Atrioventricular nodal reentrant tachycardia:
studies on upper and lower ‘common pathways’

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ABSTRACT Electrophysiologic studies were performed in 28 patients with documented atrioventricular (AV) nodal reentrant supraventricular tachycardia (SVT) to investigate the presence of AV nodal tissue situated between the tachycardia circuit and both the atrium (upper common pathway, UCP) and the His bundle (lower common pathway, LCP). All patients demonstrated a 1:1 AV relationship during SVT. The study protocol consisted of atrial then ventricular pacing at the SVT cycle length. UCPs were manifested in eight of 28 (29%) patients by either antegrade AV Wenckebach (six patients) or a paced atrium-His (AH) interval exceeding the AH in SVT (two patients, differences 5 and 9 msec). LCPs were manifested in 21 of 28 (75%) patients by either retrograde Wenckebach periodicity (two patients) or a paced HA interval exceeding the HA in SVT (19 patients, mean difference 25 ± 20 msec). By these criteria, eight patients (29%) had evidence for both UCPs and LCPs. UCPs were more likely than LCPs to be manifested by Wenckebach criteria (p < .05). Thus (1) the AV nodal reentrant SVT circuit appears to be intranodal and is frequently surrounded by AV nodal tissue (UCP and LCP), (2) antegrade and retrograde conduction properties of these common pathways are discordant in some cases, and (3) conduction properties of UCP tissue differ from those of LCP tissue. These findings may have relevance in that the UCP or LCP may limit the ability of premature extrastimuli to penetrate the circuit to initiate or terminate AV nodal SVT.


ATRIOVENTRICULAR (AV) nodal reentry is one of the most common forms of paroxysmal supraventricular tachycardia (SVT).1 Available evidence suggests that the reentrant circuit consists of a slowly conducting antegrade limb and a rapidly conducting retrograde limb, contained mostly or entirely within the AV node. Experimental studies, as well as isolated case reports and small clinical series, have suggested the presence of additional AV nodal tissue (“common pathways”) extrinsic to the tachycardia circuit.2–13 These studies have shown that neither atrium nor ventricle are required for continuation of tachycardia, in at least some cases. In the present study we used a pacing protocol to identify and characterize these so-called upper (atrial side) and lower (His-bundle side) common pathways in a large group of patients with documented AV nodal reentrant tachycardia.

Methods

Patients. The patient population consisted of 28 individuals with a history of clinical episodes of SVT who underwent electrophysiologic studies, at which time the mechanism of tachycardia was determined to be AV nodal reentry by previously described criteria.14 There were 15 women and 13 men with a mean age of 53 years (range, 16 to 78).

Study protocol. Electrophysiologic studies were performed with patients in the postabsorptive state with no or mild sedation. No patients had been taking antiarrhythmic drugs for at least five half-lives before electrophysiologic testing. Four No. 6F quadripolar catheters with a 0.5 cm interelectrode distance were inserted percutaneously and advanced to the high right atrium, proximal coronary sinus, proximal AV junction (AVJ), and right ventricular apex or mid septum. The position of the AVJ catheter was adjusted to obtain the most proximal His-bundle recording with a 0.5 cm interelectrode distance. This was in all instances positioned so that a retrograde His deflection could be observed during ventricular pacing. Filters for intracardiac electrograms were set at 40 to 500 Hz. Surface leads I, aVF, and V1 and all intracardiac signals were recorded on magnetic tape and on paper (Mingograph) at a speed of 200 to 250 mm/sec.

