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'Dual atrioventricular nodal pathways' in patients with Wolff-Parkinson-White syndrome*

EDWARD L C PRITCHETT, ERIC N PRYSTOWSKY, DAVID G BENDITT, JOHN J GALLAGHER

From the Division of Cardiology, Duke University Medical Center, Durham, North Carolina, USA

SUMMARY 'Dual atrioventricular nodal pathways' were found in five patients who also had the Wolff-Parkinson-White syndrome. All five patients had a re-entrant tachycardia that used the atrioventricular node for conduction in the anterograde direction and an accessory atrioventricular pathway for conduction in the retrograde direction. One of the patients also had a re-entrant tachycardia that originated within the atrium or the atrioventricular node. Dual atrioventricular nodal pathways were identified in three of the five patients during their first electrophysiological study because the effective refractory period of the accessory atrioventricular pathway in the anterograde direction was longer than the effective refractory period of the *fast* atrioventricular nodal pathway. In the other two patients the dual atrioventricular nodal pathways were found only after operative division of an accessory atrioventricular pathway.

Re-entrant tachycardia that uses an accessory pathway may be cured by operative division of the accessory pathway. Tachycardia resulting from re-entry within the atrioventricular node cannot be cured by an operation unless the normal conduction system is divided and a permanent pacemaker implanted. These five patients indicate the importance of determining the aetiology of tachycardia by studying the tachycardia itself and not only the type of atrioventricular conduction present.

Tachycardia that begins and ends suddenly, and during which the QRS morphology on the electrocardiogram is normal is commonly called paroxysmal supraventricular tachycardia.[1–3] In patients with the Wolff-Parkinson-White syndrome, paroxysmal supraventricular tachycardia is a re-entrant arrhythmia which uses the atrioventricular node for conduction in the anterograde direction and an accessory pathway for conduction in the retrograde direction.[4–6] Paroxysmal supraventricular tachycardia may also be the result of re-entry within the atrioventricular node.[7–9] In this latter case dual atrioventricular nodal pathways are usually shown by testing with the extrastimulus technique in the atrium. In this report we describe five patients with the Wolff-Parkinson-White syndrome who also had dual atrioventricular nodal pathways. This study shows the importance of basing criteria for diagnosing tachycardias on the behaviour of the tachycardia rather than on the type of atrioventricular conduction that is present.

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Patients and methods

The patients were among those referred to the Duke University Medical Center for treatment of Wolff-Parkinson-White syndrome and paroxysmal arrhythmias between July 1974 and March 1978. All five patients had a delta wave on the QRS complex of electrocardiograms recorded during sinus rhythm. All five patients also had spontaneous occurrence of a regular tachycardia during which the QRS complex on the electrocardiogram was normal. Patients were identified by reviewing pre-admission records and results of electrophysiological studies performed while they were in hospital. When available, electrophysiological studies performed after surgical correction of ventricular pre-excitation were also reviewed. Illustrations from records of cases 4 and 5 have been previously published.[5 10]

All patients gave informed consent before investigation. Complete medical history, physical examina-

tion, chest x-ray films, and electrocardiograms were obtained on admission. Antiarrhythmic treatment was discontinued at least 48 hours before study.

Laboratory techniques used in these studies have been reported previously.[5 11 12] Electrode catheters were positioned to record simultaneously from the right atrium, coronary sinus, bundle of His, and right ventricle. Heparin (100 units/kg) was given intravenously after catheters were positioned. Atrioventricular conduction in the anterograde and retrograde directions was studied with fixed-rate pacing and the extrastimulus technique. When using the extrastimulus technique, every eighth driven stimulus was followed by a premature stimulus at progressively shorter coupling intervals until the refractory period of the chamber was reached. Re-entrant tachycardias which occurred spontaneously during study or which were induced by programmed stimulation were studied with endocardial mapping and programmed premature beats. Endocardial mapping was performed by recording from the right atrium and atrial septum with a specially constructed bipolar electrode catheter[13] and by recording from the left atrium with a quadripolar catheter in the coronary sinus. Endocardial mapping was used to locate the site of earliest atrial activation during re-entrant tachycardia. Premature ventricular stimuli were coupled to every eighth beat of the tachycardia by programmed stimulation and induced throughout the cardiac cycle.[14] These premature stimuli were used to show atrial pre-excitation (early retrograde atrial activation).

