Sequence of Retrograde Atrial Activation in Patients with Dual Atrioventricular Nodal Pathways

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SUMMARY To characterize the sequence of retrograde atrial activation in the presence of dual atrioven-
tricular (AV) nodal pathways, we analyzed electrophysiologic data from seven patients in whom discontinuous 
AV nodal and ventriculoatrial conduction curves could be induced with programmed electrical stimulation. In 
all patients, electrograms of the high right atrium (HRA), lateral right atrium (LRA), low septal right atrium 
(SRA) and proximal coronary sinus (PCS) near the coronary sinus ostium were simultaneously recorded at a 
paper speed of 150–250 mm/sec. During programmed ventricular extrastimulation and incremental ventri-
cular pacing, ventriculoatrial conduction via the fast AV nodal pathway resulted in SRA activation before 
PCS, HRA and LRA activation. However, the sequence of retrograde atrial activation abruptly changed with 
a shift from retrograde fast to retrograde slow AV nodal pathway conduction. Characteristically, during vent-
riculoatrial conduction via the slow AV nodal pathway, activation of the PCS preceded SRA activation by 
5–20 msec and was accompanied by an alteration of the temporal relationship between HRA and LRA activa-
tion in all patients. These observations suggest that, anatomicanly, the proximal common AV nodal pathway 
is a broad area that permits the slow AV nodal pathway to have a retrograde exit located posteriorly, inferiorly 
and to the left of that of the fast AV nodal pathway, and that the retrograde atrial activation sequence rec-
corded during tachyarrhythmias should be determined with caution while attempting to differentiate 
retrograde normal AV pathway from retrograde anomalous bypass tract conduction.

IN DIFFERENTIATING retrograde conduction via the normal atrioventricular (AV) pathway from an 
anomalous bypass tract, one must evaluate ventriculoatrial conduction properties during an electrophysi-
ologic study.1–3 Ventriculoatrial conduction over the normal AV pathway is characterized by the earliest 
activation of the low septal right atrium (SRA) recorded in the His bundle electrographic lead and progressive 
prolongation of ventriculoatrial conduction time with increasing prematurity of ventricular extrastimulation. In contrast, ventriculoatrial conduction over an anomalous bypass tract has the 
earliest atrial activation at the site of its atrial insertion, and, characteristically, there is a lack of refrac-
tory-dependent ventriculoatrial conduction delay despite progressively premature ventricular extra-
stimulation.1–3

dual AV nodal pathways are generally believed to 
be intranodal structures.4–9 Both fast and slow AV 
nodal pathways possess AV nodal properties with refractory-dependent conduction delay in both antegrade and retrograde directions.4–9 However, studies on the sequence of retrograde atrial activation 
have been limited to the fast AV nodal pathway.9, 10 In the present study, we characterize and compare 
retrograde atrial activation sequences that result from 
ventriculoatrial conduction via the fast and the slow 
AV nodal pathways.

Materials and Methods

Electrophysiologic evidence of antegrade dual AV nodal pathway conduction was based on the induction 
of discontinuous AV nodal conduction curves (A1A2, 
H2H3) with programmed atrial extrastimulation,5–7 and retrograde dual AV nodal pathway conduction on 
the induction of discontinuous ventriculoatrial conduction curves (V1V2, A1A2) with programmed ven-
tricular extrastimulation.5–9 Data from 19 patients 
with antegrade dual AV nodal pathways in whom multiple atrial electrograms were simultaneously 
recorded were analyzed. Seven of these patients also had retrograde dual AV nodal pathway conduction, 
which allowed comparative analysis of retrograde fast 
and retrograde slow AV nodal pathway conduction. 
These seven patients constituted the study population.

