CASE REPORT

Participation of Fast and Slow A-V Nodal Pathways in Tachycardias Complicating the Wolff-Parkinson-White Syndrome

Report of a Case

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SUMMARY Electrophysiological studies in one patient with type B pre-excitation and dual A-V nodal pathway revealed several types of paroxysmal narrow QRS tachycardia (PSVT). One type of PSVT reflected antegrade fast A-V nodal pathway and retrograde anomalous pathway conduction. This PSVT was characterized by early retrograde activation of right atrial appendage, P following QRS and cycle length of 440 msec. A second PSVT reflected antegrade slow A-V nodal pathway and retrograde anomalous pathway conduction. This PSVT was characterized by early retrograde atrial activation sequences, P simultaneous with QRS, and cycle length of 320 msec. All PSVT inductions could be explained in terms of antegrade and retrograde properties of fast and slow A-V nodal and anomalous pathways.

MOST PATIENTS with A-V nodal re-entrant paroxysmal supraventricular tachycardia (PSVT) have discontinuous A-V nodal conduction curves suggesting dual (fast and slow) A-V nodal pathways.1-9 The discontinuity in curves suggests that both fast and slow A-V nodal pathways are capable of antegrade conduction in these patients. The usual A-V nodal re-entrant paroxysmal tachycardia reflects antegrade slow pathway and retrograde fast pathway conduction.1-9

In most patients with Wolff-Parkinson-White syndrome, PSVT is re-entrant utilizing the normal A-V pathway for antegrade conduction and an anomalous extranodal pathway (Kent bundle) for retrograde conduction.10, 11 If a patient with Wolff-Parkinson-White syndrome also had dual A-V nodal pathways, then several re-entrant loops might be possible. For example, antegrade conduction during PSVT could be via either fast or slow A-V nodal pathways. Such a patient might also have typical intra-A-V nodal re-entrance (antegrade slow and retrograde fast pathway conduction).

In this study, we report one patient with Wolff-Parkinson-White syndrome, in whom three electrophysiologically distinct paroxysmal tachycardias were demonstrated; the first, characterized by antegrade fast pathway and retrograde anomalous pathway conduction; the second, by antegrade slow pathway and retrograde anomalous pathway conduction; and the third, by antegrade slow pathway and retrograde fast pathway conduction. We believe this to be the first documented report of the coexistence of dual A-V nodal pathways and the Wolff-Parkinson-White syndrome, in which three different types of re-entrant paroxysmal tachycardias were observed to occur in the same patient.

Methods

Electrophysiological studies were performed in a patient with Wolff-Parkinson-White syndrome. All drugs were discontinued 48 hours prior to the study. Informed consent was obtained. A tripolar electrode catheter was percutaneously introduced into the right femoral vein and placed at the tricuspid valve for recording of His bundle and low right septal atrial electrograms.12 A quadripolar electrode catheter was introduced into the left femoral vein and advanced to the high lateral right atrium and positioned at the superior vena cava right atrial junction, for both stimulation and recording. A bipolar electrode catheter, introduced via a right antecubital vein, was positioned at the right ventricular apex for ventricular pacing. After recording basic intervals, the atria were paced at increasing rates in 10 beats/min increments up to a maximum paced rate of 200 beats/min. Refractory periods were determined with extrastimulus technique,13 utilizing stimuli of 2 msec duration and twice diastolic threshold in intensity. Stimuli were supplied by a digital programmable pulse generator (manufactured by M. Bloom). Intracardiac electrograms, as well as electrocardiographic standard leads I, II, III, and chest lead V5, were simultaneously displayed on an oscilloscope and recorded on a multichannel recorder (Electronics for Medicine DR-16) at paper speeds of 100 and 200 mm/sec.

To localize the atrial insertion of anomalous pathways, multiple atrial sites were paced and stimulus-delta intervals measured.14 During episodes of induced sustained parox-
ysmal tachycardia, retrograde intracardiac atrial mapping was performed with recordings of bipolar electrograms from the low septal right atrium, high right atrium, low lateral right atrium, and right atrial appendage. The latter site was explored by positioning the tip of the electrode catheter in the high right atrium, and then rotating the tip anteromedially. The catheter location was confirmed using biplane fluoroscopy.

