Clinical and Electrophysiologic Studies on the Wolff-Parkinson-White Syndrome in Aged Cases

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SUMMARY

Clinical and electrophysiologic studies were performed in 10 aged cases (50–82 years of age) with WPW syndrome and following results were obtained: 1) Episodes of paroxysm of supraventricular tachyarrhythmias were observed in 7 and complication of sinus node dysfunction was noted in 3. 2) P-delta intervals on electrocardiogram exceeding 0.12 sec were observed in 4 cases. 3) Patterns of normalization of QRS complex in 4 cases were characterized by tachycardia-dependent in 2 and bradycardia-dependent in 2. 4) Effective refractory period of the accessory pathway (AP) in antegrade direction exceeded that of A-V node in 5 cases and the former exceeding 500 msec was observed in 2 cases. The relations between the changes of the electrophysiologic properties of the AP and altered clinical manifestations of the WPW syndrome in aged cases were discussed.

Additional Indexing Words:

Accessory pathway Sinus node dysfunction Effective refractory period Alternating normalization of QRS complex Phase 4 block

RECENT advances in electrophysiologic studies utilizing pacing technique have contributed to our understanding of the Wolff-Parkinson-White (WPW) syndrome and related arrhythmias.^{1)–4)} However, only a few electrophysiologic studies on WPW syndrome in aged subjects have been reported.^{2), 5)}

In this study, the alteration of clinical manifestations of WPW syndrome in 10 aged cases was studied and these modifications were related to the

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changes of electrophysiologic properties of the accessory pathway (AP) and the atrioventricular (AV) node. Furthermore, sinus node function was assessed in these patients, since it is conceivable that "overdrive suppression"⁶⁾ of the sinus node by paroxysm of supraventricular tachyarrhythmias (SVT) may be augmented in aged patients.⁷⁾

METHODS

Study group:

Ten aged patients with recent electrocardiographic documentation of WPW syndrome were studied (Table I). Their ages ranged from 50 to 82 years, with an average of 69.0 years. One case was in 6th decade, 4 in 7th, 4 in 8th, and 1 in 9th decade. Three cases were male and 7 were female.

The diagnostic criteria for WPW syndrome^{1),8)} used in this study were as follows: 1) P-R interval of 0.12 sec or less (in the presence of normal P waves), 2) QRS duration of 0.11 sec or more, and 3) a distinct delta wave slurring in the initial part of QRS complex. In cases with P-R interval exceeding 0.12 sec, the diagnosis of WPW syndrome was confirmed when an electrocardiogram (ECG) obtained successively or on another occasion showed a definitely longer P-R interval, narrower QRS and absence of delta wave.

Methods:

In 10 patients clinical manifestations including documented history of paroxysm of SVT, ECG findings and symptoms associated with SVT were studied. ECG during and after spontaneous cessation of SVT were recorded on magnetic tape to determine the presence or absence of sinus node dysfunction.

Electrophysiologic studies utilizing technique of intracardiac recordings and programmed single atrial premature stimulation (PAS) and rapid atrial pacing were performed in 8 of them (Cases 1 to 8, Tables I and II). His bundle electrogram and right atrial electrogram were simultaneously recorded with surface ECG (Leads I, aV_F, V₁) by an oscillographic recorder (Electronics for Medicine, DR-12) and a magnetic tape recorder. Effective refractory period (ERP) of the AV node and AP in antegrade direction was determined by programmed single atrial premature stimulation (PAS) method during basic sinus rhythm. Sinus node function was also assessed by measuring "sinus node recovery time"^{6),7)} following abrupt cessation of rapid atrial pacing or duration of sinus pause after spontaneous cessation of paroxysm of SVT during pacing study. Details of the method of electrophysiologic study have been described elsewhere.^{7),9)}

RESULTS

I. Clinical and electrocardiographic findings (Table I)

1. Incidence of paroxysm of SVT and associated symptoms

Recent episodes of SVT were documented in 7 patients and SVT has recurred in 6, while in 1 patient (Case #7) the episode of SVT did not recur after recent development of sinus brady-arrhythmia associated with occasional

