The Excitable Gap in Atrioventricular Nodal Reentrant Tachycardia
Characterization With Ventricular Extrastimuli and Pharmacologic Intervention
Claudio D. Schuger, MD, Russell T. Steinman, MD, and Michael H. Lehmann, MD

Our purpose was to characterize the excitable gap during atrioventricular nodal reentrant tachycardia (AVNRT) to elucidate the electrophysiologic substrate of this clinically familiar microreentrant arrhythmia. Accordingly, in 11 patients with classic slow-fast AVNRT (mean cycle length, 342±41 msec), a single ventricular extrastimulus (V2) was periodically delivered after a spontaneous tachycardia beat (V1) until ventricular refractoriness was reached. With this technique, an excitable gap was considered present when atrial preexcitation of at least 20 msec could be achieved along with tachycardia resetting (noncompensatory pause after V2). The range of V1V2 intervals that resulted in atrial preexcitation constituted the preexcitation zone. Five patients (45%) showed evidence of an excitable gap at baseline, with a maximal atrial preexcitation achievable of 33±6 msec, representing 9±1% of the tachycardia cycle length. Verapamil was then administered to all 11 patients with the purpose of slowing the anterograde tachycardia wavefront before arrival of V2. This resulted in widening of the preexcitation zone in three patients by a mean of 50±37 msec, with a corresponding increase in maximal atrial preexcitation to 70±32 msec, or 16±4% of AVNRT cycle length, and the appearance of atrial preexcitation in two patients who lacked it during baseline. In the remaining six patients, AVNRT was not sustained after verapamil or was too unstable for evaluation. During baseline, V2A2 conduction time increased by only 5±3 msec throughout the preexcitation zone, with values at the outer border unchanged after verapamil, implying a fully excitable gap in the retrograde limb. In all patients with a preexcitation zone, AVNRT was consistently reset by V2, both at baseline and after verapamil, with a “flat” but mainly “increasing” response pattern as V1V2 was shortened. Hence, a significant number of patients with AVNRT have evidence of an excitable gap whose demonstrability can be facilitated by pharmacologic intervention; documentation of an increasing resetting response pattern, most apparent after verapamil, provides new evidence for a reentrant mechanism in AVNRT; and while not definitively proven, the presence of a fully excitable gap during AVNRT is most consistent with a microreentry circuit that incorporates an anatomic obstacle. (Circulation 1989;80:324–334)

Atrioventricular nodal reentrant tachycardia (AVNRT) is one of the most common types of paroxysmal supraventricular tachycardia. The small size of the AV node, however, limits precise characterization of the tachycardia mechanism, so that the widely assumed reentrant substrate has been difficult to document in a definitive manner.1–3 The reentrant process is believed to result from longitudinal dissociation of the AV node, typically into a slow anterograde and a fast retrograde limb,4–6 both of which appear connected to lower and (probably) upper common nodal pathways.7,8 Whether this dissociation simply reflects functional heterogeneity of nodal tissues or also involves the presence of an anatomic obstacle is unknown.9–18 However, it remains an especially relevant concern in light of recent reports of surgical ablation of the arrhythmia.17–19

One aspect of AVNRT that has important bearing on this issue but also is not well understood is the nature of the excitable gap (i.e., the extent of electrical recovery achieved by tissues within the microreentry circuit during sustained AVNRT). Whereas the “leading circle” type of functional reentry implies an excitable gap consisting of only partially recovered tissue, circuits incorporating an
anatomic obstacle are the most well established to have an imposed increment in conduction time that may permit the existence of a fully excitable gap during tachycardia.20

Investigators have previously used single premature ventricular extrastimuli to elicit atrial preexcitation and resetting phenomena during ongoing reentry in well-defined macroreentry circuits,21-24 attesting to the presence of an excitable gap in these settings. Such an approach also has been useful diagnostically in some published cases of AVNRT,6,25 but concomitant data relating to the nature of the excitable gap during this microreentrant arrhythmia have rarely been reported.26

In the present study, we systematically used single ventricular extrastimuli in association with pharmacologic intervention during AVNRT to demonstrate and characterize the excitable gap and to investigate resultant resetting responses. Our findings shed new light on the electrophysiologic substrate of AVNRT and may have implications for other putative microreentry circuits in man.