Standard surface electrocardiogram leads and intracardiac electrograms were recorded on magnetic tape at $3\frac{3}{4}$ in/s and written on paper at 200 or 250 mm/s by a Siemens Mingograf ink-jet recorder. Refractory periods were measured with an interactive computer programme.[15] Using this programme independent observers have made measurements that were reproducible within 2 ms.

Definitions

S_1, A_1, H_1, and V_1 respectively refer to the stimulus, atrial electrogram, His bundle electrogram, and ventricular electrogram of the basic drive complex. A_2, H_2, and V_2 refer to the atrial electrogram, His bundle electrogram, and ventricular electrogram caused by the extrastimulus S_2. A_{E1} and H_{E1} refer to the atrial electrogram of the first atrial echo beat and the His bundle electrogram that it initiates. All echoes specifically referred to in *this* paper were initiated by conduction in the retrograde direction over an accessory atrioventricular pathway.

Accessory atrioventricular pathway: An accessory atrioventricular muscle bundle.[16]

Re-entrant tachycardia which used the atrioventricular node for conduction in the antegrade direction and an accessory atrioventricular pathway for conduction in the retrograde direction. A tachycardia which satisfied at least one of the following criteria:

(1) The direction of atrial activation (determined using the technique of endocardial mapping) was retrograde and eccentric,[13] that is atrial activation appeared not to begin at the atrioventricular node.

(2) Bundle-branch block in the ventricle with the accessory atrioventricular pathway lengthened the ventriculoatrial conduction time by 25 ms or more.[17]

(3) Premature ventricular beats pre-excited the atrium during tachycardia at a time when the His bundle was refractory[14] and the sequence of atrial activation of the pre-excited P wave was identical to the sequence recorded during tachycardia.

Dual atrioventricular nodal pathways: A type of atrioventricular nodal function characterised by a discontinuous curve of H_1–H_2 intervals generated by the extrastimulus coupling intervals (A_1–A_2).[18] This definition is based on the characteristics of atrioventricular nodal refractoriness, which is indirect evidence. No anatomical evidence of distinct, specialised conduction pathways has been reported.

Published definitions have been used to define the effective and functional refractory periods of the accessory atrioventricular pathway[19], the atrium,[20] the fast atrioventricular nodal pathway[18], and the slow atrioventricular nodal pathway.[18]

Results

All five patients had a re-entrant tachycardia that used the atrioventricular node for conduction in the anterograde direction and an accessory atrioventricular pathway for conduction in the retrograde direction. In cases 1 and 2 (Table) continuous block of conduction in the anterograde direction over the accessory pathway was present with the pacing cycle length used to measure refractory periods. All basic driven and premature complexes at all coupling intervals were conducted with normal QRS appearances. In case 1 tachycardia was initiated by premature atrial stimuli that were conducted over either the fast or slow atrioventricular nodal pathway (Fig. 1). In case 2 tachycardia was initiated only by premature impulses that were conducted over the slow atrioventricular nodal pathway (Fig. 2). For example, premature stimuli with a coupling interval of 320 ms were repeated four times, and H_1–H_2 intervals of 409, 352, 343,

Table *Electrophysiological data*

Case no.	*Pacing site*	*Cycle length*	*AP ERP*	*Atrium*		*Atrioventricular node*				*Upper limit echo zone*	*AP location*
						Fast		*Slow*			
				ERP	*FRP*	*ERP*	*FRP*	*ERP*	*FRP*		
1	RA	600	*	185	250	315	360	275	510	315	Left
	CS	600	*	215	255	310	360	260	490	355	
2	RA	400	*	205	268	307	358	<281	≤423	301	Left
	CS	400	*	210	259	312	343	279	405	323	
3	RA	800	429	235	302	391	471	345	607	399	Right
	CS	800	424	236	254	337	423	313	592	374	
4	RA	400	†	210	245	<245	≤285	NA	NA		3 APs
	RA	300	†	160	205	240	275	<205	≤310		
	CS	400	†	172	225	240	285	<225	≤320		
	CS	300	†	170	210	240	270	<210	≤315		
5	RA	500	†	220	250	310	345	<250	≤370		Right
	CS	500	†	255	290	<290	≤360	NA	NA		

AP, accessory pathway; CS, coronary sinus; ERP, effective refractory period; FRP, functional refractory period; NA, not achieved; RA, right atrium.
*Continuous block of conduction in anterograde direction at this cycle length.
†No accessory pathway present at postoperative study.