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The pacing protocol consisted of initiation of SVT with rapid atrial pacing or programmed atrial premature depolarizations. Tachycardia was allowed to continue at least 2 min to ensure stability of cycle length, at which time the tachycardia was terminated by rapid atrial pacing or programmed atrial premature beats. Thereafter the high right atrium was paced at the tachycardia cycle length for 1 to 2 min, and after an interval of at least 1 min the right ventricular apex was paced at the tachycardia cycle length. If pacing at this cycle length produced antegrade or retrograde Wenckebach periodicity, the cycle length of pacing was gradually increased in 10 msec increments to the point of 1:1 AV or VA conduction. The longest paced cycle length that did not result in 1:1 conduction was recorded as the Wenckebach cycle length; pacing was repeated at least once and usually twice to confirm these measurements. After these pacing maneuvers, the tachycardia was reinitiated to ensure reproducibility of cycle length. Adjustment of the AVJ catheter position was sometimes necessary to obtain an adequate His-bundle deflection during ventricular pacing. In these cases, the entire pacing protocol was repeated with the new catheter position. In each case, the mean tachycardia cycle length before and after pacing differed by no more than 10 msec, and cycle length variability during tachycardia was less than 20 msec (beat to beat). The complete pacing protocol was performed in all patients. In some patients, single and double atrial and/or ventricular premature depolarizations were delivered during tachycardia in an attempt to dissociate the atria or ventricles from the tachycardia.

Measurements
Upper common pathway (UCP). We hypothesized that, during atrial pacing at the tachycardia cycle length, the same course of impulses would be taken within the AV node as during tachycardia (slow pathway antegrade, lower common pathway if any, and His bundle). Thus, in the absence of UCP tissue within the AV node, the time from the point where the paced impulse entered the AV node to the onset of the His deflection (AHpace, measured from the end of atrial electrogram to the onset of the His electrogram in the AVJ recording) should equal the AH during tachycardia (AHVT, measured from the onset of the atrial electrogram, where the impulse presumably leaves the AV node to enter atrial tissue, to the onset of the His deflection). If AHpace exceeded AHVT, this would indicate the presence of tissue between the upper portion of the tachycardia circuit and the atrium (UCP) (figure 1). Additionally, atrial pacing at the tachycardia cycle length resulting in AV Wenckebach would imply the presence of UCP tissue, since impulses should traverse the same course in the node during pacing or tachycardia once the antegrade limb is entered.

Lower common pathway (LCP). Analogously, we hypothesized that in the absence of tissue between the lower portion of the circuit and the His-bundle (LCP), the course of impulses taken through the AV node would be the same during tachycardia and ventricular pacing at the same cycle length. Consequently, in the absence of tissue between the lower portion of the circuit and the His-bundle (LCP), the time from the His deflection to atrial deflection during ventricular pacing (HAVT, measured from the end of the His deflection, where the impulse presumably enters the AV node retrogradely, to the onset of the atrial electrogram) should be the same as the HA interval during tachycardia (HAVT, measured from the onset of the His deflection where the impulse presumably exits the AV node during tachycardia, to the onset of the atrial deflection). If HApace exceeded HAVT, this would indicate the presence of tissue between the lower portion of the tachycardia circuit and the His bundle (LCP; see figure 2). Finally, if ventricular pacing at the tachycardia cycle length resulted in His-A Wenckebach cycles, this could occur only in LCP tissue, since the course of impulses taken within the AV node is otherwise the same during pacing and tachycardia.

In this study, all measurements were made in the proximal AVJ recording, which in all cases had the earliest atrial activation during ventricular pacing and tachycardia. Measurements were made on at least three sequential cycles of pacing or tachycardia at 200 to 250 mm/sec paper speed, with a high degree of reproducibility. The onset of the atrial electrogram in the AVJ recording during tachycardia was determined by (1) comparison of the AVJ electrogram morphology between sinus rhythm or atrial pacing and tachycardia or (2) when this was inconclusive, separation of the local ventricular electrogram from the atrial electrogram in the AVJ recording by introducing programmed ventricular premature beats during tachycardia, which did not reset the tachycardia (see figure 3). The atrial deflections in the high right atrium and proximal coronary sinus recordings were always clear and were used to corroborate measurements during pacing and tachycardia in some cases. The presence of UCP or LCP was defined as differences in AH or HA intervals, respectively, between pacing and tachycardia of at least 5 msec, or if Wenckebach periodicity was observed.