Table I. Clinical and

Case No.	Name (HBE No.)	Age & Sex	Types of WPW	Mode of Normalization of QRS Complexes
1	H. W. (#204)	50 M	A	intermittent normalization
2	H. O. (#201)	65 F	A	alternating normalization (bradycardia-dependent)
3	H. S. (#202)	67 M	A	persistent WPW pattern
4	T. K. (#224)	70 F	A	alternating normalization (tachycardia-dependent)
5	K. Hi. (#51)	71 F	A	bradycardia-dependent normalization
6	K. Ho. (#60)	74 F	A	persistent WPW pattern
7	T. F. (#111)	76 F	B	tachycardia-dependent normalization
8	M. K. (#183)	82 F	B	intermittent normalization
9	S. M.	66 F	B	intermittent normalization
10	S. Y.	69 M	B	intermittent normalization
	Range	50-82		
	Mean	69.0		

Abbreviations: M=male, F=female, SVT=supraventricular tachyarrhythmia, P-R int.=P-R interval observed during normalization of QRS complexes, PAT=paroxysmal atrial tachycardia,

sinus arrest and junctional escape beats. Fainting occurred at the time of spontaneous cessation of SVT in 1 patient (Case #8) and prolongation of sinus pause was recorded immediately following abrupt cessation of SVT in 2 (Cases #1 and #8).

2. Types of WPW syndrome

According to the classification of Rosenbaum,¹⁰⁾ 6 cases were of type A and 4 cases of type B.

3. Patterns of normalization of QRS complexes¹¹⁾ (Figs. 1-4)

WPW pattern was persistent in only 2 cases (Cases #3 and #6) and it was intermittent finding in 4 cases (Cases #1, #8, #9, and #10), while normalization of QRS complexes was observed in every other beat (alternating normalization of QRS complexes) in 2 cases (Cases #2 and #3, Figs. 1 and 2), and bradycardia-dependent normalization (Case #5, Fig. 3), or tachycardia-dependent normalization of QRS complex (Case #7, Fig. 4) was noted in other 2 cases.

4. P-delta intervals or P-R intervals on ECG (Fig. 5)

P-delta intervals associated with WPW pattern in 10 cases ranged from 0.08 sec to 0.16 sec, an average being 0.116 ± 0.025 (SD) sec. P-delta inter-

Electrocardiographic Findings

Electrocardiographic Findings			
Types of SVT	Sinus Node Dysfunction	P-delta (sec)	P-R int. (sec)
PAT	Sinus arrest following SVT	0.11	0.16
None	None	0.16	0.22
PAT	None	(*0.12) **0.14	
Af	None	0.11	0.14
PAT	None	0.11	0.23
PAT	None	0.11	
H/O PAT, Af-SVT ceased	Sinus arrest, junctional rhythm	0.13	0.20
PAT	Sinus arrest, syncope	0.08	0.16
None	None	0.08	0.14
None	None	0.14	0.17
	mean±SD	0.08—0.16 0.116±0.025	0.14—0.23 0.178±0.033

Af=atrial fibrillation

* ECG on June 9, 1973. ** ECG on March 3, 1976

vals exceeded 0.12 sec in 4 cases, they were 0.13 sec in 1, 0.14 sec in 2, and 0.16 sec in 1 case.

P-R intervals associated with normal QRS complex in 8 cases ranged from 0.14 sec to 0.23 sec, with an average of 0.178 ± 0.033 (SD) sec, in 2 patients it exceeded 0.21 sec.

II. Electrophysiologic study

1. His bundle electrograms (HBE) and PAS study

Results of electrophysiologic studies in 8 cases are shown in Table II.

In 2 cases (Cases #1 and #5), since QRS complex remained normal at the time of the study, the conduction over AP was not observed. In Case #1, ERP of AP could not be determined because of frequent provocation of SVT accompanying narrow QRS complexes by PAS with relatively short coupling intervals.