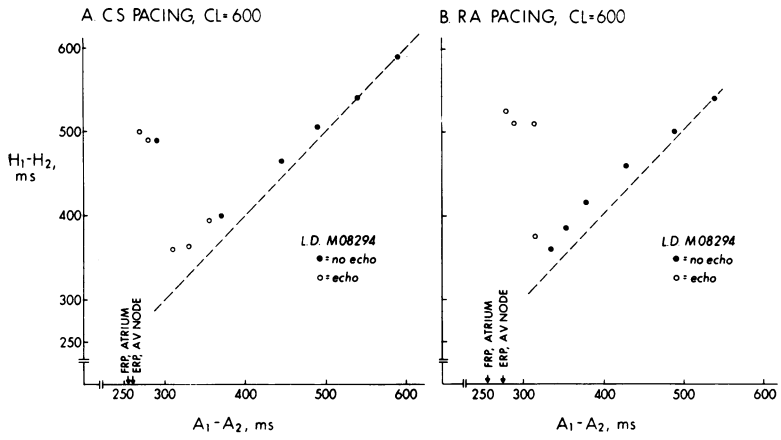

Fig. 1 *Case 1. Refractory period measurement using stimulation at cycle length 600 ms. (A) Coronary sinus pacing site. (B) Right atrial pacing site. At this cycle length there was continuous block of the accessory atrioventricular pathway in the anterograde direction. Therefore all driven impulses and all premature stimuli were conducted to the ventricle with normal QRS morphology. H_1–H_2 intervals could be measured at every coupling interval. The discontinuous curves are a result of dual atrioventricular nodal pathways. Open circles indicate H_1–H_2 intervals that initiated atrial echoes and tachycardia. Note that echoes and tachycardia were initiated by premature stimuli conducted over either the fast or slow atrioventricular pathways.*

and 351 ms were recorded. Only the H_1–H_2 interval of 409 ms initiated an atrial echo. This echo was recorded first in the lateral coronary sinus, confirming that it was initiated by conduction in the retrograde direction over the accessory pathway. All echoes were initiated by conduction in the retrograde direction over the accessory atrioventricular pathway, and the site of earliest atrial activation was always the lateral coronary sinus.

The tachycardia in cases 1 and 3 used only the slow atrioventricular nodal pathway for conduction in the anterograde direction. During tachycardia there was continuous block in the anterograde direction of the fast atrioventricular nodal pathway. Fig. 3 shows initiation of tachycardia in case 3. The AH interval recorded during the tachycardia varied between 250 ms and 330 ms. The RR intervals were similarly variable and the average interval was 491 ms (average heart rate=122 beats/min).

In cases 4 and 5 the diagnosis of dual atrioventricular nodal pathways was made during an electro-