![FIGURE 1. Ladder diagram depicting the use of atrial pacing to assess a UCP. Diagrams on the left show events during SVT; those on the right show atrial pacing at the SVT cycle length. Top (no UCP), Atrium, AV nodal (AVN) tachycardia circuit and His bundle are as shown. During VT the impulse travels antegrade down a slow AV pathway, retrogradely up a fast pathway. The AH interval measured as shown by dotted lines is 350 msec. During atrial pacing at SVT cycle length, the impulse takes the same course antegrade as in SVT (slow pathway); the AH measured as shown is also 350 msec. Bottom, An upper common pathway of AVN tissue has been inserted between the AVN circuit and atrium (stippled area). In SVT, the AH as measured above is now 320 msec, although the time spent in the slow pathway is unchanged. During atrial pacing at the same cycle length, the AH as measured above is now 380 msec or 60 msec more than SVT. (See text for further explanation.)](http://circ.ahajournals.org/doi/abs/10.1161/01.cir.75.5.931?journalCode=circ)
Statistical analysis was performed with Fisher’s exact tests with a significance level of $p < .05$.

**Results**

All patients had readily inducible, hemodynamically tolerated AV nodal reentrant tachycardia by previously established criteria. The mean tachycardia cycle length in this series was $374 \pm 70$ msec (range 260 to 520 msec); the mean $AH_{\text{SVT}}$ and $AH_{\text{AVN}}$ intervals were $316 \pm 57$ and $58 \pm 40$ msec, respectively. Individual values for these and other measurements are shown in table 1.

**Upper common pathways.** Eight patients (29%) had evidence of UCPs, indicated by (1) AV Wenckebach at a slower paced rate than the tachycardia rate in six cases (mean difference in the slowest paced rate producing Wenckebach and the tachycardia rate, $19 \pm 12$ beats/min) and (2) $AH_{\text{pace}} > AH_{\text{SVT}}$ in two cases (differences of 5 and 9 msec). An example of analog recordings illustrating this difference in AH intervals is shown in figure 4. Other responses to atrial pacing at the tachycardia cycle length included (1) $AH_{\text{pace}} < AH_{\text{SVT}}$, suggesting the impulse was conducted antegrade in the “fast” pathway (15 cases) and (2) repeated initiation of tachycardia with each attempt at atrial pacing (two cases). These responses precluded a determination of the presence or absence of UCP tissue. Of 11 patients in whom a determination could be made, eight (73%) met criteria for UCP.

**Lower common pathways.** Twenty-one (75%) patients had evidence for LCPs; this was indicated by (1) VA Wenckebach at a slower paced rate than the tachycardia in two cases (mean difference in Wenckebach versus tachycardia rates, $12 \pm 1$ beats/min) and (2) $HA_{\text{pace}} > HA_{\text{SVT}}$ in 19 cases (mean difference in HA intervals, $25 \pm 20$ msec). An example of analog recordings illustrating differences in HA intervals between pacing and tachycardia is shown in figure 5. In three cases, the difference between $HA_{\text{pace}}$ and $HA_{\text{SVT}}$ was less than 5 msec; in the remaining three cases, no clear His deflection was present during ventricular pacing despite repeated adjustments of catheter position, thus no determination of the presence of LCP tissue could be made. Of the 25 patients in whom a determination could be made, 21 (84%) had evidence for LCP.

Eight patients (29%) had evidence for both UCP and LCP. Of note, the presence of a UCP was significantly more likely to be manifested by AV Wenckebach as opposed to the AH criterion, compared with instances in which LCP tissue was detected, which was manifested more frequently by the HA criterion than VA Wenckebach ($p < .05$, Fisher’s exact test).