In other 2 cases with WPW pattern (Cases #3 and #6), antegrade conduction over AP occurred up to the ERP of the right atrium and QRS complexes could not be normalized either spontaneously or by PAS at the time of the study.

In the remaining 4 cases (Cases #2, #4, #7, and #8), normalization of

Fig. 1. Changes of QRS complexes in lead V₁ in Case #2.

- (a) All QRS complexes showed fusion beats, when P-P intervals were 0.66 sec (Feb. 16, 1976).
- (b) Normalization of QRS complexes was observed in every other beat, when P-P intervals were 0.76 sec (Jan. 30, 1976).
- (c) All QRS complexes were normalized with P-P intervals which ranged from 0.98 to 1.0 sec (Feb. 21, 1976).

Fig. 2. Changes of QRS complexes in lead II in Case #4.

- (a) All QRS complexes showed WPW pattern, when P-P intervals were 0.60 sec.
- (b) Normalization of QRS complexes was observed in every other beat, when P-P intervals were shortened to 0.52 sec.

Fig. 3. Relation between preceding P-P interval and shape of QRS complex in Case #7 (lead II), showing bradycardia-dependent normalization of QRS complex.

- (a) All QRS complexes were WPW pattern.
- (b) P-P interval which was equal to or exceeding 0.98 sec was followed by normalized QRS complex.
- (c) Distribution of P-P intervals and shapes of QRS complexes of ECG shown in (b).

Fig. 4. Relation between preceding P-P interval and shape of QRS complex in Case #5 (lead II), showing tachycardia-dependent normalization of QRS complex.

- (a) Frequent premature atrial contractions with shorter P-P intervals were followed by narrow QRS complexes.
- (b) Distribution of P-P intervals and shapes of QRS complexes of ECG shown in (a).

QRS complex could be obtained with PAS technique. In these 4 cases, in whom ERP of both AP and A-V node in antegrade direction could be determined, ERP of AP exceeded that of the A-V node.

ERP of AP determined in 6 cases ranged from less than 160 msec (Case

Fig. 5. Distribution of P-R intervals in 8 cases (upper panel) and P-delta intervals in 10 cases (lower panel). Corresponding values in each case are connected by solid lines.

Table II. Results of

Case No.	Intervals on HBE (msec)				
	P-P	P-A	A-H	P-delta (P-R)	H-delta (H-V)
1	Normalized QRS only				
	(650)	(20)	(70)	(130)	(40)
2	960	40	140	160	-20 (40)
3	WPW pattern only				
	710	40	90	140	10
4	580	30	55	85	0
5	Normalized QRS only				
	(985)	(30)	(130)	(205)	(45)
6	WPW pattern only				
	660	90	130	110	-10
7	1,306	33	114	130	-16
8	1,150	30	*	80	*
Range					
Mean					

Numbers in parenthesis indicate intervals observed during normalization of QRS complex.

Abbreviations: RA=right atrium, AVN=atrioventricular node, AP=accessory pathway, SVT=supraventricular tachyarrhythmias, PAS=premature atrial stimulation, PAT=paroxysmal atrial

#6) to 810 msec, an average being 413 msec. ERP of AP exceeding 300 msec was noted in 4, of whom it exceeded 500 msec in 2 cases (Cases #2 and #7).

In 5 cases, antegrade conduction over the A-V node occurred up to the ERP of the right atrium, which ranged from 160 to 390 msec. In the other 2 cases (Cases #1 and #7), ERP of A-V node in antegrade direction were 360 msec and 540 msec, respectively. P-delta interval exceeding 120 msec during basic sinus rhythm was recorded in 3 cases, in one of them A-H interval was also prolonged, while it was normal in the other 2 cases.

From ECG and electrophysiologic studies, the presence of "bundle of Kent" was suggested in 6 cases in whom recording of HBE and PAS study were performed during WPW pattern.

2. SVT and sinus node function (Table II)

Paroxysm of SVT was initiated by properly timed single atrial stimulation in 4 cases.