Methods

Electrophysiologic studies were performed in the postabsorptive, nonsedated state after all antiarrhythmic medications were discontinued for at least five half-lives and written informed consent had been obtained. Transvenous quadripolar and/or octopolar electrode catheters were introduced percutaneously and positioned in the high right atrium (HRA), in the right ventricular apex, across the tricuspid valve in the region of the His bundle, and in the coronary sinus. Pacing was performed at twice-diastolic threshold with a programmable stimulator (Bloom Associated Ltd, Reading, Pennsylvania). Surface electrocardiographic leads I, II, aVF, and V1 as well as intracardiac recordings filtered at 30–500 Hz were recorded on magnetic tape (Honeywell 101) and on paper (Siemens Mingograf) at a speed of 100 mm/sec.

Inclusion Criteria

Patients were included in the study if sustained AVNRT (slow-fast type) with a stable cycle length (not exceeding a variability of ±10 msec) could be induced during electrophysiologic studies. The induced tachycardia was considered compatible with classic AVNRT when there was an AH/HA ratio of more than 3.7, a normal retrograde atrial activation sequence,27 a VA interval of less than 95 msec measured at the HRA,28 and no evidence of an accessory AV connection.

Study Protocol

After induction of AVNRT, the tachycardia was allowed to continue for at least 1 minute to achieve stability of cycle length, after which time single

**FIGURE 1.** Delineation of the atrial preexcitation zone by ventricular extrastimulation during atrioventricular nodal reentrant tachycardia (patient 1). Tracings (from top to bottom) are electrocardiogram lead II, high right atrium (HRA), coronary sinus (CS), His bundle electrograms (HB), and right ventricle (RV). Panel **A:** During AVNRT (A1A1, 420 msec), atrial preexcitation by 20 msec (A1A2, 400 msec) is achieved by introducing a premature ventricular extrastimulus (V1) at a coupling interval (V1V2) of 250 msec, demarcating the outer border of the preexcitation zone. Panel **B:** At a shorter coupling interval (V1V2, 230 msec), the degree of atrial preexcitation is increased (A1A2, 380 msec), corresponding in this case to the inner border of the preexcitation zone. Panel **C:** The ventricular effective refractory period is achieved at a 10 msec shorter coupling interval (220 msec). Note that in Panels **A** and **B,** the return cycle (A2Ar) exceeds A1A1 but is less than fully compensatory—indicating that the V1 impulse reset the tachycardia.
ventricular extrastimuli (S2)—giving rise to V2—were introduced after every eight tachycardia beats (V1) at progressively shorter (10 msec) coupling intervals (V1V2), beginning in late diastole and continuing until ventricular refractoriness was encountered. (The terms S2 and V2 will be used interchangeably due to the lack of increased S2V2 latency in the course of our study.) During tachycardia, atrial preexcitation was said to be present whenever shortening in the atrial cycle length (i.e., A1A2<A1A1) was achieved in response to a suitably timed V2. The atrial response at each V1V2 coupling interval was evaluated at least three times to ensure reproducibility. Using the above technique, the following terms were defined (Figure 1).

**Excitable Gap.** This was considered to be present whenever atrial preexcitation of at least 20 msec was elicited at one or more critical V1V2 intervals without any change in retrograde atrial activation sequence. We recognize that this definition is valid only after the V2 impulse is demonstrated to have truly penetrated the circuit rather than have simply activated the atria retrogradely via bystander AV nodal tissue. Evidence for such impulse penetration emerged from an analysis of the resetting responses (see below).

**Outer Border of the Preexcitation Zone.** This is the longest V1V2 interval associated with atrial preexcitation of at least 20 msec.

**Inner Border of the Preexcitation Zone.** This is the longest V1V2 interval associated with the shortest achievable A1A2.

**Maximal Atrial Preexcitation.** This is the difference between A1A2 and the shortest achievable A1A2 (max ΔAA).

**V1A2 Interval.** The retrograde conduction time measured from the onset of the S2 ventricular stimulus to the first rapid deflection of the HRA electrogram, for a given V1V2 within the preexcitation zone, is called the V1A2 interval.

**Return Cycle.** This is the interval from the preexcited atrial electrogram (A2) to the next atrial electrogram (Ar) measured at the HRA. Although preferably the H1Hr interval should be used,29 the retrograde H2 deflection was always obsvrved by the V2 electrogram in this series.

