde His–Purkinje system was an integral portion of the reentrant circuit,20–23 9) the interruption of tachycardia by a VPC without disturbing the timing of the retrograde His bundle deflection, and 10) exclusion of atrioventricular reentrant tachycardia (AVNRT) with bystander participation of the accessory pathway. This exclusion was based on the APC-induced advancement of the following preexcited QRS complex, the next retrograde His bundle deflection where apparent, and the subsequent atrial deflection, thus indicating a macroreentrant loop.5,24 In two patients with inducible AVNRT at the time of the baseline electrophysiological study, the possibility of AVNRT with a bystander accessory pathway conduction as the mechanism of the preexcited tachycardia was also rejected on the basis of significant differences in the AVNRT and preexcited tachycardia cycle lengths and the absence of any clear transition from narrow to wide complex tachycardias without disturbing the HH intervals.25

**Tachycardia Intervals**

Respective ART component intervals were defined as follows: VH interval, onset of the preexcited QRS complex as observed on the surface ECG to the onset of the His bundle deflection, where visible, as illustrated in Figure 1; HA interval, end of the His bundle deflection to the onset of the earliest retrograde atrial deflection in the region of the low medial right atrium reflecting actual AVN conduction (Figure 1); VA interval, onset of the preexcited QRS to the first rapid atrial deflection on the His bundle electrogram; and interatrial conduction time, onset of the retrograde atrial impulse recorded on the His bundle electrogram, indicating earliest atrial depolarization, to the local atrial activation overlying the accessory pathway origin.

Respective ORT component intervals were defined as follows: AH interval, earliest rapid atrial deflection recorded from the His bundle catheter to the earliest distinct deflection of the His potential; HV interval, the earliest distinct deflection of the His potential to the earliest area of ventricular activation observed on intracardiac or surface ECG tracings; shortest VA interval, onset of the surface QRS or earliest intracardiac ventricular deflection to the earliest retrograde atrial deflection near the accessory pathway; and the interatrial conduction time, earliest local retrograde atrial deflection near the accessory pathway to the end of the atrial deflection recorded from the His bundle catheter.

**Accessory Pathway and Atrioventricular Conduction System Characteristics**

**Pacing protocols.** The anterograde and retrograde characteristics of the normal AVCS and the accessory pathway were determined using standard methods.26 To examine the anterograde (and retrograde) effective refractory periods (ERPs) of the accessory pathway and AVCS, the diastolic interval was scanned with an extrastimulus delivered after an 8-beat drive train of 400-, 500-, or 600-msec pacing cycle lengths near the atrial origin of the accessory pathway or right ventricular apex for accessory pathway retrograde characterization. The basic drive cycle lengths used were comparable in both patient groups. The shortest pacing cycle lengths sustaining 1:1 conduction (shortest 1:1 CL) via either the accessory pathway or the AVCS in the anterograde or retrograde directions were determined during anterograde incremental pacing from a pacing site near the accessory pathway or retrograde incremental pacing from the right ventricular apex, respectively.

**Accessory pathway and AVCS ERP.** Intervals of interest from both extrastimulus testing and incremental pacing were hand digitized, providing a numeric description of the response to each sequential coupling interval or pacing cycle length. These primary data were reviewed independently by two of the investigators (D.L.P. and E.N.P.) to determine whether the anterograde and retrograde ERPs and shortest 1:1 CLs via the accessory pathway or the AVCS could be determined and their numeric values. The overall agreement between observers was 98%.

The longest A₁A₂ coupling interval measured from an atrial site near the origin of the accessory pathway that failed to produce a preexcited ventricular complex was taken as the accessory pathway anterograde ERP. Similarly, the longest A₁A₂ interval measured from the low medial right atrium that failed to conduct via the normal AVCS was taken as the anterograde ERP of the atrioventricular node. Establishment of anterograde block in the atrioventricular node was manifest in the majority of patients by block in the accessory pathway at longer A₁A₂ coupling intervals than that producing block in the atrioventricular node or the shortest A₁A₂ achievable in patients with an accessory pathway ERP greater than atrioventricular node ERP where atrioventricular node ERP determination was limited by the atrial functional refractory period (FRP) of the atrium. In five patients with ART, a minimum estimate of the atrioventricular node ERP was derived from the A₁A₂ interval responsible for initiation of ART, as atrioventricular node block could have occurred at a longer A₁A₂ interval before ART initiation. Block in the accessory pathway disclosing complete atrioventricular block, indicating atrioventricular node block at the same or longer coupling intervals, also provided a minimum possible estimate of the ERP of the AVCS in six control patients. This approach was never applied in ART patients because it would falsely decrease the apparent AVCS ERP. Finally, in one patient with ART without evidence of dual atrioventricular nodal physiology, the atrioventricular node ERP was manifest as a jump in the A₁H₂ interval after gradual prolongation of atrioventricular
node conduction time, followed by a plateau in the relation between $A_2H_2$ and $A_1A_2$, indicating block in the atrioventricular node followed by retrograde His activation. In 12 patients with ART and five in the control group, the atrioventricular node ERP could not be determined or was not assessed due to recurrent atrial fibrillation.