Sinus node recovery time after rapid atrial pacing or sinus pause following spontaneous cessation of SVT observed during the study was prolonged in 2, while it was normal in 5 other cases. In 1 patient sinus node recovery

Electrophysiologic Studies

Atrioventricular Effective Refractory Period (msec)			SVT Initiated by PAS	Sinus Node Recovery Time
RA	AVN	AP		
Undetermined	360	Undetermined	(+) PAT	Sinus arrest with junctional escape Normal
390	<390	510		
260	Undetermined	<260	(+) AF	Normal
200	<200	330	(+) PAT	Normal
320	<320	Undetermined		Normal
160	<160	<160		Normal
440	540	810		Undetermined due to premature ectopic P wave
300	<300	390	(+) PAT	Sinus arrest for 2.5 sec
160-440	<160-450	<160-810		
295	324	413		

tachycardia, AF=atrial flutter

* His electrogram within QRS complex

Fig. 6. Changes of QRS complexes during basic sinus rhythm and following premature atrial stimulation (PAS) in Case #2.

During sinus rhythm, P-P interval of sinus cycles (SCL) is plotted on abscissa and R-R interval (V_1-V_1) on ordinate and during PAS study coupling interval of PAS (A_1-A_2) on abscissa, R-R interval (V_1-V_2) on ordinate.

time could not be estimated because of occurrence of ectopic premature atrial contraction immediately after the cessation of rapid pacing.

III. Presentation of cases showing unusual electrophysiologic properties of AP

In Case #2 (Fig. 6), AP became refractory in other beat during slow rate with P-P interval exceeding 900 msec, while PAS with shorter coupling interval was propagated over AP up to the refractoriness of AP (510 msec). Fig. 7 illustrates a possible mechanism of alternation of narrow and wide QRS complexes, while P-P interval remains exactly constant. The first beat shown in the ladder diagram in Fig. 7 is a fusion beat. In the second beat, A-V transmission of the atrial impulse is feasible only through the A-V nodal pathway, thus giving rise to a narrow QRS complex. The conduction over Kent bundle in this beat is assumed to be blocked because of spontaneous slow diastolic depolarization (phase 4 block). If Kent bundle is depolarized in the retrograde direction from the ventricle, renewal of the action potential

Fig. 7. Intracardiac electrograms in Case #2 showing alternating normalization of QRS complex (see text). K: Kent bundle, H: A-V node-His bundle, HRA: high RA electrogram

Atrial Pacing (150/min)

Fig. 8. Intracardiac electrograms in Case #2 during rapid atrial pacing (see text).

Arrow in the middle of the record indicates the stimulus which failed to capture the atrium. Abbreviations as in Fig. 7.

and resetting of cycle of Kent bundle can occur, thus permitting subsequent antegrade conduction, resulting again in a fusion beat in the third beat.

A record obtained in Case #2 during rapid atrial pacing at a rate of 150 beats/min is shown in Fig. 8, which illustrates 1:1 A-V conduction except a pacing stimulus in the middle of the record, indicated by an arrow, which failed to capture the atrium. It should be noted that QRS complex showed abrupt normalization after the non-captured stimulus. This phenomenon can be explained by the same assumption mentioned above. In the diagram shown in the bottom of Fig. 8, the initial 3 successive beats are fusion beats with predominant conduction over AP, while the P wave immediately after the pacing stimulus that failed to activate the atrium is followed by narrow QRS, indicating the failure of conduction through AP. It can be assumed

Fig. 9. Relation between coupling intervals of PAS and shapes of QRS complexes in Case #7.

- PAS with coupling interval of 650 msec was followed by wide QRS complex.
- When coupling interval was shortened to 440 msec, subsequent QRS complex was narrow.
- When coupling interval was further shortened to 410 msec, subsequent QRS complex became wide.

that this failure of conduction is due to mechanism of phase 4 block. Subsequent block of atrial impulses through AP may be explained by phase 3 block due to prolonged REP (510 msec).