**Resetting of AVNRT in Response to V2.** This is considered to have occurred if the sum of A1A2 plus A2Ar (return cycle) was at least 10 msec less than two AVNRT cycle lengths (i.e., the pause after the preexcited atrial deflection was less than fully compensatory). This is to be distinguished from the term “resetting response pattern,”29 which is used to describe the relation between the return cycle and V1V2 throughout the preexcitation zone.

During tachycardia, after the whole diastole was scanned and the presence or absence of an excitable gap was determined at baseline, all patients were infused with intravenous verapamil (1–2 mg over 1 minute repeated at 3–5-minute intervals; total dose not exceeding 10 mg) during tachycardia to prolong the tachycardia cycle length by at least 20 msec. After stability of the cycle length was achieved, ventricular extrastimuli were again delivered starting in late diastole with 10-msec decrements until ventricular refractoriness. The presence of an excitable gap, outer and inner preexcitation zones, and characteristics of the return cycle were again assessed.

**Statistical Analysis**

All values are given as mean±SD. Student’s paired and unpaired t test and nonparametric Spearman’s correlation coefficient analysis were used when applicable. A p value of less than 0.05 was considered statistically significant.

**Results**

Eleven consecutive patients presenting with AVNRT satisfied the inclusion criteria and were included in our study. There were two men and nine women (mean age, 47±20 years). The mean tachycardia cycle length for the entire group was 342±41 msec (range, 290–420 msec), mean AH was 301±37 msec (range, 245–380 msec), and HA was 41±13 msec (range, 20–65 msec). The mean AH/HA ratio was 8±3 (range, 4.7–13.5) and the mean VA interval (at the HRA) was 48±21 msec (range, 0–80 msec).

**Atrial Preexcitation in AVNRT During Baseline Study**

As indicated in Table 1, five patients exhibited atrial preexcitation during the baseline study (patients 1–5; Figure 1). The mean tachycardia cycle length in patients with compared to those patients without evidence of an excitable gap was slightly longer, but the difference did not reach statistical significance (364±40 vs. 324±32 msec; p=NS). Also, there was no difference in the AH/HA ratio between the two groups. The V1V2 interval, preexcited atrial interval (A1A2), and conduction time of the ventricular extrastimuli (V3A1, measured at the HRA level) for both the outer and inner borders of the preexcitation zone are provided in Table 1. In every case, the inner border of the preexcitation zone at baseline corresponded to the shortest achievable V1V2 before the ventricular effective refractory period. Thus, with the V2 technique, the full extent of the excitable gap could not be defined because of ventricular refractoriness. However, the fact that the maximal preexcitation achievable (max ΔAA) in the five patients with evidence of atrial preexcitation at baseline was 33±6 msec (range, 25–40 msec) established a minimum estimate for the excitable gap duration, namely, 9±1% (range, 8–11%) of the tachycardia cycle length.

**Verapamil Administration**

After completion of the baseline study, verapamil was administered during the ongoing tachycardia to all 11 patients. After a mean dose of 5.8±3 mg (range, 2–10 mg) verapamil, AVNRT cycle length prolonged by a mean of 60±34 msec (range, 20–105
TABLE 1. Responses to Ventricular Extrastimulation During Atrioventricular Nodal Reentrant Tachycardia Before and After Verapamil

<table>
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<tr>
<th>Patient</th>
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<td>preexcitation zone</td>
<td>msec</td>
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All values given in msec.
AVNRT, atrioventricular nodal reentrant tachycardia; CL, cycle length; preexcitation, (atrial) preexcitation; Max ΔAA, maximum difference between A1A2 and A1A2; VERP, ventricular effective refractory period.

* After verapamil, AVNRT was not sustained.
† After verapamil, AVNRT CL was unstable.

msec) in five patients, while in the remaining six patients AVNRT could no longer be sustained or became unstable (Table 1). Prolongation of tachycardia cycle length was due almost exclusively to prolongation of the A1H1 interval (59±34 msec) with H1A2 unchanged in four cases and increasing by only 5 msec in the remaining one (patient 7). After cycle length stability was achieved, the V2 extra-stimulus protocol was repeated.