**Shortest atrial pacing cycle length with 1:1 accessory pathway or atrioventricular node conduction.** The shortest atrial pacing cycle lengths resulting in 1:1 preexcited conduction via the accessory pathway (shortest 1:1 CL-AP) was determined. In most cases, this could be directly measured in both ART and control patients or was 230–240 msec or less, the shortest pacing cycle lengths typically used during electrophysiological testing. The shortest 1:1 CL of the atrioventricular node (shortest 1:1 CL-AVN) was more difficult to determine. In the ART patients, this could be estimated in only nine patients and was typically manifest as either abrupt loss of atrioventricular conduction at a shorter pacing cycle length than producing block in the accessory pathway or from the pacing cycle length initiating ART. Accessory pathway block at longer pacing cycle lengths also disclosed the shortest 1:1 CL-AVN at shorter pacing cycle lengths in 12 control patients. In four other patients, accessory pathway block at longer pacing cycle lengths revealed complete atrioventricular block, providing a minimum estimate of the shortest 1:1 CL-AVN. In one patient in the control group, without evidence of dual atrioventricular nodal physiology, the shortest 1:1 CL-AVN was manifest by the jump and plateau of the AH interval indicating block in the atrioventricular node followed by retrograde His activation, as described earlier. Because the ORT cycle length in most patients tested is within 30–40 msec of the atrial pacing cycle length producing Wenckebach block during ORT (unpublished data from our laboratory), the ORT cycle length provided an indirect estimate of the shortest 1:1 CL-AVN in both ART and control group patients.

**Retrograde accessory pathway and VACS ERP.** The retrograde ERP of the accessory pathway and normal VACS and shortest 1:1 CLs with conduction over the accessory pathway or VACS were also determined. The absence of posteroseptal accessory pathways in patients with ART and the exclusion of similar patients from the control group clearly facilitated the differentiation of retrograde conduction via the normal VACS and the accessory pathway. The longest $V_1V_2$ coupling interval that failed to conduct with an eccentric sequence of retrograde atrial activation via the accessory pathway was taken as the accessory pathway retrograde ERP. In some cases, the determination of the accessory pathway ERP was limited by encountering the right ventricular FRP. For the determination of retrograde block in the normal conduction pathway, the VACS was approached as a single component system. Although this did not allow the discrimination between block in either the atrioventricular node or His-Purkinje system, it did allow an examination of retrograde conduction as an all-or-none process required for the completion of the ART circuit. Retrograde block in the VACS was typically established by either the occurrence of block in the VACS at shorter $V_1V_2$ coupling intervals than that producing block in the accessory pathway or the shortest $V_1V_2$ interval resulting in retrograde VACS conduction in patients in whom precise determination of the VACS ERP was limited by the right ventricular FRP.

In patients in whom these indicators of block were not observed, the ERP could be estimated through careful analysis of changes in the retrograde atrial activation sequence occurring during ERP determination. In seven control and three ART patients, the onset of ORT with a VPC provided a minimum estimate of the VACS ERP. Because VACS block could have occurred at longer $V_1V_2$ coupling intervals without ORT initiation, this represents a potential underestimation of the VACS ERP. Retrograde block in the accessory pathway disclosely complete ventriculoatrial block, indicating VACS block at the same or longer coupling interval, similarly provided a minimum estimate of the retrograde VACS. In five patients in the control group and none with ART, the application of both criteria tended to minimize observed differences between ART and control patients. Finally, in two patients in the control group, the VACS ERP was inferred from an abrupt change in the retrograde atrial activation sequence from a concentric sequence or fused to an eccentric sequence accompanied by a $\geq 40–50$-msec prolongation of the VA interval recorded from the His bundle catheter.

Conduction via the accessory pathway precluded determination of the retrograde ERP of the VACS in three patients with ART and seven control patients. The preponderance of such patients in the control group would have contributed to an even longer average VACS ERP, if known. Thus, their exclusion from the analysis was unlikely to produce relevant distortions of the comparisons.

**Shortest right ventricular pacing cycle length with 1:1 VACS or accessory pathway conduction.** The shortest right ventricular pacing cycle length accompanied by 1:1 ventriculoatrial conduction via the accessory pathway or VACS was determined. Retrograde block in the VACS was disclosed in six ART and six control patients after accessory pathway block at longer pacing cycle lengths with accompanying change to a septal sequence of retrograde atrial activation. In three patients with ART and seven patients in the control group, the pacing cycle length producing VACS block was signaled by an abrupt change from a concentric or fused pattern of retrograde atrial activation to an eccentric sequence accompanied by a $\geq 40–50$-msec prolongation of the ventriculoatrial interval recorded from the His bundle catheter. Estimates of the shortest 1:1 CL-VACS were also derived from 1) the 230–240-msec minimum pacing cycle length used for this determination (six ART and one control patient), 2) the ART cycle length (nine ART patients), or 3)
the decremental pacing cycle length resulting in initiation of ORT (one ART and five control patients). In 12 patients in the control group, the shortest 1:1 CL-VACS could not be determined.