Retrograde anomalous pathway properties were assessed by pacing the right ventricle up to rates of 180 beats/min and with ventricular extrastimulus technique. Ventricular extrastimulus technique was also utilized during episodes of paroxysmal tachycardia as previously described.

Electrophysiological Definitions

HRA1, LSRA1, H1, V1, and Δ1 were respectively the high right atrial, low septal right atrial, His bundle, ventricular electrogram, and delta wave of sinus or driven beats (S1). HRA2, LSRA2, H2, V2, and Δ2 were electrograms in response to the extrastimulus (S2). Definitions of conduction intervals, refractory periods, as well as criteria for dual A-V nodal pathway have been previously described.1-3 Ventricular atrial conduction times (V-A) were measured from the ventricular stimulus artifact to the first high frequency potential of the LSRA electrogram.

Report of Case

This patient was a 25-year-old female with type B preexcitation and recurrent symptomatic paroxysmal supraventricular tachycardias (PSVT) and paroxysmal atrial fibrillation, refractory to medical management. Electrophysiological studies revealed a sinus rate of 99 beats/min, a P-Δ of 100 msec and a QRS duration of 140 msec. His bundle potentials were masked in the ventricular electrogram. Anomalous pathway conduction persisted up to atrial paced rates of 200 beats/min. Pacing at multiple atrial sites revealed shortest stimulus–delta intervals (60 msec) with pacing of the right atrial appendage. Epicardial mapping done at the time of surgery demonstrated anterior right ventricular pre-excitation, which was cured by incision in the right anterior A-V ring.

Atrial extrastimulus testing was performed at an atrial driven cycle length of 545 msec (fig. 1). As A1-A2 coupling intervals were decreased from 530 msec to 330 msec, Δ1-Δ2 intervals decreased from 530 msec to 330 msec (figs. 1 and 2A). At an A1-A2 of 320 msec, the anomalous pathway effective refractory period was achieved, and antegrade conduction occurred via the normal pathway with a narrow QRS complex and V1-V2 of 490 msec. PSVT #1 (see below) was simultaneously induced (figs. 1 and 2B). At an A1-A2 interval of 290 msec (figs. 1 and 2C), A1-H2 interval was 180 msec and V1-V2 was 490 msec. A decrease in A1-A2 interval of 10 msec to 280 msec resulted in a sudden decrease of A2-H2 interval to 320 msec, and of V1-V2 interval to 610 msec (figs. 1 and 2D), suggesting dual A-V nodal pathways. PSVT #2 was simultaneously induced (see below). A-V conduction was atrial limited with an atrial functional refractory period of 210 msec.

V-A interval was 100 msec at ventricular paced rates of 100 to 180 beats/min. Ventricular extrastimulus testing was performed at a driven cycle length of 500 msec. As V1-V2 coupling intervals were decreased from 480 to 260 msec, A1-A2 intervals decreased from 480 to 260 msec, while V-A intervals remained unchanged. The ventricular functional refractory period limited V-A conduction.

Three types of PSVT were observed during the study: PSVT #1 (table 1, figs. 1, 2B and 2C) was characterized by a cycle length of 290 to 350 msec, an A-H interval of 140 to 180 msec, a V-A interval of 110 msec, and a retrograde P wave following the QRS complex. Mapping of atrial activation during PSVT #1 revealed earliest atrial activation at the right atrial appendage (fig. 3). PSVT #1 was reproducibly induced during coupled atrial stimulation (cycle length of 545 msec) at A1-A2 intervals of 320 to 290 msec. QRS complexes conducted with incomplete right bundle branch block during PSVT #1 were accompanied by a 40 msec increase in V-A time, also suggesting retrograde conduction via a right-sided anomalous pathway (figs. 2B and 2C).4-6 The outer limit of the zone of induction coincided with the anomalous pathway effective refractory period, the inner limit with the fast pathway effective refractory period. Ventricular extrastimulus technique was also utilized during episodes of paroxysmal tachycardia as previously described.