Unusual electrophysiologic properties of AP was also observed in another case. In Case #7, with the i.v. injection of 1.0 mg of atropine basic P-P interval changed from 1,306 msec to 1,093 msec and ERP of A-V node, AP, and right atrium changed from 540 to 440 msec, from 810 to 630 msec, and from 440 to 360 msec, respectively. PAS study after atropine injection (Fig. 9) revealed that PAS with coupling interval between 630 and 420 msec was followed by narrow QRS complex, while PAS with coupling interval between 410 and 380 msec again showed wide QRS complex, indicating unexpected conduction of Kent bundle (supernormal conduction of AP).

DISCUSSION

Studies on the natural history of WPW syndrome^{8),12)} suggest that electrocardiographic signs of preexcitation disappear with increasing age in a significant number of patients. Clinical observation of diminished incidence of tachycardia with increasing age was attributed to progressive lengthening of refractory period with ultimate block of AP,⁵⁾ while electrophysiologic study reported by others²⁾ did not confirm any significant difference in the length of refractory period between different age groups. In the present study on 10 aged patients, a mean value of the length of ERP was 413 msec, which is longer than those previously reported for any age group,^{2),5)} and this finding may support the hypothesis of progressive lengthening of refractory period with increasing age.

The incidence of documented paroxysm of SVT in this study was 70%, which indicates that the recurrence of SVT is still a major clinical problem in aged patients with WPW syndrome. The high incidence of paroxysm of SVT may have additional clinical significance in aged patients, since overdrive suppression of the sinus node by SVT may be exaggerated when aged patient with WPW syndrome develops sinus node dysfunction, and this was recognized in 3 cases in the present study. In addition to various mechanisms which are responsible for initiation of sustained SVT involving AP and/or A-V node in WPW syndrome, a reentry mechanism involving the sinoatrial junction initiated by ectopic premature atrial contraction may be another possibility in of SVT sick sinus syndrome associated with WPW syndrome.

Case of preexcitation with prolonged P-delta interval due to slow Kent bundle conduction has been reported.¹³⁾ In this study 4 cases showed prolonged P-delta interval exceeding 0.12 sec. In 3 cases (Cases #2, #7, and

#10), diagnosis of WPW syndrome could be verified by the documentation of longer P-R intervals and constant P-J intervals with normalized QRS complexes on other occasions. In remaining 1 case (Case #3), lengthening of P-delta interval from 0.12 sec to 0.14 sec at 2 years' interval was recorded (Table I). In addition, results of electrophysiologic study performed in 3 cases (Cases #2, #3, and #7) were compatible with the presence of Kent bundle as the anatomical basis of widened QRS with initial slurring in these cases. Marked prolongation of ERP of AP in Cases #2 and #7, 510 msec and 810 msec, respectively, is in accordance with a case showing slow Kent bundle conduction reported by Rosen et al.¹³⁾

QRS complexes are often normalized in cases with WPW syndrome. Recording of HBE during spontaneous or induced normalization of QRS complexes suggested that QRS complexes became normal in the following circumstances¹¹⁾; 1) when AP failed to conduct at high atrial rates and also early premature beats; 2) AP became refractory by virtue of phase 4 depolarization after long pauses and in slow rate; 3) when the origin of the ventricular impulses was situated within the His bundle below the take-off of AP; and 4) in circumstances in which conduction velocity through A-V node-His axis supercede that of AP. Among 10 aged cases included in the present study, normalization of QRS complex could be related to tachycardia in 2 cases (Cases #4 and #7) and to bradycardia in 2 cases (Cases #2 and #5). Prolonged ERP over 500 msec in Cases #2 and #7, suggesting altered electrophysiologic properties of AP, are in accordance with observation of normalization of QRS complexes on ECG.

In conclusion, it may be stated that although occurrence of changes of clinical and electrocardiographic manifestations in some aged patients with WPW syndrome was shown in this small series, more systemic studies, including preferably electrophysiologic study, are mandatory to elucidate the natural history of WPW syndrome.

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