Verapamil-Induced Changes in the Preexcitation Zone

Of the five patients in whom the tachycardia was sustained after verapamil, three (patients 1–3) had evidence of atrial preexcitation during baseline studies. The outer border of the preexcitation zone after verapamil was extended by a mean of 50±37 msec (range, 10–100 msec) in all three patients (i.e., atrial preexcitation was now elicited at a longer V1V2 coupling interval) (Figure 2). The inner border was still limited by the ventricular effective refractory period, which increased by a mean of only 14±5 msec (range, 10–20 msec) owing to the longer tachycardia cycle lengths after verapamil. As a result, the overall preexcitation zone became wider (Table 1). Moreover, the maximal atrial preexcitation achievable after verapamil in these patients was 70±32 msec (range, 45–115 msec) or 16±4% of the tachycardia cycle length, representing increases of 33±30 msec (range, 5–75 msec), or 6±4% (range, 1–11%) of the tachycardia cycle length, respectively, from the baseline values.

The remaining two patients (6 and 7) had no atrial preexcitation during the baseline study but, after verapamil, showed atrial preexcitation induced by V2 (with maximal achievable preexcitation of 65 and 25 msec, respectively), indicating the presence of an excitable gap (Figure 3).

For the three patients in whom the preexcitation zone widened after verapamil administration, the increase in the zone’s outer border (∆V2A2) correlated well with the increase in A1H1 (∆A1H1) of the tachycardia, as depicted in Figure 4. For the two patients who manifested atrial preexcitation only after verapamil, ∆V2A2 was calculated by subtracting the V1V2 interval at the outer border of the preexcitation zone (after verapamil) from the ventricular effective refractory period at baseline. When these values were included with the other plots of ∆V2A2 against ∆A1H1, all five points fell close to the line of identity (Figure 4), suggesting that in the last two cases, the shortest V2A2 achievable during baseline studies was just beyond the outer border of the preexcitation zone (which was then extended by verapamil).

V2A2 Conduction Time Within the Preexcitation Zone

To assess the extent of electrical recovery of the retrograde pathway throughout the preexcitation zone, retrograde V2A2 conduction times during baseline and after verapamil administration were measured. For the whole population with evidence of an excitable gap, V2A2 conduction time from outer to inner borders of the preexcitation zone increased by a mean of only 5±3 msec (range, 0–10 msec) during baseline and by 13±7 msec (range, 5–25 msec) after verapamil. Figure 5 shows a representative example of such a flat response. V1V2 intervals are plotted against V2A2 conduction time. During the baseline study, there is no V2A2 prolongation over a 40-msec preexcitation zone. After verapamil administration, there is only a 10-msec increment in V2A2 over a 100-msec preexcitation zone. The V2A2 values at
TABLE I. (Continued)

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<th>Inner border preexcitation zone</th>
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the outer border of the preexcitation zone before and after verapamil were essentially identical in the three patients (1–3) in whom they could be compared. In no case of atrial preexcitation (either before or after verapamil) did \( V_2 \) suddenly fail to activate the atria before attainment of ventricular refractoriness.

Return Cycle and Tachycardia Resetting Response Patterns

Throughout the preexcitation zone, before and after verapamil, the return cycle (\( A_2\overline{A}_r \)) was longer than the tachycardia cycle length (except in two patients where it was not prolonged at the outer border). At the same time, \( A_2\overline{A}_r \) was never fully compensatory, indicating that the \( V_2 \) impulse consistently reset the tachycardia (Figures 1–3). Specifically, during control studies, the return cycle at the outer and inner borders of the preexcitation zone exceeded the tachycardia cycle length by 6±4 msec (range, 0–10 msec) and 10±3 msec (range, 5–15 msec), respectively. After verapamil, \( A_2\overline{A}_r \) at the outer and inner borders exceeded a now-prolonged \( A_1A_1 \) by 6±4 msec (range, 0–10 msec) and 27±10 msec (range, 15–40 msec), respectively.