Electrophysiological Data

Table 1. Electrophysiological Data

<table>
<thead>
<tr>
<th>Case</th>
<th>Cycle length (msec)</th>
<th>A-H (msec)</th>
<th>V-A (msec)</th>
<th>Earliest atrial activation</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSVT 1</td>
<td>290-350</td>
<td>140-180</td>
<td>110</td>
<td>RAA</td>
<td>F-K</td>
</tr>
<tr>
<td>PSVT 2</td>
<td>440</td>
<td>320</td>
<td>110</td>
<td>RAA</td>
<td>S-K</td>
</tr>
<tr>
<td>PSVT 3</td>
<td>320</td>
<td>280</td>
<td>15</td>
<td>LSRA</td>
<td>S-F</td>
</tr>
</tbody>
</table>

Abbreviations: RAA = right atrial appendage; LSRA = low right atrial septum; F = fast pathway; S = slow pathway; K = Kent bundle.
stimulus testing utilized during episodes of PSVT #1 confirmed the presence of an extranodal pathway by demonstrating that the atrium could be pre-empted at a time when the antegrade His bundle had just been utilized.  

PSVT #2 (table 1, figs. 1 and 2D) was characterized by a cycle length of 440 msec, an A-H interval of 320 msec, a V-A time of 110 msec, and a retrograde P wave following the QRS complex. Atrial activation sequences during this PSVT were similar to PSVT #1. PSVT #2 was reproducibly induced during coupled atrial stimulation (cycle length of 545 msec) at A1-A2 intervals of 280 to 260 msec. The outer limit of the zone of induction was the effective refractory period of the fast pathway, while the inner limit was the functional refractory period of the atria. Ventricular extrastimulus testing was not performed during PSVT #2.

A third PSVT (PSVT #3), was induced during delivery of a ventricular extrastimulus during PSVT #1 (figs. 4A and B). PSVT #3 (table 1, fig. 4B) was characterized by a cycle length of 320 msec, an A-H interval of 280 msec, and a V-A time of 15 msec, with a retrograde P wave simultaneous with the QRS complex. Examination of atrial activation sequences during PSVT #3 revealed that low septal right atrial activation preceded high right atrial activation. Induction of PSVT #3 was dependent upon critical ventricular coupling interval during PSVT #1. A ventricular extrastimulus coupled at 215 msec (V-V2) shortened A2-A4 to 215 msec without affecting PSVT #1 (fig. 4A). As V-V4 was decreased to 205 msec, A2-A4 was shorted to 205 msec (fig. 4B) resulting in PSVT #3.

Comment

The analysis of A-V conduction curves in figure 1 suggested the presence of triple A-V pathways. The lower curve reflected conduction via the anomalous pathway, the middle curve corresponded to the fast A-V nodal pathway and the upper curve corresponded to the slow A-V nodal pathway.

Induction of PSVT #1 coincided with failure of the anomalous pathway. The fast A-V nodal pathway was used for antegrade conduction and the anomalous pathway for retrograde conduction. The participation of the anomalous pathway as the retrograde limb in the circus movement was suggested by a) abnormal atrial activation during PSVT, b) lengthening of V-A time with ipsilateral functional bundle branch block during PSVT, c) demonstration that the atrium could be pre-empted during PSVT with a ventricular extrastimulus at a time when the His bundle had just been utilized.

Induction of PSVT #2 coincided with failure of the fast A-V nodal pathway, as shown by the sudden increase in A2-H2 interval at critical A2-A4 coupling intervals. This discontinuity in A-V conduction curves is characteristic of dual A-V nodal pathways. The postulated re-entrant loop involved antegrade conduction via an A-V nodal slow pathway and retrograde conduction via an anomalous pathway. The participation of an A-V nodal antegrade slow pathway was suggested by a) long A-H intervals (320 msec) compared with 140 msec during PSVT #1, resulting in a PSVT with a relatively long cycle length; and b) an echo zone for induc-
During PSVT curred a right of 215 PSVT participation without retrograde pathway. Recorded trograms as PSVT #1. Shown are electrograms recorded from the right atrial appendage (RAA), low septal right atrium (LSRA), high right atrium (HRA), low lateral right atrium (LLRA). Note that the earliest atrial activation was recorded in right atrial appendage (also note that no ventricular electrograms are seen at this site).

![Figure 3](image)

**Figure 3. Atrial mapping during PSVT #1.** Shown are electrograms recorded from the right atrial appendage (RAA), low septal right atrium (LSRA), high right atrium (HRA), low lateral right atrium (LLRA). Note that the earliest atrial activation was recorded in right atrial appendage (also note that no ventricular electrograms are seen at this site).