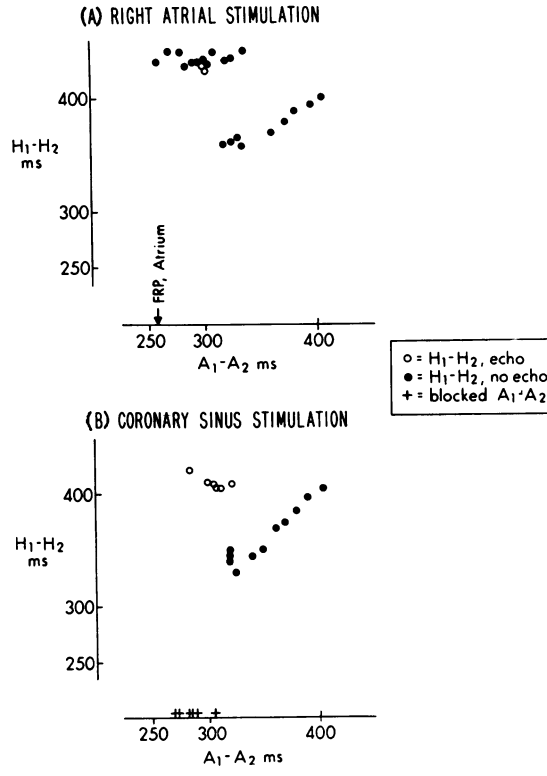

Fig. 2 *Case 2. (A) Refractory period measurement using stimulation of the right atrium at cycle length 400 ms. At this cycle length there was continuous block of the accessory atrioventricular pathway in the anterograde direction. All driven impulses and all premature stimuli were conducted to the ventricle with normal QRS morphology. H_1–H_2 intervals could be measured at every coupling interval. There is a discontinuity in the graph. Atrial echoes were generated only by premature stimuli that were conducted over the slow atrioventricular nodal pathway. Atrial refractoriness prevented the effective refractory period of the slow atrioventricular nodal pathway being determined exactly. (B) Refractory period measured using stimulation of the coronary sinus at cycle length 400 ms. There is again discontinuity in the graph. Only premature stimuli that were conducted over the slow atrioventricular nodal pathway initiated echoes. All atrial echoes were initiated in the lateral coronary sinus.*

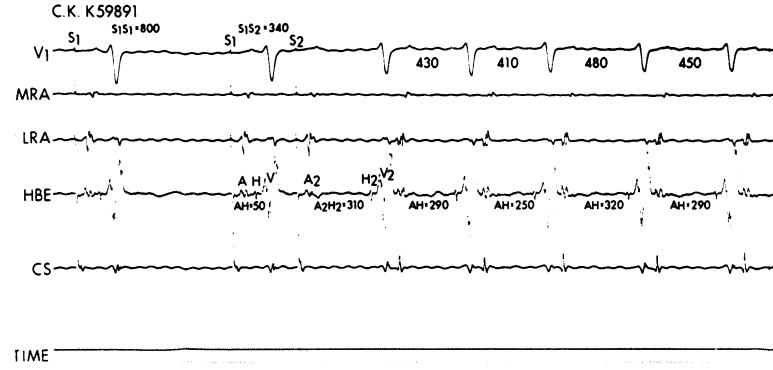

Fig. 3 *Reciprocating tachycardia (case 3). The fast atrioventricular nodal pathway was blocked during the tachycardia. AH intervals were variable and caused irregularity in the rhythm. The mean cycle length of the tachycardia was 491 ms (heart rate 122 beats/minute). CS, coronary sinus; HBE, His bundle electrogram; LRA, lateral right atrium; MRA, mid right atrium; V_1, ECG lead V1.*

physiology study performed several days after operative division of an accessory atrioventricular pathway. Data in the Table from cases 4 and 5 were recorded during the postoperative study. In the preoperative study of case 5 the effective refractory period of the accessory atrioventricular pathway in the anterograde direction was measured at the same cycle length used to measure refractory periods of the atrioventricular node in the postoperative study. During stimulation of the right atrium, the effective refractory period of the accessory pathway in the anterograde direction was 260 ms. During stimulation of the coronary sinus, the effective refractory period of the accessory pathway in the anterograde direction was 280 ms. Since these values were less than the effective refractory period of the fast atrioventricular nodal pathway measured using stimulation from corresponding sites, only slow atrioventricular nodal conduction times could be recorded in the preoperative study from either stimulation site.

Case 5 had two re-entrant tachycardias. One used

the accessory atrioventricular pathway for conduction in the anterograde direction and one used the accessory atrioventricular pathway for conduction in the retrograde direction. This patient was advised that operative division of her accessory atrioventricular pathway would prevent only the re-entrant tachycardia which used the accessory atrioventricular pathway for conduction in the retrograde direction and that the other tachycardia might persist. She requested operation despite this reservation. At the time of the operation both tachycardias were observed. One tachycardia, as expected, used the atrioventricular node for conduction in the anterograde direction and an accessory atrioventricular pathway for conduction in the retrograde direction. The other tachycardia used both the atrioventricular node and the accessory atrioventricular pathway for conduction in the anterograde direction. Endocardial mapping of the right atrium was performed, and the origin of this latter tachycardia was found to be the anterior atrial septum. The accessory atrioventricular pathway was located in the right atrioventricular groove and was successfully divided. Dual atrioventricular nodal pathways were demonstrated by the extrastimulus technique during pacing of the right atrium in a postoperative study. No atrial echo or tachycardia was induced during the postoperative study, and the patient has had no spontaneous tachycardia in the postoperative period.