**Other evidence for the presence of common AV nodal pathways.** In addition to the specific criteria used in this study, several other observations during AV nodal reentrant tachycardia suggest the presence of common AV nodal pathways. These include, first, the occurrence of an atrial echo before the inscription of the His electrogram, suggesting the presence of an LCP. Second, the sudden failure of resetting of a tachycardia after initial resetting by progressively premature extrastimuli suggests that the circuit was not penetrated due to block of the premature impulse in a UCP (figure 6). Finally, an unexpected delay (“retarding”) of the first postpacing electrogram after extrastimuli during tachycardia would suggest concealed conduction within a common pathway (figure 7). Dissociation of both atrium and ventricle from the tachycardia without affecting any tachycardia intervals during constant pac-
Discussion

AV nodal reentrant tachycardia has been recognized as a distinct clinical entity since the early 1970s. The physiologic substrate permitting reentry, functional separation of two pathways within the AV node, has been indicated by the frequent presence of discontinuous AV nodal refractory curves with atrial extrastimulus testing. It was not initially clear how much of the AV node participated in the reentrant tachycardia circuit, or if the two pathways were joined at the upper (atrial) or lower (His-ventricular) aspects into common intranodal pathways. Experiments in dog and rabbit hearts concerning the mechanism of nonstimulated ventricular echo beats suggested that (1) functional longitudinal dissociation of the AV node into at least two pathways was quite prevalent and (2) the two pathways were generally joined at the lower end into a "final common pathway" with reentry occurring in the upper AV node ("N" region). These studies were less conclusive concerning the presence of an upper common AV nodal pathway, and many authors postulated that there was no such pathway but that at least some atrial tissue formed a necessary link in the reentrant circuit.

Several clinical reports suggested that neither atrium nor ventricle were essential components of the tachycardia circuit. These reports included cases in which spontaneous 2:1 block or Wenckebach occurred during tachycardia (in both antegrade and retrograde directions) as well as cases in which premature extrastimuli or rapid pacing were used at electrophysiologic study to capture large portions of the atria (including that surrounding the AV node–AVJ recording) and all
of the ventricles without affecting the tachycardia. Several investigators have reported isolated cases of both antegrade and retrograde Wenckebach periodicity occurring at a paced cycle length greater than tachycardia cycle length during electrophysiologic testing, without commenting on its possible significance.17-19 Akhtar et al.17 used ventricular extrastimulus testing to determine retrograde refractory periods of the AV node in patients with several forms of SVT and found that patients with the typical ("slow-fast") form of AV nodal reentry consistently had longer retrograde conduction times (\(H_2A_2\)) when corrected for tachycardia cycle length, than the \(HA_{svt}\). Wah et al.,13 in a preliminary report, used closely spaced electrodes to record proximal His potentials during tachycardia and ventricular pacing at the tachycardia cycle length and

![Figure 4](image-url)  

**FIGURE 4.** Atrial pacing used to detect UCPs. Abbreviations are as in figure 3. A, Sinus rhythm, for comparison of electrogram morphologies (A, H, and V as shown) in HBE recordings. B, SVT, 380 msec cycle length. Arrows mark points of measurement of AH interval, which is 319 msec. C, Atrial pacing at SVT cycle length. The His deflection is superimposed on the atrial electrogram; AH measured between arrows is 328 msec, 9 msec more than during SVT. These and other analog recordings traced from original. (See text for further explanation.)

![Figure 5](image-url)  

**FIGURE 5.** Ventricular pacing used to detect LCPs. Abbreviations and format are as in figure 3. A, SVT is present at a cycle length (CL) of 330 msec. The HA as measured between arrows is 25 msec. B, Ventricular pacing at the SVT cycle length. HA as measured between arrows is 40 msec, or 15 msec more than during SVT. A vertical dotted line denotes the onset of atrial activity in the HBE recording. Note the similarity of atrial electrogram morphology (SVT vs pacing) in each recording, as well as the identical sequences of atrial activation. C, A sinus beat is shown for comparison of electrogram morphology in the HBE recording. (See text for further explanation.)
TABLE 1

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Mean ± SD 53 ± 15 374 ± 70 316 ± 57 58 ± 40

SVT-CL = supraventricular tachycardia cycle length; eg = electrogram; W = Wenckebach occurred during pacing at SVT-CL.