After the patients gave informed consent, all car-
diotoxic and antiarrhythmic medications were discon-
tinued 48–72 hours before the study. The study was 
performed with patients in a postabsorptive, non-
seated state. With a conventional technique,11 a 
tetrapolar electrode catheter (Elecath #5F) was in-
trouduced from the right femoral vein and placed in the 
right atrium across the tricuspid valve to record the 
His bundle potential (HBE), from which the SRA 
electrogram was recorded as well (fig. 1). To assure 
recording of the HBE, two pairs of electrodes with interelectrode distances of 1 mm and 10 mm, designated 
HBE1 and HBE2, were used. A hexapolar electro-
decatheter (USCI 004178 #7F) was introduced through 
an antecubital vein in the right arm. The dis-
tal pair of electrodes was placed at the right ven-
tricular apex (RVA) for ventricular pacing, and the 
proximal two pairs of electrodes were placed in the 
right atrium to record high right atrial (HRA) and 
lateral right atrial (LRA) electrograms. The HRA

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Presented in part at the 29th Annual Scientific Sessions of the 
American College of Cardiology, March 1980, Miami, Florida.

Dr. Juma was supported by the Ontario Heart Association.

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Received July 23, 1980; revision accepted February 12, 1981. 
Circulation 64, No. 5, 1981.
and LRA electrographic leads had a recording inter-electrode distance of 10 mm and were located 20 mm apart. During programmed atrial stimulation, the HRA lead was used for pacing and the LRA lead for recording. A tetrapolar electrode catheter (USCI 5675 #6F) was inserted from an antecubital vein in the left arm and placed in the coronary sinus (CS). The two pairs of electrodes of the CS catheter, designated CS₁ and CS₂, were adjusted under fluoroscopy so that they were located underneath the HBE for recording CS atrial electrograms (CS₁ was believed to be at the CS ostium) (fig. 1). These two CS electrographic leads had a recording inter-electrode distance of 10 mm and were located 10 mm apart.

Programmed atrial and ventricular stimulation consisted of extrastimulation and incremental pacing. During programmed extrastimulation, the HRA and the RVA endocardium were stimulated at one or two cycle lengths (CLs) (A₁A₂, or V₁V₂) with a programmed digital stimulator (Biocelestrix) that delivered 2-msec stimuli (S₁ and S₂) at approximately twice diastolic threshold. After every eighth paced beat (A₁ or V₁), a single premature atrial or ventricular beat (A₂ or V₂) was delivered at progressively shorter coupling intervals (A₁A₂ or V₁V₂) until the effective refractory period of the atrium or the ventricle was encountered.

**Definition of Terms**

AV and ventriculoatrial conduction intervals and refractory periods were defined and measured as conventionally described. **A₁**, H₁, and V₁ were SRA, His bundle and ventricular responses, induced by the driving stimuli (S₁). A₂, H₂ and V₂ were low SRA, His bundle and ventricular responses, induced by the premature stimuli (S₂).

When discontinuous AV nodal conduction curves (A₁A₂, H₁H₂) and discontinuous ventriculoatrial conduction curves (V₁V₂, A₁A₂) were induced during antegrade and retrograde conduction studies, the refractory periods of the fast and slow AV nodal pathways were measured and defined as described by Denes and Wu and co-workers. Briefly, the curve to the right of the discontinuity reflected the fast AV nodal pathway conduction and that to the left represented the slow AV nodal pathway conduction. In discontinuous A₁A₂, H₁H₂ curves, the longest A₁A₂ interval at which A₂ was blocked in the fast or slow AV nodal pathway was defined as the antegrade effective refractory period of the fast or slow AV nodal pathway. Similarly, in discontinuous V₁V₂, A₁A₂ curves, the longest V₁V₂ interval at which V₂ was blocked in the fast or slow AV nodal pathway was defined as the retrograde effective refractory period of the fast or slow AV nodal pathway.

**Results**

**Antegrade Dual AV Nodal Pathway Conduction**

At atrial driving CLs of 500–700 msec, discontinuous AV nodal conduction curves (A₁A₂, H₁H₂) suggestive of antegrade dual AV nodal pathway conduc-
tion could be induced with atrial extrastimulation in all seven patients (figs. 2 and 3). The antegrade effective and functional refractory periods of the fast AV nodal pathway ranged from 320-540 msec and from 410-580 msec, respectively. The antegrade effective and functional refractory periods of the slow AV nodal pathway ranged from < 260-390 msec and from 460-550 msec, respectively (table 1). The atrial echo phenomenon was observed during antegrade slow AV nodal conduction in patients 4-6. However, only patient 6 had sustained AV nodal reentrant tachycardia of the slow-fast form. Using the slow nodal pathway for antegrade conduction and the fast AV nodal pathway for retrograde conduction.