**Surgical Intervention**

After laboratory evaluation, 21 ART patients and 28 control patients underwent surgical division of their accessory pathway after detailed epicardial mapping.10,11 None of the patients had atrioventricular node-modifying procedures.28,29 Given the distance of the accessory pathway from the atrioventricular node in the vast majority of cases, all dissection was performed well away from the normal AVCS. Neither patient with an anteroseptal accessory pathway underwent surgery. From 4 to 8 days after surgery, anterograde and retrograde incremental pacing was performed in 18 patients in the study group and 26 of the control population using temporary transthoracic pacing wires placed during surgery to determine the shortest 1:1 CL of atrioventricular or ventriculoatrial conduction, respectively. In some patients, the response to single atrial or ventricular complexes after an 8-beat drive train was also assessed.

**Statistical Analysis**

The significance of differences observed between the patients with ART and the control group with no preexcited tachycardia during electrophysiological evaluation was determined with the Kruskal-Wallace test. A value of $p \leq 0.05$ was considered significant.

**Results**

**Antidromic Tachycardia Intervals**

During electrophysiological testing, ART with a mean cycle length of 286±31 msec (range, 240–355 msec) was initiated in the study population. The local accessory pathway conduction time averaged 52±19 msec (range, 26–125 msec). A readily discernible His bundle deflection was observed in 18 patients. In 12 patients, the His depolarization appeared before or early during the ventricular deflection recorded on the His bundle electrogram as shown in Figure 1. This short VH interval averaging 63±29 msec in these patients suggested retrograde conduction via the bundle branch ipsilateral to the accessory pathway. In the remaining six patients, a long VH interval due to retrograde conduction via the contralateral bundle branch was observed. The mean VH interval of the latter group of 117±27 msec was longer than that observed in short VH interval patients ($p = 0.002$). An example of both long and short VH interval ART from a single patient with a left lateral accessory pathway is shown in Figure 2. There was no correlation between accessory pathway location and VH interval. The average HA interval in all ART patients with an observable His deflection measured 96±42 msec. The average HA of 124±28 msec in the patients with a short VH interval was longer than the 50±9-msec average HA in patients with a long VH interval ($p = 0.0001$). Thus, a reciprocal relation between the VH and HA intervals was present, similar to that observed between the VA and AH intervals in ORT in the presence and absence of bundle branch block ipsilateral to the accessory pathway location. The VA conduction time ranged between 137 and 250 msec (mean, 180±33 msec).

Twenty-six patients also had ORT, with an average cycle length of 322±49 msec (range, 250–450 msec) that was significantly longer than the corresponding ART cycle length ($p = 0.0001$). As shown in Table 2, there was no evidence for directional differences in atrial or ventricular conduction times in corresponding components of each tachycardia circuit to account for this difference in cycle length. During ART, the conduction time from the atrial deflection recorded from the His bundle to the accessory pathway origin averaged 53±24 msec, whereas the conduction in the opposite direction observed during ORT averaged 56±33 msec. Similarly, the 145±34-msec conduction time from the point of local atrial activation near the origin of the accessory pathway to the end of the His bundle deflection during ART was not significantly different from the 147±27-msec sum of the HV and earliest VA intervals during ORT.

In contrast, the atrioventricular node conduction times during ART were shorter than those seen during ORT. The HA interval during ART was compared with the AH interval during ORT in 13 patients with both reentrant arrhythmias and discernible atrial and His deflections in each. Although it might have been anticipated that the shorter ART cycle lengths would be accompanied by longer HA intervals, the mean HA interval of 94±425 msec during ART was less than the average AH interval of 128±49 msec during ORT ($p = 0.077$), explaining in part the observed differences in ART and ORT cycle lengths.

**Components of the ORT Circuit**

The functional characteristics of various components of the ORT circuit are given in Table 3. Of the 18 patients with ART in whom the anterograde atrioventricular node ERP could be estimated, 15 had values of ≤280 msec, whereas eight had anterograde atrioventricular node ERPs of ≥250 msec. No difference in the shortest 1:1 CL-AVN, was observed between patients with and without ART. There also was no difference in the ORT cycle lengths between groups (322±49 versus 324±50 msec, $p = \text{NS}$), used as an estimate of the shortest 1:1 CL-AVN, or the AH interval during ORT (118±46 versus 121±30 msec).