^aAH pace < AH_{svt}, indicating conduction in fast pathway.
^bAH pace = AH_{svt}, indicating conduction in slow pathway.
^cAtrial pacing always induced SVT.
^dNo His deflection visible during pacing.

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found evidence for LCP tissue in five of nine patients studied (HA_{pace} > HA_{svt}). Despite this body of information, there have been no large systematic studies to date in which a prospective evaluation of the presence of upper and lower common pathways has been undertaken. The current study demonstrates that (1) upper and lower common pathways are frequently present in patients with typical AV nodal reentrant tachycardia, (2) these pathways frequently have discordant antegrade and retrograde conduction characteristics, and (3) upper and lower common pathway tissues differ with respect to electrophysiologic properties. Each of these points will be discussed separately.

Prevalence of common pathways. In this study, evidence for upper and lower common pathways was common. Twenty-one of 25 patients had LCPs and eight of 11 patients had UCPs among cases in which data were adequate to draw conclusions. Because of the large number of cases in which no statement can be made concerning the presence or absence of UCP tissue, we cannot be certain that a similar high proportion of all patients with AV nodal reentry would have UCPs. Three patients in our series had no LCP (differences in HA_{pace} vs HA_{svt} less than 5 msec); this may suggest a lower "turn around" point within the AV node in some patients than has been proposed by other authors. 2, 4, 5, 12

Similar differences in HA intervals between pacing
and tachycardia could conceivably be observed if there were a pathway composed of non–AV nodal tissue connecting the atrium directly to a point in the His bundle proximal to the recording site. Since this would require participation of at least some of the His bundle in the tachycardia circuit, dissociation of the His potential from the tachycardia would make this possibility quite unlikely. In several cases in this study, the proximal His potential could be advanced up to 40 msec without affecting the timing of atrial electrograms (and was thus dissociated from the tachycardia) by introducing one or two ventricular extrastimuli during the tachycardia (figure 8).

**Discordant antegrade and retrograde conduction variables.** All patients in this series had a 1:1 AV relationship during tachycardia except for two cases of transient block below the His bundle with 2:1 AV conduction. Thus, Wenckebach periodicity at the same or longer paced cycle length as tachycardia suggests discordance between conduction variables in UCP or LCP tissue depending on the direction of impulse input. Prior studies have shown significant differences in antegrade and retrograde AV conduction, which may be due to these factors operating in common AV nodal pathways or in other areas of the AV node. Reasons for this discordance are not clear but may relate to heterogeneity in fiber orientation and its resultant effects on conduction (tissue anisotropy).

Prior histologic studies have shown fiber disarray within animal and human AV nodes. Regional differences in cellular electrophysiology that have been demonstrated in animal AV nodes as well as differential impedance of tissues to an approaching wavefront of activation may also play a role.

Pacing-induced Wenckebach at a rate slower than the tachycardia strongly suggests that these pathways are composed of AV nodal tissue, since normal working myocardium almost never displays Wenckebach periodicity under the conditions present in this study. This is less certain in the case of atrial pacing-induced Wenckebach, because of differential input of impulses into the AV node. Prior studies have demonstrated slight differences in the rate at which atrial pacing-induced Wenckebach occurs depending on the site of stimulation in the atria, with left atrial or coronary sinus pacing generally producing Wenckebach at faster rates than right atrial stimulation. These differences are almost always 10 beats/min or less and no significant differences in either Wenckebach rates or effective refractory periods of either fast or slow pathways have been observed in these studies. Since pacing was not routinely performed from multiple atrial sites in the present study, it is possible that some patients could have been falsely identified as having UCP by Wenckebach criteria. For this reason, we have included only cases in which the difference in Wenckebach and tachycardia rates exceeded 10 beats/min as indicating the presence of UCP tissue, since differences of this magnitude are unlikely to be observed.