Retrograde Dual AV Nodal Pathway Conduction

At the same CLs chosen for atrial pacing, discontinuous ventriculoatrial conduction curves (V1V2, A1A2) suggestive of retrograde dual AV nodal pathway conduction could be induced with ventricular extrastimulation in all patients (fig. 3B). The retrograde effective and functional refractory periods of the fast AV nodal pathway ranged from 320-450 msec and from 400-560 msec, respectively. The retrograde effective and functional refractory periods of the slow AV nodal pathway ranged from 220-300 msec and from 500-610 msec, respectively (table 1). Single ventricular echo phenomena could be induced during retrograde slow AV nodal pathway conduction in patients 1, 3, 4, 5, and 7. None of the patients had sustained AV nodal reentrant tachycardia of the fast-slow form.7, 8

Retrograde Atrial Activation Sequence

Simultaneous recordings of multiple atrial electrograms provided detailed analysis of retrograde atrial activation sequence during programmed ventricular stimulation in these patients. As would be expected, the sequence of retrograde atrial activation resulting from retrograde conduction over the fast AV nodal pathway was first the SRA in the HBE lead, followed by the proximal CS (CS1 and CS2), the HRA and the LRA in all patients. The SRA activation preceded proximal CS activation by 10-30 msec (table 2). In contrast, the retrograde atrial activation sequence resulting from retrograde conduction via the slow AV nodal pathway was characterized by earliest activation at the proximal CS. The activation of the proximal CS preceded low SRA activation by 5-20 msec and was accompanied by an alteration of the temporal relationship between LRA and HRA activation during ventriculoatrial conduction via the slow AV nodal pathway (table 2).

Figure 3B demonstrates the induction of discontinuous ventriculoatrial conduction curves (V1V2, A1A2, and V1V2, A1A2) with ventricular extrastimulation during right ventricular (RV) pacing at a CL of 650 msec. The retrograde effective and functional refractory periods of the fast AV nodal pathway were 350-360 and 400 msec, respectively, and the retrograde effective and functional refractory periods of the slow AV nodal pathway 290 and 610 msec, respectively. The ventricular echo zone coincided with the entire retrograde slow AV nodal pathway conduction curve. Retrograde slow AV nodal pathway conduction time (HAW) was 280-320 msec.

Tracings corresponding to fig 3B are presented in figures 4 and 5. During RV pacing at a CL (S1S1) of 650 msec, a ventricular premature complex (S2) at a premature coupling interval (S1S2) of 350 msec could be conducted to the atrium via either the fast or the slow AV nodal pathway (fig. 4). When the ventricular premature complex (S2) was conducted by way of the fast AV nodal pathway, it lengthened ventriculoatrial conduction time (the S2A2 interval measured from the

**Figure 2.** Induction of antegrade dual atrioventricular (AV) nodal pathway conduction with atrial extrastimulation (case 1). The high right atrium is driven at a cycle length (S1S1) of 650 msec. (A) An atrial premature beat (S2) at a premature coupling interval (S1S2) of 550 msec lengthens the AV nodal conduction time (AH) from 140 to 170 msec. (B) An atrial premature beat (S2) at a premature coupling interval (S1S2) of 540 msec suddenly prolongs the AV nodal conduction time to 250 msec, resulting in discontinuous AV nodal conduction curves (A1A2, H1H2, and A1A2, A2H2) (fig. 3A). Paper speed = 150 mm/sec. LRA = lateral right atrial electrogram; CS1 and CS2 = coronary sinus leads. HBE = His bundle electrogram.
when the ventricular premature complex (S₂) was conducted by way of the slow AV nodal pathway, it abruptly prolonged the ventriculoatrial conduction time (S₂A₂) to 440 msec (fig. 4B), thereby producing discontinuous ventriculoatrial conduction curves (V₁V₂, A₁A₂ and V₁V₂, V₂A₂) (fig. 3B). This was attributed to failure of retrograde fast with resultant retrograde slow AV nodal pathway conduction. A ventricular echo phenomenon was initiated (fig. 4B). Progressive shortening of the ventricular premature coupling interval (S₂S₄) continued to induce retrograde slow AV nodal pathway conduction with the ventricular echo phenomenon (fig. 5A) until ventriculoatrial conduction failed at a ventricular premature coupling interval (S₃S₄) of 290 msec (fig. 5B). The retrograde His bundle potential (H⁻) was recorded. Retrograde AV nodal conduction time (H⁻A) was 80 msec for the fast AV nodal pathway (fig. 4A) and 310–320 msec for the slow AV nodal pathway (figs. 4B and 5A).

