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Ventriculo-atrial conduction time during reciprocating tachycardia with intermittent bundle-branch block in Wolff-Parkinson-White syndrome[1]

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Records from patients with the Wolff-Parkinson-White syndrome were reviewed with particular emphasis on the occurrence of bundle-branch block aberration during reciprocating tachycardia and the significance of this observation with respect to accessory pathway location. Increase by greater than 25 ms in the ventriculo-atrial interval during reciprocating tachycardia with bundle-branch block, when compared to reciprocating tachycardia with normal intraventricular conduction, occurred only with right or left free wall accessory pathways. No patient with a septal accessory pathway proven by epicardial mapping showed a ventriculo-atrial interval prolongation greater than 20 ms during bundle-branch block aberration. Measurement of ventriculo-atrial interval during bundle-branch block aberration also helped to diagnose accessory pathways with unidirectional retrograde conduction. Measurement of cycle length changes alone with failure to measure AH and HV intervals as well as ventriculo-atrial times, may give misleading information. In one patient increase in cycle length during left bundle-branch block was the result of prolonged HV interval rather than prolonged ventriculo-atrial interval. In another patient cycle length remained the same during bundle-branch block while the ventriculo-atrial interval increased by an increment identical to the decrease in AH interval.

Techniques have recently been developed for epicardial mapping of ventricular excitation (Durrer and Roos, 1967) and for surgical division of accessory pathways (Sealy *et al.*, 1974) in patients with the Wolff-Parkinson-White syndrome. Application of these techniques demands precise localization of accessory pathways before operation. Bundle-branch block occurs in supraventricular tachycardia of any mechanism when the relative refractory period of either bundle-branch system is encountered (Damato and Lau, 1970). When associated with the Wolff-Parkinson-White syndrome, increase of the cycle length during reciprocating tachycardia associated with bundle-branch block has been reported to implicate participation of an accessory pathway in the ventricle showing the bundle-branch block (Motté *et al.*, 1973; Coumel and Attuel, 1974). In this communication we make some additional observations about this phenomenon and stress the need to measure ventriculo-atrial conduction time correctly to interpret cycle length changes. This has been commented on previously (Neuss, Schlepper, and Thormann, 1975).

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Methods

Between January 1968 and January 1976, 125 patients with the Wolff-Parkinson-White syndrome were studied in the Clinical Electrophysiology Laboratory of the Duke University Medical Center. Records from these patients are the source of this study. All patients gave informed consent for these studies. Antiarrhythmic therapy was discontinued 48 hours before the first day of study and all electrophysiological phenomena described in this report were recorded before the administration of any antiarrhythmic treatment during the study. Four catheters were positioned for intracardiac recording and stimulation. A 6F tripolar catheter was

introduced percutaneously from the right femoral vein and positioned across the tricuspid valve for recording the His bundle electrogram as described by Schlerlag *et al.* (1969). A 6F quadripolar or hexapolar catheter was introduced from a left antecubital vein into the coronary sinus for recording left atrial and posterobasilar left ventricular electrograms. Recording and stimulation of the right atrium and right ventricle were done by two 6F quadripolar catheters introduced from the right antecubital vein and right femoral vein, respectively. Heparin 100 units per kg bodyweight was administered after introduction of these catheters.

Intracavitary electrograms were obtained by filtering out frequencies below 50 Hz and above 1 kHz. Simultaneous recordings of these electrograms and surface electrocardiogram lead V1 were recorded on magnetic tape at a speed of 9·5 cm/s. Refractory periods were determined and arrhythmias were induced using a stimulator[1] using photoelectric isolation which delivered programmed impulses of 2 ms duration of twice diastolic threshold. The tapes were played back on a Mingograf 800 8 channel ink jet recorder at paper speeds of 100 mm/s or 200 mm/s.