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**FIGURE 6.** Resetting of SVT prevented by a UCP. Recordings and abbreviations are as in figure 3. **Top,** SVT, cycle length 400 msec, is shown with a single atrial premature beat delivered at 270 msec after the HRA electrogram. This results in advancement (resetting) of the next His deflection, after which SVT continues. The vertical dotted line shows where the next His deflection would have occurred if there were no resetting. **Middle,** An atrial premature beat delivered at 240 msec likewise resets the His deflection, which occurs even more prematurely than above. **Bottom,** An atrial premature beat delivered at 220 msec captures the high right atrium and atrial electrogram near the circuit (HBE) but does not reset the tachycardia. This abrupt loss of the resetting suggests the circuit could not be penetrated due to block of the impulse in UCP.
FIGURE 7. Concealed conduction in an LCP. Recordings and abbreviations are as in figure 3. Dotted lines denote the onset of atrial activity for comparison of atrial activation sequence. Two beats of SVT are shown followed by the delivery of 2 premature atrial beats from the high right atrium, after which SVT resumes. The first atrial cycle after these premature beats has the same internal activation sequence as SVT but occurs before it would have been expected, suggesting delay of conduction in an LCP (the impulse taking longer to get to the His bundle than during SVT). A less likely explanation is extremely slow conduction of the atrial echo after the second premature beat (due to concealed conduction in a UCP), which could conceivably produce this response.

with pacing from other atrial sites. “Pseudo Wenckebach” during atrial pacing, caused by occasional AV nodal echoes, must also be excluded by careful examination of recordings. Additionally, two patients in this study had transient 2:1 AV conduction during tachycardia, which could be interpreted as evidence for an LCP; in each case, however, His bundle recordings proved the location of the conduction block to be below the His bundle.

Differences in upper vs lower common pathways. UCPs were significantly more likely to be manifested by Wenckebach criteria than were LCPs, despite the lack of conclusive data for UCP in 17 patients. The reason for this difference is not clear but may again be related to regional differences in fiber orientation and cellular electrophysiology within the AV node. Mignone et al. postulated that the site of initial block that allowed reentry within the AV node occurred within the upper portion (“N” region) based on these differences.

One might assume that, since the differences in AH or HA intervals between pacing and tachycardia consist of the sum of antegrade and retrograde conduction times in the common pathway of interest, the conduction time in one direction (reflecting relative “length” of the pathway) would be roughly one-half the difference in AH or HA times obtained. This assumption may not be valid in light of the disparity in antegrade and retrograde conduction in common pathways; thus no estimate of the absolute or relative “lengths” of these pathways can be made from our data.

Limitations. The current study has several potential limitations, the most important of which are described below:

(1) Reliability of determination of local electrogram onset. Careful comparison of electrogram morphology in the AVJ recordings between tachycardia, sinus rhythm, and atrial or ventricular pacing generally sufficed to make these determinations. In several instances, premature ventricular extrastimuli during tachycardia that did not result in resetting (and thus were unlikely to have entered the circuit to disturb activation sequences) were used to “separate” atrial activity from the larger ventricular electrogram (figure 3). Careful catheter positioning allowed recording of a clear His deflection during ventricular pacing in all but three cases in this study.

(2) The influence of changes in autonomic tone produced by pacing. Tachycardia was reinitiated after
completion of the pacing protocol to determine whether cycle length and AH/HA intervals were stable enough to exclude significant differences caused by modulations of autonomic tone; so significant difference was found in these intervals before and after pacing. Autonomic reflexes activated by ventricular pacing30 cannot be excluded, although there was likely little effect during atrial pacing in the cases in which UCP were found, since the timing of atrial and ventricular activation was nearly the same during pacing and tachycardia (figure 4).

(3) Accuracy of measurement. We believe a 5 msec difference in intervals between pacing and tachycardia using a recording speed of 200 to 250 mm/sec can be readily detected and was reproducible in this study.