The sequence of retrograde atrial activation was analyzed with measurements of ventriculoatrial conduction time (S₃A₃) at each atrial recording site. During retrograde fast AV nodal pathway conduction, the SRA in the HBE lead was activated 20–30, 40 and 60 msec before CS₁ and CS₂, the HRA and the LRA, respectively (fig. 4A). In contrast, during retrograde

![Figure 3. Discontinuous atrioventricular (AV) nodal and ventriculoatrial conduction curves suggestive of antegrade and retrograde dual AV pathway conduction. The retrograde His bundle (H⁻) is represented by open triangles. Retrograde slow AV nodal pathway conduction time (H⁻-A₂) measures 280–320 msec. HRA = high right atrium; CL = cycle length; SRA = septal right atrium; RV = right ventricular.](http://ahajournals.org)

**Table 1. Electrophysiologic Data**

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<th>FP (msec)</th>
<th>SP (msec)</th>
<th>AERP (msec)</th>
<th>AFRP (msec)</th>
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<th>RFRP (msec)</th>
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**Abbreviations:** AERP = antegrade effective refractory period; AFRP = antegrade functional refractory period; DCL = driving cycle length; FP = fast atrioventricular nodal pathway; RERP = retrograde effective refractory period; RFRP = retrograde functional refractory period; SP = slow atrioventricular nodal pathway.
Table 2. Temporal Relationship of Atrial Electrogams During Retrograde Dual Atrioventricular Nodal Pathway Conduction

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<th>SRA (msec)</th>
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<th>CS₂ (msec)</th>
<th>LRA (msec)</th>
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The numbers indicate msec after the earliest activation site, which is designated as the zero reference point.

Abbreviations: CS = coronary sinus; FP = fast atrioventricular nodal pathway; HRA = high right atrium; LRA = lateral right atrium; PCS = proximal coronary sinus; SP = slow atrioventricular nodal pathway; SRA = low septal right atrium.

slow AV nodal pathway conduction, the sequence of retrograde atrial activation was abruptly changed. The CS₁ and CS₂ were activated 5–10, 25 and 40 msec before the SRA, the LRA and the HRA, respectively (figs. 4B and 5A). The change in the temporal relationship of retrograde atrial activation occurred not only between the SRA and CS₁ and CS₂, but also between the HRA and the LRA. Further, the change in the retrograde atrial activation sequence consequent upon the shift from retrograde fast to retrograde slow AV nodal pathway conduction was accompanied by alterations of the morphology of the atrial electrograms (figs. 4 and 5).

The sequence of retrograde atrial activation resulting from each AV nodal pathway conduction in the retrograde direction was consistently reproducible with repeated ventricular extrastimulation. The characteristic alteration of the retrograde atrial activation sequence consequent upon the shift from retrograde fast to retrograde slow AV nodal pathway conduction could also be observed during ventriculoatrial Wenckebach phenomenon induced by rapid ventricular pace in all patients. One example is given in figure 6. The right ventricle was driven at a CL of 540 msec, which induced 4:3 ventriculoatrial Wenckebach phenomenon. Ventriculoatrial conduction time cor-