The participation of the anomalous pathway as the retrograde limb in the circus movement was suggested by retrograde conduction similar to that observed during PSVT #1.

PSVT #3 appeared to reflect re-entry within the A-V node without participation of the anomalous pathway. This occurred only during delivery of a ventricular extrastimulus during PSVT #1, as shown in figures 4A and 4B. Our interpretation of this induction is illustrated in figure 5. During PSVT #1, a ventricular extrastimulus (Vt) introduced at a V-V of 215 msec conducted retrogradely via the anomalous pathway resetting the atria (fig. 5A). The reset atrial activation encountered a refractory fast pathway, so that antegrade conduction in the next cycle was via the slow pathway. There was deep enough antegrade penetration of the fast pathway by As, so that the fast pathway was not available for retrograde conduction in the subsequent cycle and PSVT #1 resumed. With a ventricular extrastimulus at slightly shorter coupling intervals, the reset atrial response was also conducted via the slow pathway because of antegrade fast pathway refractoriness (fig. 5B). However, we would postulate that penetration of the fast pathway was less deep, so that the fast pathway was available for retrograde conduction in the subsequent cycle, pre-empting the atria (from the retrogradely conducting Kent bundle) and allowing induction of PSVT #3. PSVT #3 appeared to be typical A-V nodal re-entrant paroxysmal tachycardia with antegrade slow pathway and retrograde fast pathway conduction.

The evidence for A-V nodal re-entrance as a mechanism of this PSVT was a) the simultaneous occurrence of the atrial echo and the ventricular depolarization, suggesting that the ventricular tracere were not a necessary part of the re-entry, b) demonstration of the necessity of critical A-H for PSVT induction, c) the change in atrial activation sequence, with early activation of the low septal right atrial electrogram, and d) the second discontinuity of the conduction curve (sudden increase in A-H).

**Discussion**

Current evidence suggests that patients with the Wolff-Parkinson-White syndrome have extranodal anomalous pathways that bypass the normal conduction system. The existence of a dual A-V conduction system (normal and anomalous pathway) with dissimilar electrophysiological properties predisposes to re-entrant supraventricular tachycardias. PSVT in patients with pre-excitation is often initiated by an atrial premature beat which blocks in the anomalous pathway, conducting antegrade through the normal pathway and returning to the atria via the anomalous pathway. This allows initiation of circus movement tachycardia characterized by narrow QRS complexes. Less commonly, the anomalous pathway is used as the antegrade limb and the normal pathway as the retrograde limb of the re-entrant circuit, resulting in a wide QRS tachycardia.

In patients without Wolff-Parkinson-White syndrome, the
most common mechanism of PSVT is A-V nodal re-entrance. The majority of patients with A-V nodal re-entrant supraventricular tachycardias have discontinuous A-V conduction curves suggesting dual A-V nodal pathways. In these cases, A-V nodal re-entrant tachycardias are usually characterized by antegrade slow and retrograde fast pathway conduction. Since functional longitudinal dissociation of the A-V node is demonstrable in some patients, it is conceivable that patients with pre-excitation could have several possible re-entrant loops (involving A-V nodal fast pathway, A-V nodal slow pathway, and anomalous pathway, in various combinations).