**Components of the ART Circuit**

The anterograde ERP of the accessory pathway determined in 26 patients with ART averaged 247±23 msec, shown in Figure 3, which was significantly less than the 280±53-msec accessory pathway ERP seen in 32 control group patients ($p = 0.0041$). In 10 patients with ART but only two of the control group, the accessory pathway ERP was less than the atrial FRP. In these patients, the atrial FRP was used as an upper-limit indicator of the accessory pathway

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**Table 2**

<table>
<thead>
<tr>
<th>Component</th>
<th>ART Cycle Length (msec)</th>
<th>ORT Cycle Length (msec)</th>
<th>p-value</th>
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<td>AH</td>
<td>96±42</td>
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<tr>
<td>VA</td>
<td>137±25</td>
<td>147±27</td>
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**Table 3**

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<th>Component</th>
<th>ART ERP (msec)</th>
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<tr>
<td>Anterograde</td>
<td>247±23</td>
<td>≤0.0041</td>
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FIGURE 2. Recordings of short and long VH antidromic reciprocating tachycardias in a patient with a left free wall accessory pathway. Top panel: Short VH tachycardia (VH interval, 58 msec) and a presumed HA duration of 40 msec in a patient with a left free wall pathway. The His bundle deflection is observed shortly after the onset of the surface QRS before local ventricular activation, suggesting retrograde His bundle activation via the left bundle branch. The tachycardia cycle length was 215 msec. In contrast, the bottom panel demonstrates a long VH tachycardia with a His deflection inscribed following the ventricular deflection in the His bundle electrogram; the VH interval is 158 msec and is accompanied by a significantly longer antidromic reciprocating tachycardia cycle length (CL) of 335 msec. Shown are surface leads V1 and lead II as well as intracardiac recordings from the right ventricle (RV), right atrium (RA), high interventricular septum (HBE), and proximal coronary sinus (CS) positions. The polarity of the CS recording has been changed between panels and the amplitude decreased.

ERP. The difference between ART and control groups remained significant despite this source of potential overestimation of the anterograde ERP of the accessory pathway in ART patients. The anterograde ERP of the accessory pathway could be compared with the accompanying anterograde atrioventricular node ERP in 18 patients with ART. These values were within 10 msec in two
TABLE 2. Comparison of Directional Conduction Times in 26 Patients With ART and ORT

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<th>Conduction time (msec)</th>
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<td></td>
<td>ART</td>
<td>ORT</td>
<td>p</td>
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<tr>
<td>Tachycardia cycle length</td>
<td>286±31</td>
<td>322±49</td>
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<tr>
<td>HBEA-&gt;AP (atrial origin)</td>
<td>53±24</td>
<td>56±33</td>
<td>NS</td>
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<tr>
<td>AP (atrial origin)-&gt;AVN-H junction</td>
<td>145±34</td>
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<tr>
<td>Trans-AVN conduction (n=12)</td>
<td>96±42</td>
<td>118±46</td>
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</table>

ART, antidromic reciprocating tachycardia; ORT, orthodromic reciprocating tachycardia; HBEA, His bundle atrial electrogram; AP, accessory pathway; AVN, atrioventricular node; H, His bundle deflection.

The difference in the anterograde shortest 1:1 CL-AP between groups was also marked. In the 26 patients with ART in whom this could be determined, the shortest 1:1 CL-AP was 251±28 msec, which was significantly shorter than the 327±115-msec value observed in the 32 control patients (p=0.0004). Similarly, the shortest RR interval between two consecutively preexcited complexes (SRRPE) during atrial fibrillation as an indicator of the propensity for rapid ventricular activation via the accessory pathway ranged between 150 and 305 msec with an average value of 223±58 msec in ART patients, which tended to be shorter than the 255±71-msec mean value (range, 150–405 msec) in control patients (p=0.065). Of the patients with ART, 25% had SRRPEs of >250 msec, whereas 61% had values of ≤220 msec. This is at substantial variance from the 47% of control patients with SRRPEs of >250 msec and the 41% with values of ≤220 msec. Eighteen of the patients (60%) with ART also had a clinical history of atrial fibrillation, and four (13%) had a prior history of ventricular fibrillation. In contrast, although 21 of the control group (58%) had clinically occurring atrial fibrillation, only two (6%) had a prior history of ventricular fibrillation, thus suggesting enhanced ventricular fibrillation risk in ART patients.

FIGURE 3. Scatterplot comparing accessory pathway (AP) antegrade effective refractory periods (ERPs) in 26 patients with antidromic reciprocating tachycardia (ART) and 32 control patients.