To assess the return cycle dynamics with increasing degrees of prematurity, investigators\(^{29,30}\) have plotted the return cycle against the coupling intervals of extrastimuli introduced during a tachycardia. In this way, several specific resetting response patterns have been defined, namely "increasing," "decreasing," "flat," or "mixed."\(^{29}\) During baseline studies, both flat and mildly increasing patterns were observed, although due to the very short duration of the preexcitation zone (16±8 msec; range, 10–30 msec), the mean overall change in \( A_2\overline{A}_r \) did not reach statistical significance (Figure 6). After verapamil administration, however, the preexcitation zone became significantly broader (68±32 msec; range, 10–100 msec) and, when plotted against \( V_1V_2 \), a definite increasing \( A_2\overline{A}_r \) response

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Extension of the outer border of the preexcitation zone by verapamil (Patient 1). Tracings (from top to bottom) are electrocardiogram lead II, high right atrium (HRA), coronary sinus (CS), His bundle electrograms (HB), and right ventricle (RV). Same patient as in Figure 1. Panel A: Introduction of \( V_2 \) at a coupling interval of 350 msec fails to elicit atrial preexcitation at baseline. Panel B: After verapamil administration (5 mg), ativoventricular nodal reentrant tachycardia cycle length prolongs to 525 msec. A premature ventricular extrastimulus (\( V_2 \)) at the same coupling interval (350 msec) now succeeds in eliciting atrial preexcitation (\( A_1A_2 \), 500 msec), demonstrating an extension of the outer border of preexcitation zone by 100 msec compared with Figure 1A. Panel C: Introduction of \( V_2 \) at a shorter coupling interval (290 msec) elicits not only a greater degree of atrial preexcitation but also marked prolongation of the return cycle (\( A_2\overline{A}_r \)) to 565 msec compared with that observed at an even shorter coupling interval before verapamil (Figure 1C). Note that the \( A_2\overline{A}_r \) intervals in both Panels B and C are not fully compensatory.
could be identified in all patients studied (Figure 6). The delays in the return cycle as well as the increasing response pattern, both before and after verapamil, always could be localized to the slow anterograde limb of the tachycardia circuit (A2Hr), as HrAr never exceeded H1A1.

Discussion

Although presumed by its very name to be reentrant in nature, proof of the mechanistic basis for AVNRT remains indirect, relying on accumulated clinical and experimental observations. In the present study, ventricular extrastimuli introduced during ongoing AVNRT resulted in atrial preexcitation and tachycardia resetting—both of which were facilitated by verapamil administration—in more than half the cases of AVNRT studied. These findings not only provide further support for a reentrant mechanism in AVNRT but, more specifically, also point to the existence of a fully excitable gap during this common microreentrant tachycardia.

Tachycardia Resetting in AVNRT: Evidence That Atrial Preexcitation Resulted From Penetration of an Excitable Gap by the V2 Impulse

Atrial preexcitation during ongoing AVNRT has been observed occasionally by others using a V2 extrastimulus6,25,26 or, more readily, with a train of ventricular impulses.31,32 The ability to demonstrate this phenomenon is a necessary but not sufficient condition, however, for the existence of an excitable gap (or a reentrant mechanism); the atria could conceivably be activated retrogradely via bystander AV nodal tissue distinct from the site of origin of the tachycardia. To prove that atrial preexcitation indeed resulted from penetration of an excitable gap, it must be demonstrated that the same externally applied stimulus that elicited preexcitation also reset the tachycardia in a manner consistent with reentry. In the present study, whenever atrial preexcitation was induced by a single V2, we were able to induce a non–fully compensatory return cycle (A2Ar), indicating that the tachycardia was reset. Because resetting can occur in triggered and automatic as well as reentrant rhythms,33–35 investigators have searched for specific resetting response patterns that may differentiate between the two. Recent observations suggest that “increasing” or “mixed” (“flat” plus increasing) patterns of resetting are more typical of known reentrant tachycardias23 compared with either a “decreasing” pattern (seen with triggered activity)33–35 or a flat pattern (nondiagnostic).
FIGURE 4. Plot of increase in A1H1 after verapamil versus the outer border of preexcitation zone. ■, Patients who showed widening of the preexcitation zone; ▲, patients with new appearance of a preexcitation zone after verapamil; ---, line of identity. (See text for additional details.)

In our study, both flat and increasing response patterns were observed during baseline but with the very short preexcitation zones that were manifest, it was not possible to characterize fully a specific resetting pattern.29 After verapamil administration, however, we consistently observed an increasing response pattern. The fact that this type of response was more clearly seen after verapamil most likely reflected the latter's effect on a reentrant circuit (expressed over a broader preexcitation zone compared with baseline) rather than a pharmacologically mediated transformation of the expected resetting pattern in triggered activity. This can be inferred from observations in an experimental model of triggered activity where the fundamental relation between pacing cycle length and afterdepolarization amplitude (ultimately responsible for the resetting pattern) was not qualitatively altered by verapamil.36 Hence, it is reasonable to conclude that the occurrence of atrial preexcitation by V2 with attendant tachycardia resetting (in a manner compatible with reentry) signified the presence of an excitable gap during AVNRT.