There are a few previously reported cases of A-V nodal re-entrance in patients with the Wolff-Parkinson-White syndrome. Friedberg and Schamroth in 1973 reported a case of Wolff-Parkinson-White syndrome with alternation of antegrade conduction times (electrocardiographically diagnosed) during PSVT. They postulated the existence of dual A-V nodal pathways with alternation of conduction in slow and fast pathways. Spurr el al. similarly reported the presence of A-V nodal re-entrant tachycardia apparently involving three intranodal pathways in a patient with Wolff-Parkinson-White syndrome. During PSVT, there was alternation in antegrade conduction times (similar to Friedberg's case), while a third intranodal pathway was postulated to be conducting retrogradely. The Kent bundle was apparently not involved in the circus movement. Atrial mapping and right ventricular stimulation were not reported in the above cases. Neuss and Schlepper observed one patient with pre-excitation in whom dual A-V nodal pathways were suspected. During coupled atrial stimulation, a sudden increase in A2-H2 was observed, without A-V nodal re-entrance. They also reported a second patient with evidence of A-V nodal re-entrance with short runs of PSVT in which the accessory pathway was not a necessary link in the tachycardia circuit. Zipes et al. reported one patient with Wolff-Parkinson-White syndrome with two types of PSVT. During one type of PSVT, A-V nodal re-entrance was suspected because of the occurrence of atrial echoes at a time when the initiating atrial impulse had simultaneously blocked in the anomalous pathway and distal to H. In contrast to our case, antegrade conduction during PSVT was via the anomalous pathway. In the second type of PSVT, antegrade conduction occurred via the normal pathway with a left bundle branch block pattern and returned to the atria via the anomalous pathway. The findings in this case were interpreted by Denes and Rosen to be suggestive of dual A-V nodal pathways, and bear some resemblance to the present reported case. Wellens recently reported his experience in 71 patients with Wolf-Parkinson-White syndrome and PSVT. A-V nodal re-entrance was suspected as a mechanism of PSVT in eight patients. Rosen et al. and subsequently Mandel et al. also suggested the occurrence of A-V nodal re-entrance in case reports of patients with Wolf-Parkinson-White syndrome. However, the latter authors did not provide electrophysiological evidence of dual A-V nodal pathways.

The following characteristics of PSVT #1 from the presently reported case strongly suggested the participation of the anomalous pathway as the retrograde limb in the circus movement: 1) Abnormal retrograde atrial activation sequence during PSVT with right atrial appendage being the earliest recorded atrial site. 2) Retrograde P waves occurring at the termination of the QRS, an additional characteristic of anomalous pathway retrograde conduction. 3) Lengthening in V-A time during PSVT, with functional bundle branch block ipsilateral to the anomalous pathway. 4) Demonstration with ventricular extrastimulus technique that the atrium could be pre-empted at a time when the His bundle had just been utilized for antegrade conduction. Although ventricular extrastimulus testing was not performed during PSVT #2, the identical V-A time, the retrograde P at the termination of the QRS, and similar atrial activation sequence were supportive evidence for retrograde conduction via the anomalous pathway.

Both PSVT #1 and #2 were characterized by antegrade conduction via the normal pathway. A-V nodal antegrade conduction was characteristic of dual A-V nodal pathways. This allowed both fast and slow PSVT, depending upon which pathway was utilized for antegrade conduction. The electrophysiological characteristics suggestive of dual A-V nodal pathway were: 1) discontinuous A-V nodal conduction curves, 2) PSVT with different antegrade conduction times (A-H intervals) (table 1), 3) intra A-V nodal re-entrant PSVT (PSVT #3) with P waves occurring simultaneously with QRS during PSVT.

It has been demonstrated that changes in the cycle length of PSVT in patients with Wolf-Parkinson-White syndrome can occur when ipsilateral functional bundle branch block develops, reflecting an increase in the anatomic size of the circus movement. Recently, Denes et al. also demonstrated that the presence of bilateral anomalous pathways could allow changing cycle lengths of PSVT in a patient with pre-excitation. They described a patient with both a right-sided anomalous pathway (capable of both antegrade and retrograde conduction) and a left-sided pathway (capable of only retrograde conduction). Two types of PSVT were manifest, one utilizing the right-sided anomalous pathway for antegrade conduction and the left-sided pathway for retrograde conduction. The second type of PSVT utilized the normal pathway for antegrade conduction and the left-sided
anomalous pathway for retrograde conduction. The former PSVT was characterized by wide QRS complexes and was faster than the latter.

The present study offers additional mechanisms for changes in cycle length of PSVT in patients with Wolff-Parkinson-White syndrome. In our reported patient with pre-excitation and dual A-V nodal pathways, the following re-entrant loops were observed: 1) antegrade fast pathway and retrograde anomalous pathway, 2) antegrade slow pathway and retrograde anomalous pathway, 3) antegrade slow pathway and retrograde fast pathway.

In patients with Wolff-Parkinson-White syndrome in which PSVT shows sudden variations in cycle length, electrophysiological studies are helpful in delineating arrhythmic mechanisms.

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