In 24 of the ART patients, the mean retrograde VACS ERP was 244±32 msec, which was significantly less than the 291±46-msec average ERP in 27 control patients (p=0.0002), as displayed in Figure 4. The VACS ERP was ≤280 msec in 20 and ≤240 msec in 15 ART patients compared with similar values in only 14 and five control group patients, respectively. In 14 ART patients and only three control patients, the precise value of the retrograde VACS ERP was less than the right ventricular FRP. Thus, the inclusion of these patients in the analysis represents a

TABLE 3. Characteristics of Components of the ORT Circuit

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<th>Control patients</th>
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<tr>
<td></td>
<td>n</td>
<td>Value (msec)</td>
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<tr>
<td>Anterograde ERP-AVN</td>
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<td>257±29</td>
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<td>Shortest 1:1 CL-AVN (anterograde)</td>
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<td>308±46</td>
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<tr>
<td>Retrograde ERP-AP</td>
<td>23</td>
<td>244±31</td>
<td>33</td>
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<tr>
<td>Shortest 1:1 CL-AP (retrograde)</td>
<td>18</td>
<td>252±20</td>
<td>33</td>
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ORT, orthodromic reciprocating tachycardia; ART, antidromic reciprocating tachycardia; AVN, atrioventricular node; ERP, effective refractory period; Shortest 1:1 CL-AVN, shortest pacing cycle length with 1:1 anterograde conduction via the AVN; AP, accessory pathway; Shortest 1:1 CL-AP, shortest pacing cycle length with 1:1 conduction via the AP.
relative overestimation of the actual VACS ERPs in the ART group. Despite this, the observed difference remained highly significant.

The shortest 1:1 CL-VACS was assessed in 16 patients with ART and 22 patients in the control group. The ART group showed retrograde ventriculoatrial conduction to an average pacing cycle length of 262±34 msec (Figure 5), which was significantly less than the 347±73-msec value observed in the control group (p=0.0004). Using the ART cycle length as a more inclusive estimate of the shortest 1:1 CL-VACS, the mean value in all 30 ART patients of 274±39 msec was also significantly less than that observed in the control group (347±73 msec) (p=0.0001). It is noteworthy that all 30 patients with ART had ventriculoatrial conduction during ART or right ventricular pacing to cycle lengths ≤360 msec, 23 had conduction at cycle lengths of ≤300 msec, and 16 showed 1:1 propagation at cycle lengths of ≤260 msec.

Overlap in the shortest 1:1 CL-VACS in ART and control groups underscored the importance of other factors facilitating ART. When the anterograde ERP of the accessory pathway in patients from both groups with shortest 1:1 CL-VACS of ≤360 msec were compared, the average value of 247±23 msec in ART patients was, again, less than the 284±56-msec average seen in overlapping control patients (p=0.001), as shown in Figure 6. This suggests the additional importance of short accessory pathway anterograde ERPs given the rapid retrograde ventriculoatrial conduction in this patient population. Furthermore, the accessory pathways in the 21 patients with ART undergoing surgery were significantly further from the atrioventricular node than those seen in 22 operated control patients with overlapping shortest 1:1 CL-VACS values (3.8±0.8 versus 2.9±0.8 mapping units, p=0.0025).

FIGURE 4. Scatterplot of comparison of the retrograde effective refractory period (ERP) of the normal ventriculoatrial conduction system (VACS). The retrograde ERP VACS in 24 patients with antidromic reciprocating tachycardia (ART) of 244±32 msec was significantly less than the 291±46-msec value observed in 27 control patients. ORT, orthodromic reciprocating tachycardia.

FIGURE 5. Scatterplot of comparison of the shortest right ventricular pacing cycle length (CL) with 1:1 conduction via the normal ventriculoatrial conduction system (VACS). Here, the average shortest 1:1 CL-VACS in 16 patients with antidromic reciprocating tachycardia (ART) of 262±34 msec was significantly less than the 347±73-msec values seen in 22 control patients. This indicator of ventriculoatrial conduction could also be inferred using the ART cycle length in 14 additional patients as an upper limit of the shortest 1:1 CL-VACS (see text for discussion). ORT, orthodromic reciprocating tachycardia.

FIGURE 6. Plots of contrasting anterograde effective refractory period of the accessory pathway (ERP-AP) and location of the AP in epicardial mapping units from the interatrial septum in antidromic reciprocating tachycardia (ART) and control patients with overlapping shortest 1:1 cycle length ventriculoatrial conduction system (CL-VACS). Left panel: In patients with ventriculoatrial conduction to overlapping cycle lengths of ≤360 msec, the anterograde ERP-AP was significantly less in patients with ART than in the control group. Right panel: AP locations in mapping units were significantly further from the septum in ART patients than those observed in control patients.
Initiation of ART

The mode of induction of ART is shown in Table 4. In 16 patients, APCs produced ART, as exemplified in Figure 7, where a single APC blocks in the atrioventricular node, conducts anterogradely down a left freewall accessory pathway, and then returns via the retrograde VACS to complete the circuit. Atrial incremental or burst pacing was required in 10 other patients without APC-inducible ART. There was no evidence of any relation between pacing site and ART initiation. ART was observed in response to rapid ventricular pacing in the absence of any other atrial induction modality in only one patient.

Of the 30 patients with ART, 26 also had inducible ORT (Table 4). Of 12 of these patients in whom ORT was also initiated with atrial pacing techniques, the precise relation between atrioventricular node and accessory pathway ERPs could be determined in eight. In one patient, the atrioventricular node ERP was longer than that of the accessory pathway. Two other patients, in whom the accessory pathway ERP was 5–10 msec longer than that of the AVCS, developed ORT after the introduction of APCs, whereas ART was produced with an APC in one and burst pacing in the other. Interestingly, four of five patients with an anterograde ERP of the accessory pathway that was 10–45 msec greater than the atrioventricular node ERP at the pacing cycle length used for the ERP determination developed ART with atrial burst or rapid incremental pacing.