Refractory periods of the fast and slow atrioventricular nodal pathways were interesting because the effective refractory period and functional refractory period of both fast and slow atrioventricular nodal pathways measured using stimulation of the coronary sinus were equal to or less than the same refractory period measured using stimulation of the right atrium.

Discussion

This study shows the importance of basing criteria for diagnosing tachycardias on the behaviour of the tachycardia and not on the type of atrioventricular conduction present. For example, the presence of an initial delta wave on the QRS complex of an electrocardiogram recorded during sinus rhythm is a clinical marker for an abnormality of atrioventricular conduction in the anterograde direction in patients with the Wolff-Parkinson-White syndrome.[21] Dual atrioventricular nodal pathways are a clinical electrophysiological marker for another abnormality of atrioventricular conduction.[9] Paroxysmal supraventricular tachycardia can occur in patients with either abnormality.[21 22] In this study both abnormalities of atrioventricular conduction were present in five patients with paroxysmal supraventricular tachycardia. It is important to know the correct aetiology because paroxysmal supraventricular tachycardia caused by re-entry over an accessory pathway can be cured by operative division of the accessory pathway. In contrast, the operative treatment of paroxysmal supraventricular tachycardia resulting from re-entry within the atrioventricular node is division of the His bundle and implantation of a permanent pacemaker.

How often these abnormalities of atrioventricular conduction coexist is not known. Wellens has suggested that tachycardia caused by re-entry within the atrioventricular node should occur in patients with the Wolff-Parkinson-White syndrome with an incidence similar to that seen in the general population.[24] Simultaneous occurrence has been inferred from alternating cycle lengths recorded during tachycardia,[23] but well studied and documented cases are rare.[25] The prevalence of dual atrioventricular nodal pathways was 10 per cent in a large study of patients with conduction system disease[18] and this figure is consistent with the finding of dual atrioventricular nodal pathways in two of 19 (cases 4 and 5 of this report) consecutive patients with the Wolff-Parkinson-White syndrome studied after surgical correction of ventricular pre-excitation.[26]

In many patients with Wolff-Parkinson-White syndrome, a diagnosis of dual atrioventricular nodal pathways cannot be made.[6] Discontinuities in the curve of H_1–H_2 intervals generated by changing coupling intervals A_1–A_2 can only be shown in patients whose accessory atrioventricular pathway has an effective refractory period in the anterograde direction that exceeds that of the fast atrioventricular nodal pathway. Cases 4 and 5 are examples of this. In these patients dual atrioventricular nodal pathways were identified only after operative division of the accessory pathway.

We have seen one additional patient with the Wolff-Parkinson-White syndrome who was suspected of having dual atrioventricular nodal pathways because tachycardia with two distinct cycle lengths was observed. The sequence of retrograde activation of the atrium was identical during tachycardias with the two cycle lengths and began in the lateral left atrium; the only difference between the two tachycardias was the length of the AH interval. Sudden transitions from the slow to the fast tachycardia may occur both spontaneously and after programmed ventricular stimuli.[25 27]

Study by the atrial extrastimulus technique may suggest that a complex form of re-entrant tachycardia is present.[25] When refractory periods were measured with the extrastimulus technique during

pacing of the atrium in cases 1 and 3, atrial echoes were initiated by premature stimuli that were conducted over both the fast and slow atrioventricular nodal pathways. In most descriptions of tachycardia caused by re-entry within the atrioventricular node, atrial echoes were initiated by premature stimuli which were conducted only over the slow atrioventricular nodal pathway.[7 8] Note that Fig. 2 is identical to figures made from data recorded in patients with tachycardia resulting from re-entry within the atrioventricular node and dual atrioventricular nodal pathways.[7 8] Endocardial mapping with a catheter in the coronary sinus was required to demonstrate the retrograde atrial activation from an accessory atrioventricular pathway.

Electrophysiological testing has been very useful in the evaluation of atrioventricular conduction, in determining the aetiology of arrhythmias, and in providing the basis for development of surgical techniques to manage arrhythmias that are refractory to control with drugs.[28] The results of this study clearly show the important distinction between abnormalities of atrioventricular conduction and the arrhythmias that accompany them. Criteria for the diagnosis of arrhythmias must be based on study of the arrhythmias themselves.

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Requests for reprints to Dr Edward L C Pritchett, PO Box 3477, Duke University Medical Center, Durham, North Carolina 27710, USA.