Figure 4. Induction of retrograde dual atrioventricular (AV) nodal pathway conduction with ventricular extrastimulation (same patient as in figure 2). H indicates retrograde His bundle potentials. Retrograde atrial activation sequence resulting from retrograde fast AV nodal pathway conduction (A) is first the low septal right atrium (SRA) in the His bundle electrographic lead (HBE), followed by the proximal coronary sinus (CS₁ and CS₂), high right atrium (HRA) and lateral right atrium (LRA), while that sequence from retrograde slow AV nodal pathway conduction (B) is first the proximal coronary sinus (CS₁ and CS₂), followed by LRA, SRA and HRA. The morphology of the atrial electrogram changes as a result of the shift from retrograde fast to retrograde slow AV nodal pathway conduction. Ventriculoatrial conduction time (SA interval) is measured from the stimulus to the onset of atrial electrogram at each recording site and is expressed in msec. Paper speed = 250 mm/sec.
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**Figure 5.** Further shortening of the ventricular premature coupling interval \((S_3 S_2)\) continues to induce ventriculoatrial conduction by way of retrograde slow atrioventricular \((AV)\) nodal pathway conduction \((A)\) until ventriculoatrial conduction fails at the ventricular premature coupling interval \((S_3 S_2)\) of 290 msec \((B)\). Note that the retrograde atrial activation sequence and the morphology of the atrial electrograms resulting from retrograde slow \(AV\) nodal pathway conduction are different from those by way of retrograde fast \(AV\) nodal pathway conduction during basic driving beats \((S_1)\). Retrograde slow \(AV\) nodal pathway conduction time \((H-A)\) measures 320 msec. Paper speed = 250 mm/sec. \(HRA = \) high right atrium; \(LRA = \) lateral right atrium; \(CS_1 \) and \(CS_2 = \) coronary sinus leads; \(HBE = \) His bundle electrogram.

responding to the first paced beat was 170 msec, measured from the HBE lead. In the second and third paced beats, ventriculoatrial time was then abruptly lengthened to 370 and 390 msec, respectively, suggesting failure of retrograde fast \(AV\) nodal pathway and resultant retrograde slow \(AV\) nodal pathway conduction. The retrograde atrial activation sequence resulting from retrograde fast \(AV\) nodal pathway conduction (corresponding to the first paced beat) and that resulting from retrograde slow \(AV\) nodal pathway conduction (corresponding to the second and third paced beats) were similar to those induced with programmed ventricular extrastimulation (figs. 4 and 5). These findings were consistently reproducible with

**Figure 6.** Four-to-three ventriculoatrial Wenckebach phenomenon induced by rapid ventricular pacing at a cycle length of 540 msec \((case\ 1)\). The ventriculoatrial conduction time \((SA\ interval)\) is measured from the stimulus \((S)\) to the onset of atrial electrogram at each recording site and is expressed in msec. Note sudden prolongation of ventriculoatrial conduction time after the second paced beat, suggestive of failure of retrograde fast atrioventricular \((AV)\) nodal pathway with resultant retrograde slow \(AV\) nodal pathway conduction. The retrograde atrial activation sequence resulting from retrograde slow \(AV\) nodal pathway conduction is different from that by way of retrograde fast \(AV\) nodal pathway conduction. Paper speed = 250 mm/sec. \(HRA = \) high right atrium; \(LRA = \) lateral right atrium; \(CS_1 \) and \(CS_2 = \) proximal coronary sinus leads; \(HBE_1 \) and \(HBE_2 = \) His bundle electrographic leads.
repeated rapid ventricular pacing in all patients. An additional example (patient 5) is shown in figure 7.

**Discussion**

Observations made in this study indicated characteristic alteration of the retrograde atrial activation sequence resulting from a shift from retrograde fast to retrograde slow AV nodal pathway conduction in patients with dual AV nodal pathways. In previous studies, either only HRA and SRA electrograms were recorded for analysis of the sequence of retrograde atrial activation or the description of retrograde atrial activation sequence was limited to that resulting from retrograde fast AV nodal pathway conduction. The present study, therefore, provides further information pertaining to the understanding of retrograde dual AV nodal pathway conduction.