Demonstrability of an Excitable Gap During AVNRT: Role of Pharmacologic Intervention

Figure 7 schematically depicts the status of the circulating AVNRT wavefront with its tail of absolute refractoriness (dashed line) at various times as viewed from the vantage point of the V2 impulse as it attempts to penetrate the circuit. In Panel A, at a V1V2 coupling interval of X, access to the circuit is preempted by the circulating impulse. At an earlier time (X−Δ1), V2 reaches the distal entry site of the circuit ahead of the circulating impulse, and finds the retrograde limb recovered (Panel B), thereby defining the outer border of the preexcitation zone (and onset of the excitable gap accessible to V2). The shortest achievable V1V2 (X−Δ2) associated with atrial preexcitation is typically limited by ventricular refractoriness and constitutes the inner border of the zone (Panel C).

We hypothesized that if an adequate delay could be achieved in the anterograde propagation of the tachycardia wavefront, the excitable gap could become accessible (and atrial preexcitation elicited) at a V1V2 coupling interval that failed to do so before that anterograde delay was achieved (conversion from Panel A to Panel D). Indeed, just such an effect occurred in three patients with atrial preexcitation at baseline and persistent, but slower, AVNRT after verapamil: the outer border of the preexcitation zone was extended (by Δ1 in Panel D compared with Panel B). Overall duration of the preexcitation zone widened because the ventricular effective refractory period, which limited the inner border, changed minimally (Panel F). This finding clearly indicated a greater accessibility of V2 to the excitable gap, but it cannot be stated whether the gap itself actually widened after verapamil because ventricular refractoriness prevented delineation of the true inner temporal border of the excitable gap (at some time earlier than X−Δ2).

Patients who did not exhibit atrial preexcitation at baseline tended to have shorter AVNRT cycle lengths than those who did, confirming previous observations.25 The fact that an atrial preexcitation zone appeared in two of these patients after verapamil suggested the possibility that the excitable gap had been inaccessible in these two cases at baseline because ventricular refractoriness supervened (before V2 could arrive at the distal entry point of the circuit ahead of the anterogradely propagating impulse). This situation is similar to conversion from Panel A to Panel D in Figure 7,
where X would now represent the shortest achievable V₂V₂ coupling interval. That such unmasking of a previously "hidden" excitable gap truly occurred is supported by the identical manner in which both this phenomenon and extension of the outer border of the preexcitation zone were quantitatively associated with verapamil-induced delays in anterograde conduction during AVNRT (Figure 4). Although unmasking seems to be the most likely explanation for occurrence of preexcitation only after verapamil, the possibility of an excitable gap somehow being "created" by an inherent property of the drug cannot be absolutely excluded.

**Coexistence of a Fully and Nonfully Excitable Gap During AVNRT**

V₂A₂ delays were absent or minimal over a wide range of coupling intervals during AVNRT before and after verapamil, with virtually identical V₂A₂ values at the outer border of the preexcitation zone before and after the drug. Moreover, H₂A₁ during tachycardia was essentially unchanged by verapamil, as noted previously. These findings collectively suggested complete electrical recovery of the retrograde limb of the AVNRT circuit (and, presumably, lower and upper common pathways) at the outer border and throughout most of the preexcitation zone. A previous case report is in agreement with our results. Such observations during ongoing tachycardia are analogous to previous findings that the retrograde fast pathway usually exhibits minimal, if any, decremental conduction characteristics during rapid ventricular pacing.