(4) Assumptions concerning inputs into the AV node. We arbitrarily took the point of entry into the AV node during atrial pacing to coincide with the end of the atrial electrogram in the AVJ recording (figure 1). This may not be true, but it constitutes a more stringent criterion than if the AHpac were measured from the onset or rapid deflection of the atrial electrogram in the AVJ recording. With these latter measurements, two more UCPs (in patients 16 and 19) would have been detected in this study (table 1). This limitation is mitigated to some extent in that only two UCPs were detected using these measurements, while the other six cases were manifested by antegrade Wenckebach criteria (which is independent of these measurements).

As noted above, pacing from other atrial sites might have resulted in either different AH intervals or Wenckebach rates but was not routinely performed in this study. Accordingly, only cases in which differences between pacing and tachycardia rates exceeded those likely to occur with pacing from a different site (> 10 beats/min) were considered to meet criteria for UCP.

Similarly, we assumed that the entry to the AV node during ventricular pacing coincided with the end of the His electrogram in the AVJ recording (figure 2). This is again a more stringent criterion than measurement from the beginning of the His potential. Indeed, if the latter measurement were used, the remaining three cases in which a His potential was visible during ventricular pacing would fulfill criteria for the presence of an LCP (table 1).
(5) Assumptions regarding the point from which His potential was recorded. If there existed His-bundle tissue proximal to the site from which the His deflection was recorded, it is possible that some or all of the "LCP" could be composed of proximal His bundle tissue. This is unlikely in that (a) the His position was generally proximal judging from atrial electrogram amplitude equal to or exceeding the ventricular electrogram in the AVJ recording, (b) the mean difference in HA intervals (pacing versus tachycardia) was 25 msec, slightly longer than the time required to traverse the entire length of a normal His bundle, and (c) premature ventricular depolarizations during tachycardia could dissociate the His deflection from atrial activity in several cases. Thus, although the presence of a His-atrial connection has not been formally excluded in all cases in this study, we believe this is an unlikely explanation for the observed differences in HA intervals as discussed above.

(6) Possible underestimation of the prevalence of common pathways (especially UCP). It is possible that the prevalence of both common pathways is higher than indicated in this study because of the lack of conclusive data in several cases (i.e., atrial pacing produced a short AHpace suggesting conduction in the "fast" pathway during pacing) and the methods of measurement as discussed above.

Summary. The results of this study suggest that (1) the AV nodal reentrant tachycardia circuit is commonly flanked by upper and/or lower common pathway tissue, which frequently exhibit AV nodal properties (i.e., Wenckebach), and (2) the antegrade and retrograde conduction properties of these upper and lower common AV nodal pathways are frequently discordant. Our study does not specifically address the question of whether or not the entire tachycardia circuit is contained within the AV node or whether perinodal atrial fibers are an essential element in the circuit, as has been suggested by several authors. However, even if this were the case, our findings indicate the presence of tissue with AV nodal properties on both atrial and His bundle aspects of the circuit in many cases. These findings may have importance when attempting to use pacing and/or programmed stimulation to initiate, terminate, or reset AV nodal reentrant tachycardia, since these procedures all require entrance into the functional tachycardia circuit.

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References

4. Janse MJ, VanCapelle PJL, Freug GE, Durrer D: Circus movement within the AV node as a basis for supraventricular tachycardia as shown by multiple microelectrode recording in the isolated rabbit heart. Circ Res 28: 403, 1971
5. Schillenberg RM, Durrer D: Further observations on the ventricular echo phenomenon elicited in the human heart: is the atrium part of the echo pathway? Circulation 45: 629, 1972
10. Hariman RJ, Chen CM, Caracta AR, Damato AN: Evidence that AV nodal re-entrant tachycardia does not require participation of the entire AV node. PACE 6: 1252, 1983
21. Spach MS, Miller WT, Geselowitz DB, Barr RC, Kootsey JM, Johnson EA: The discontinuous nature of propagation in normal canine cardiac muscle: evidence for recurrent discontinuities of
Atrioventricular nodal reentrant tachycardia: studies on upper and lower 'common pathways'.
J M Miller, M E Rosenthal, J A Vassallo and M E Josephson

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