The induction of discontinuous AV nodal conduction curves (A1A2, H1H2) with atrial extrastimulation conformed to the presence of antegrade dual AV nodal pathways (fig. 3A, table 1). Nevertheless, the induction of discontinuous ventriculoatrial conduction curves (V1V3, A1A2) with ventricular extrastimulation would also raise the possibility of coexisting anomalous bypass tracts functioning in the retrograde direction. It has been described that discontinuous ventriculoatrial conduction curves (V1V3, A1A2) can be induced with ventricular extrastimulation during retrograde conduction over the normal AV pathway and an anomalous bypass tract when either of the two fails to conduct in the retrograde direction. Analysis of discontinuous ventriculoatrial conduction curves (V1V3, A1A2) induced in our patients revealed that both fast and slow pathways exhibited AV nodal conduction properties during retrograde conduction, with the fast pathway having a retrograde effective refractory period longer than that of the slow pathway (fig. 3B, table 1). Also, despite having different sequences of retrograde atrial activation, the earliest sites of retrograde atrial activation resulting from retrograde conduction over the two pathways, the SRA and CS, were anatomically close to the AV septum (fig. 1). Klein et al. recently observed decremental retrograde conduction via anomalous bypass tracts in three of their 30 patients with the Wolff-Parkinson-White syndrome. However, the magnitude of conduction delay during retrograde conduction over the slow pathway as demonstrated in this study was much greater than that reported by Klein et al., as evidenced by recordings of atrial electrograms far apart from those of ventricular electrograms during retrograde slow pathway conduction (ventriculoatrial intervals ranged from 290–525 msec) (figs. 4–6). These observations show that the slow pathway has characteristics of the AV node in the retrograde direction in these patients.

The structural and cellular complexity of the human AV junctional area has not been completely clarified and the definition of the AV node has created considerable controversies. In the isolated rabbit heart, Paes de Carvalho and de Almeida distinguished three functional separate parts in the AV node based on different action potential recordings: atrionodal, nodal and nodo-His zones. These three cell zones were subsequently found to correlate to some extent with the three cell types (transitional, upper nodal and lower nodal) described in the histologic studies performed by Anderson. The precise location of dual AV nodal pathways in relation to the AV nodal structure, however, is speculative. In isolated rabbit hearts, Janse et al. demonstrated dual AV nodal inputs and induction of atrial echo phenomenon with atrial extrastimulation. Unfortunately, they could not completely map out the AV nodal reentrant circuit and suggested that many possible different pathways might exist within the AV node.

Our findings indicate that the proximal common
The understanding support observations in sinus a using both the middle and posterior parts of the AV node toward the coronary sinus in isolated canine and rabbit hearts. These observations support the concept that the atrium may not be a necessary link for AV nodal reentry and imply that the presence of dual AV nodal pathways may be both functional and anatomic.

Recording the retrograde sequence of atrial activation using the standard technique of intracardiac recordings has limitations. First, it is difficult to maintain the proximal CS catheter electrodes close to the HBE catheter electrodes (fig. 1). Cardiac movements associated with programmed electrical stimulation can easily displace the catheter electrodes. Therefore, the position of the CS catheter electrodes must be readjusted during the study. Second, the extent of alteration in the retrograde atrial activation sequence so recorded may depend on anatomic variation of the size and location of the AV node as well as the magnitude of conduction delay related to retrograde slow AV nodal pathway conduction. It appears that the longer the retrograde slow AV nodal pathway conduction time, the more likely the change in the retrograde atrial activation sequence. Third, some catheter electrode movement during cardiac systole and diastole is expected, particularly when associated with changes in the cardiac CL may account for some of the findings, although we could consistently reproduce the findings in our patients.

Electrophysiologic Implications

Our electrophysiologic findings may be relevant to the understanding of what causes the permanent form (or the fast-slow form) of AV junctional reciprocating tachycardia with an RP interval longer than the PR interval. 

Figure 8. Schematic representation of retrograde dual atrioventricular (AV) nodal pathway conduction. The coronary sinus (CS) ostium is located posterior and inferior to the AV node. (left) Retrograde fast AV nodal pathway conduction. (middle) Retrograde slow AV nodal pathway conduction. (right) Retrograde slow AV nodal pathway conduction with a ventricular echo phenomenon. It is suggested that the slow AV nodal pathway has a retrograde exit site located posterior and inferior to that of the fast AV nodal pathway: A = atrium; H = His bundle; V = ventricle; FP = fast AV nodal pathway; SP = slow AV nodal pathway.
bypass tract has been implicated. Various anatomic substrates may be responsible for the fast-slow form of AV junctional reciprocating tachycardia. Further anatomic studies are necessary to substantiate these theories.

References