Within the preexcitation zone, the occurrence of progressive abbreviation of A₁A₂ in response to V₂ (beginning at a coupling interval some 20 msec shorter than A₁A₁), provided a means of assessing anterograde limb excitability during AVNRT. We observed that at relatively long A₁A₂ coupling intervals, there was a mild delay in anterograde pathway conduction (expressed as A₂Hr prolongation), which typically increased as A₁A₂ was abbreviated (most strikingly after verapamil) and yielded an increasing resetting response pattern. Two possible interpretations of this delay may be considered: the premature orthodromic (A₂) wavefront engendered by V₂ encountered an incompletely recovered anterograde limb or prior antidromic penetration by V₂ retarded anterograde propagation of A₂. The first explanation seems much more plausible because distal antidromic penetration by V₂ into the slow pathway (whether associated with intact retrograde conduction or block before arrival of the A₁ impulse), if anything, would be expected to facilitate subsequent anterograde conduction—for any given A₁A₂—by a "peeling back" mechanism. Hence, the frequent occurrence of A₂Hr delays signified that the time period of full excitability within the anterograde pathway during AVNRT was either nonexistent or certainly shorter than that of the retrograde limb, particularly after verapamil (when A₁H₁ was already prolonged).

The observed differences in extent of electrical recovery between the retrograde and anterograde limbs of the circuit during AVNRT indicate that the nature of the excitable gap (full vs. partial) can vary dynamically over the course of each reentrant cycle—a phenomenon that may be exaggerated after pharmacologic intervention.

Our finding of a fully excitable gap during AVNRT in patients with a preexcitation zone effectively rules out the possibility of a functional "leading circle" mechanism in which the reentrant pathway is the shortest one possible and the excitable gap, if present, can consist only of partially recovered tissue. Although the reentrant substrate still could be functional in nature, presumably related to anisotropic conduction,
is not yet known if this mechanism plays a significant role in clinical microreentry circuits. Recent computer and experimental models of anisotropic reentry have been shown to exhibit only partially excitable gaps (M.A. Allessie, personal communication). Given our current state of knowledge, therefore, the finding of a fully excitable gap in AVNRT favors a circuit incorporating an anatomic obstacle over all known models of purely functional reentry. This conclusion cannot necessarily be drawn in cases of the patients who did not demonstrate a preexcitation zone, and it in no way excludes a role for functional heterogeneity and anisotropy of AV nodal tissue in contributing to the reentrant process.

**Limitations**

In six patients, verapamil administration resulted in nonsustained or unstable tachycardia that prevented testing for the potential presence, widening, or unmasking of an excitable gap. Use of alternative agents that could be more efficiently titrated or with shorter serum half-lives might overcome this limitation. Moreover, use of a stimulation site closer to the AVNRT circuit or delivery of double ventricular extrastimuli would also likely increase the
“yield” of atrial preexcitation\textsuperscript{45,46} and allow broader preexcitation zones for the study of unmasking phenomena as well as return cycle dynamics.

As stated above, a retrograde H\textsubscript{2} could not be discerned within the V\textsubscript{2} electrogram; consequently, an H\textsubscript{2}H\textsubscript{r} interval, as a more precise measure of the return cycle, could not be directly assessed. We chose A\textsubscript{2}Ar (on the HRA electrogram) as an indirect measure of the return cycle in light of the constancy of relation of the various atrial electrograms associated with A\textsubscript{1}, A\textsubscript{2}, and Ar. Use of A\textsubscript{2} Ar also offered a distinct advantage over V\textsubscript{2}V\textsubscript{r} because V\textsubscript{2}H\textsubscript{r} and H\textsubscript{r}V\textsubscript{r} are unlikely to be identical,\textsuperscript{47} whereas both A\textsubscript{2} and Ar are orthodromically generated. Moreover, A\textsubscript{2}Ar provided a lower limit estimate for the true return cycle registered at the distal entry point of V\textsubscript{2} into the circuit; consideration of this interval obviated the problem of possible asymmetric retrograde compared with anterograde lower common pathway conduction inherent in using H\textsubscript{2}H\textsubscript{r}. Hence, an increasing A\textsubscript{2}Ar pattern of necessity implied an increasing response in the true return cycle.

**Clinical Implications**

Although not directly proven, our findings suggest that AVNRT is often due to reentry along a discrete anatomically defined microreentry circuit. Thus, our observations can provide an appealing theoretic framework for the interpretation of recent reports of successful surgical or catheter ablation of the tachyarrhythmia.\textsuperscript{17–19,48,49} Finally, the ability to actively influence an excitable gap by pharmacologic means, as demonstrated herein, supports the use of such an approach for investigating other elusive reentry circuits in humans.\textsuperscript{50,51}

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