Postoperative Evaluation

After surgery, 18 of the 21 ART patients and 26 of the 28 control patients underwent repeat electrophysiological evaluation. The time to postoperative testing was similar in the ART and control patients (6.4±1.3 versus 6.7±1.5 days). No evidence of a functional accessory pathway was observed. There was no significant difference in the postoperative anterograde shortest 1:1 CL-AVN observed between groups (324±53 versus 326±66 msec). During retrograde incremental pacing, the shortest 1:1 CL VACS was 289±60 msec in the 17 ART patients with intact ventriculoatrial conduction, which was significantly less than the 439±167-msec value observed in control patients (p=0.0003). The postoperative values noted were not significantly different from those observed in the preoperative study. One operated patient with ART had evidence of dual atrioventricular nodal physiology after surgery but no inducible AVNRT. On long-term follow-up, none of the patients with ART has had a known recurrence of symptomatic reciprocating tachycardia.

Discussion

Although the anatomic substrate of ART has been described,2 a detailed assessment of the electrophysiological properties or physiological substrate in patients with this uncommon arrhythmia was previously
unavailable. The findings of the present study provide a more quantitative characterization of the distinctive characteristics of refractoriness and conduction of both the accessory pathway and VACS in patients with ART and the WPW syndrome. Importantly, the marked difference between these properties and those observed in patients with WPW without inducible ART provides new insight into the prerequisites for the initiation and maintenance of this rhythm disturbance. Furthermore, information from the present study provides an elucidation of the relation among these characteristics, accessory pathway location, and mode of ART initiation.

**Characteristics of the Ventriculoatrial Conduction System**

The comparisons of retrograde ventriculoatrial conduction and refractoriness made between groups disclosed several distinguishing characteristics of patients with ART. Significantly shorter refractoriness in the VACS viewed as a single component system was observed. Such diminished refractoriness, compared with control groups, should facilitate tachycardia development by requiring less “distance” delay or prolongation of conduction times in other parts of the circuit. In theory, any degree of refractoriness in the VACS would not exclude the possibility of completing an antidromic circuit if the pathway were sufficiently removed from the atrioventricular node or conduction time in intervening tissue delayed the arrival of the reentrant impulse into the VACS sufficiently to allow recovery of AVN refractoriness from prior activation. In reality, however, the accessory pathway–atrioventricular node distance is limited, and expected atrial or ventricular conduction times between the accessory pathway and atrioventricular node, in the absence of other cardiac disease are rapid. Despite substantial accompanying right
ventricular conduction slowing and retrograde His–Purkinje system delay in patients with Ebstein’s malformation, the occurrence of ART in this patient population with a single accessory pathway is rare.2

Right ventricular decremental pacing and ART cycle length analysis also revealed enhanced retrograde conduction in the VACS in patients with, compared with those without, inducible ART. All of the patients with ART showed retrograde conduction via the normal VACS at a pacing cycle length of 360 msec, 23 had 1:1 conduction with a 300-msec pacing cycle length, and 16 had conduction with pacing cycle lengths of <260 msec. In addition, little decrement was observed over the course of the incremental pacing. The marked difference in retrograde conduction capability was confirmed at postoperative testing, including that in four patients in whom the VACS could not be preoperatively characterized. Enhanced sympathetic tone after surgery might have facilitated postoperative ventriculoatrial conduction, although this effect should have systematically affected both patients with and without ART. Given these findings and the typical atrial and ventricular conduction properties of WPW patients, it would be less likely for sustained ART to occur in patients with 1:1 conduction via the VACS to only 350–500 msec as previously suggested.6

It should be noted that the VACS in all patients with the WPW syndrome does not in general appear to differ categorically from that in patients without preexcitation. Patients with WPW also show a spectrum of retrograde conduction capabilities.31–33 It is possible that patients with ART represent the more rapid end of the spectrum of ventriculoatrial conduction and refractoriness and that poor retrograde nodal conduction in other patients with WPW contributes substantially to the low occurrence of ART. This facilitated ventriculoatrial conduction has an important additional implication. It is unnecessary to postulate the presence of a second accessory pathway in such patients, even if a retrograde His deflection in ART is not observed. None of the patients undergoing operative dissection of the accessory pathway in the study population had a second pathway observed during surgery, any arrhythmia induced at the final postoperative electrophysiological study, or recurrent tachycardia during follow-up.

Effect of Pathway Location on ART Occurrence

Rapid retrograde conduction via the normal ventriculoatrial conduction system by itself could tend to mitigate against maintenance of ART if the return reentrant impulse encroached upon refractory atrial or accessory pathway tissue. A feature distinguishing patients with ART from those in the control group, however, was the presence of shorter accessory pathway anterograde refractoriness. The maintenance of tachycardia was also facilitated by the significantly greater distance of the accessory pathway from the normal AVCS, as displayed in Figure 6, even in those ART and control patients with equally short retrograde ventriculoatrial conduction times. This additional distance delay contributed to recovery of the accessory pathway before anterograde penetration by the next reentrant impulse.

Contributors to Atrioventricular Nodal Recovery Time in ART Initiation

After anterograde block in the atrioventricular node generated by either an APC or atrial burst pacing, the subsequent arrival of a retrograde impulse into the VACS must be sufficiently delayed to allow recovery of the atrioventricular node from that anterograde block. Such delay may arise from several regions of the prospective ART circuit. One contributor to this additional recovery time is the anatomic or distance delay resulting from the accessory pathway location. In the present study, the left freewall accessory pathways were at least 5 cm from the atrioventricular node. The absence of ART in patients with posteroseptal accessory pathways, as seen here and previously reported,2 provides additional support for the importance of a longer tachycardia circuit. A critical required distance between the atrioventricular node and accessory pathway is also supported by the findings of Lehmann et al.6 In a pacing model of WPW, these investigators produced antidromic echoes in two patients with 150–180-msec programmed atrioventricular delays, simulating conduction times from the atria via the accessory pathway to the right ventricular apex. Importantly, antidromic echoes were never observed with shorter programmed atrioventricular distance delays.

In theory, ART induction may also be facilitated by introducing an APC at a site contralateral to the accessory pathway. The additional distance delay from the APC site to the atrial origin of the accessory pathway might allow additional time for retrograde atrioventricular node recovery from the anterogradely penetrating APC. However, this factor did not appear to contribute to ART induction in these patients.

Findings from the present study also elucidate the theoretical role of His–Purkinje system delay facilitating ART initiation. In an early study, Wellens and Durrer7 presented five patients with WPW in whom retrograde delay in the His–Purkinje system also appeared necessary to facilitate antidromic echoes after APC-induced anterograde atrioventricular node block. The importance of delay in this component of the ART circuit, permitting recovery of the atria or AVCS from the prior anterogradely conducted APC, has also been suggested by others.8 Because we were unable to discriminate between the His–Purkinje system and atrioventricular node components of retrograde ventriculoatrial conduction, the importance of His–Purkinje system delay in these patients could not be established.

Alternatively, it seems reasonable to presume that the increased “effective” His–Purkinje delay accompanying retrograde block into a bundle branch ipsi-
lateral to the accessory pathway followed by transseptal conduction and retrograde return conduction via the contralateral bundle branch may be a more important contributing delay facilitating the recovery of anterograde atrioventricular node block and ART initiation. An example of this delay is shown in Figure 8, where the second, ART-initiating APC is followed by a return VH interval of 115 msec, leading to the onset of ART. The VH interval in patients with the His bundle deflection occurring after the ventricular deflection recorded from the His bundle electrogram, presumably indicating such transseptal conduction and contralateral bundle branch return, was at least 40–50 msec longer than that observed in patients with a His deflection occurring before or early within the ventricular deflection. The importance of this “septal delay” to the initiation of ART is also suggested by the findings of a long VH interval following an initiating APC in the previously published reports of ART initiation.\(^1,4,22,24\) In our experience, the alternative initiation of ART with a short VH sequence with the His deflection preceding the ventricular depolarization is unusual. A later transition to a short VH interval during sustained tachycardia is not uncommon, however, as shown in Figure 8 and indicated by the presence of short VH intervals during ART in 12 of our patients.

Despite the separate treatment of the various contributors, it is evident that initiation and maintenance of ART are critically dependent on the interplay of multiple factors, including accessory pathway location; characteristics of the VACS, including enhanced retrograde conduction; probable transseptal conduction with return via the contralateral bundle branch; and, to a lesser degree, longer anterograde refractoriness of the atrioventricular node than the accessory pathway in response to APCs. Obviously, no one component completely transcends the others in terms of importance in facilitating ART.

**ART Initiation**

ART was initiated in the majority of these patients with an atrial pacing maneuver producing requisite block in the atrioventricular node, and subsequent preexcited conduction to the ventricles via the accessory pathway.\(^4\) In at least 10 patients with ART, however, the measured atrioventricular node ERP, determined with a single APC introduced after an 8-beat drive train, was less than the observed accessory pathway ERP. Nevertheless, ART was initiated in many of these patients with double APCs or brief atrial burst pacing. This may be explained by the differential response of the atrioventricular node and accessory pathway to repetitive pacing. With rapid atrial pacing, a rate-related decline in accessory pathway refractoriness is generated by successive beats of the train or burst.\(^26\) In the atrioventricular node, however, a rate-related prolongation of effective refractoriness would be expected with successive beats of an atrial train\(^35,36\) or consecutive APCs. Shorter pacing cycle lengths may also result in a shift of the atrioventricular node block site to a more proximal location, also enhancing the chance of recovery from atrioventricular node refractoriness before arrival of the retrogradely conducting reentrant impulse. These pacing maneuvers may, therefore, modulate the actual atrioventricular node ERP in a dynamic fashion, allowing the initiation of ART even though the requisite differential refractoriness may not be “apparent” with single APC testing. Changes in autonomic tone during the course of a study may also variably affect atrioventricular nodal refractoriness during ERP determination and ART initiation.

In this series, no patient developed ART with a single VPC, although four had inducible ART with brief ventricular burst pacing. Although the initiation of ART with a single VPC is possible,\(^3,7,8,15,37\) it is much less common than with an APC, particularly if only a single pathway is present.\(^2,3\) The infrequency of a VPC mode of initiation may be related to multiple factors, including a shorter retrograde ERP of the accessory pathway than that present in the VACS and the propensity for retrograde block in the His–Purkinje system with single VPCs or brief right ventricular burst pacing.\(^38,39\) The latter condition would favor exclusive retrograde conduction via the accessory pathway leading to the development of ORT as seen following single VPCs or brief right ventricular burst pacing in these patients with ART. It may also reflect persistence of retrograde concealment into the accessory pathway preventing return of a subsequent atrial impulse via the accessory pathway to complete the ART circuit.

**Study Limitations**

Several limitations should be considered in interpreting the present study. In patients with WPW, the presence of the accessory pathway could confound the characterization of normal conduction system properties. In the majority of both ART and control patients, however, the characteristics of the accessory pathway and AVCS could be determined precisely or were limited only by the achievement of atrial or ventricular FRPs. In addition, by taking advantage of 1) the absence of posteroseptal accessory pathways, allowing a straightforward differentiation of concentric retrograde conduction via the VACS from the eccentric sequence of retrograde atrial activation via an accessory pathway; 2) the initiation sequences of ART and ORT; and 3) the ORT and ART cycle lengths as indicators of normal anterograde and retrograde atrioventricular conduction system capabilities, respectively, it was possible to characterize qualitatively, if not completely quantitatively, the properties of the VACS in most of the remaining patients. Furthermore, estimates of VACS and accessory pathway characteristics were included in statistical comparisons only if they tended to minimize the significance of differences between groups.

The actual level of block in the retrograde VACS could not be determined with the methods used in
the present study. Therefore, with the exception of the estimates of block inferred from the initiation of ORT, where retrograde block was presumed to be within the His–Purkinje system, the retrograde ERPs or shortest 1:1 CLs of the VACS reflect the characteristics of the weakest component of the pathway without specifying the precise level of block. The absence of this information, however, does not negate the overall description of the entire retrograde pathway used in the ART circuit.

An additional limitation of the present study is the inability to determine the time to recovery of retrograde atrioventricular nodal refractoriness created by an anterogradely conducted APC. The recovery time could, therefore, only be inferred from the conduction time from the low medial right atrial region through the accessory pathway and retrogradely through the His bundle during ART.

Finally, because AVNRT with bystander accessory pathway conduction may mimic ART, it is critical to eliminate this possibility as the actual mechanism for the observed preexcited tachycardia. No evidence of AVNRT or dual atrioventricular nodal physiology was detected in 28 of the 30 patients at the time of the baseline electrophysiological study. Two medically treated patients with ART did have inducible AVNRT at the baseline evaluations. Neither showed a transition between narrow complex and preexcited tachycardias without disturbing the HH interval, and both had shorter cycle lengths during ART than during narrow complex AVNRT. An additional patient showed dual atrioventricular nodal physiology at the time of postoperative electrophysiological testing without inducible AVNRT. More importantly, no patient developed AVNRT during follow-up, even though no surgical procedure was directed at the atrioventricular node or perinodal region.

Furthermore, the possibility of this alternative mechanism was excluded in a majority of patients by the response to APCs interpolated into ART. Late APCs introduced at the atrial origin of the accessory pathway when the atrium near the atrioventricular node was refractory resulted in preexcitation of the subsequent ventricular complex via the accessory pathway, advancement of the next His bundle deflection, and retrograde preexcitation of the next atrial depolarization on the His bundle electrogram, yielding subsequent ART advancement. A similar result with such a late coupled APC introduced at a site well removed from the atrioventricular node in AVNRT is unlikely, as also suggested by the unlikely occurrence of retrograde atrial advancement following an analogously timed VPC introduced during AVNRT.

Despite these considerations, this comparison of patients with and without ART provides new insight into the conditions required to facilitate ART. In addition to an accessory pathway location removed from the AVCS, patients with ART have enhanced retrograde conduction and shorter refractoriness of the VACS. These factors work in concert with the accessory pathway location to facilitate the initiation and maintenance of ART. These patients also demonstrate more rapid accessory pathway anterograde conduction and refractoriness and, therefore, may be at higher risk for sudden cardiac death.

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References


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