Fifty-one of these patients have subsequently undergone thoracotomy with epicardial mapping (Durrer and Roos, 1967) being performed before attempted surgical division of the accessory pathway (Sealy *et al.*, 1974). The operative data enabled classification of the accessory pathway location as left ventricular free wall, right ventricular free wall, septal, or multiple locations. All patients with multiple pathways and all patients who showed only one QRS configuration during electrophysiological study were excluded. One patient was excluded because of coexistence of Kent and Mahaim tracts (Tonkin *et al.*, 1975a). In the remaining 23 patients, the following measurements were made during reciprocating tachycardia with both normal intraventricular conduction and bundle-branch block (in two patients, reciprocating tachycardia did not occur but measurements could be made on single atrial echo beats). Measurements were made during one paroxysm of reciprocating tachycardia to avoid slight changes in cycle length, which may occur between paroxysms.

Definition of terms

AH interval

This interval is from the rapid component of the bipolar medial right atrial electrogram to the onset of the His bundle deflection, both electrograms being recorded by the catheter positioned across the tricuspid valve.

HV interval

This interval is from the onset of the His bundle deflection to the earliest recording of ventricular activation, either on a surface electrocardiogram lead or intracardiac electrogram.

VA interval

This interval is from the earliest recording of ventricular activation to the rapid component of the bipolar atrial electrogram, whenever possible this being recorded from the site closest to the accessory pathway.

Results

Details of the 18 patients with free wall accessory pathways are shown in Table 1. The 15 patients with a left free wall accessory pathway who had left bundle-branch block aberration during reciprocating tachycardia all showed at least a 25 ms prolongation of the ventriculo-atrial interval during that aberration. However, none of the 12 patients with a left free wall accessory pathway who had right bundle-branch block aberration during reciprocating tachycardia had prolongation of the ventriculo-atrial interval during that aberration.

These observations are shown in Fig. 1 which shows reciprocating tachycardia in a patient with a left free wall accessory pathway (Case 3, Table 1) proven by epicardial mapping. During reciprocating tachycardia with normal conduction and right bundle-branch block, the ventriculo-atrial intervals are identical. In reciprocating tachycardia with left bundle-branch block, the ventriculo-atrial interval is prolonged by 50 ms. Minor variations in cycle length are caused by variations in the AH interval. Three patients with right free wall accessory pathways showed bundle-branch block during reciprocating tachycardia. Two of these three had right bundle-branch block aberration and their ventriculo-atrial intervals were prolonged by 30 and 55 ms. One had left bundle-branch block and there was no change in ventriculo-atrial interval.

Table 2 lists 4 patients in whom the site of accessory pathway was presumed to be septal based on results of epicardial mapping and the surgical incision. No patient had a ventriculo-atrial interval prolongation greater than 20 ms during bundle-branch block aberration.

In addition to providing useful information regarding the location of accessory pathways, the observation that bundle-branch block aberration during reciprocating tachycardia lengthens the

[1]Designed by Michael Feezor, Ph.D., and built by Philip Talbert.

TABLE 1 *Bundle-branch block during reciprocating tachycardia in patients with free wall accessory pathways*

Case No.	RT with normal intraventricular conduction			RT with ipsilateral BBB			RT with contralateral BBB			Comment
	CL	HV	VA	CL	HV	VA	CL	HV	VA	
	Left free wall accessory pathways									
1	290	60	170	320	60	200	290	60	170	
2	335	35	190	Did not occur			355	35	190	Initial study,
	440	NR	140	480	NR	180	Did not occur			restudy
3	305	45	170	335	45	220	295	45	170	
4	280	40	180	Did not occur			280	40	180	
5	290	55	160	310	55	210	Did not occur			
	250	55	145	Did not occur			250	55	145	Occurred later in same study
6	365	55	120	Did not occur			365	55	120	
7		50	90		80	215	Did not occur			Measurement from single atrial echo
8		50	95	Did not occur				50	95	Measurement from single atrial echo
9	285	25	135	Did not occur			285	25	135	
10	300	35	175	Did not occur			300	35	175	
11	270	60	140	310	60	215	265	60	140	
12	275	45	150	335	65	200	285	60	145	
13	300	65	140	Did not occur			295	60	150	
14	290	55	160	335	75	220	Did not occur			
15	350	50	150	385	50	185	Did not occur			
	Right free wall accessory pathways									
16	305	50	190	340	50	225	Did not occur			
17	470	45	190	Did not occur			470	45	190	
18	340	45	160	375	45	225	Did not occur			

NR=not recorded.

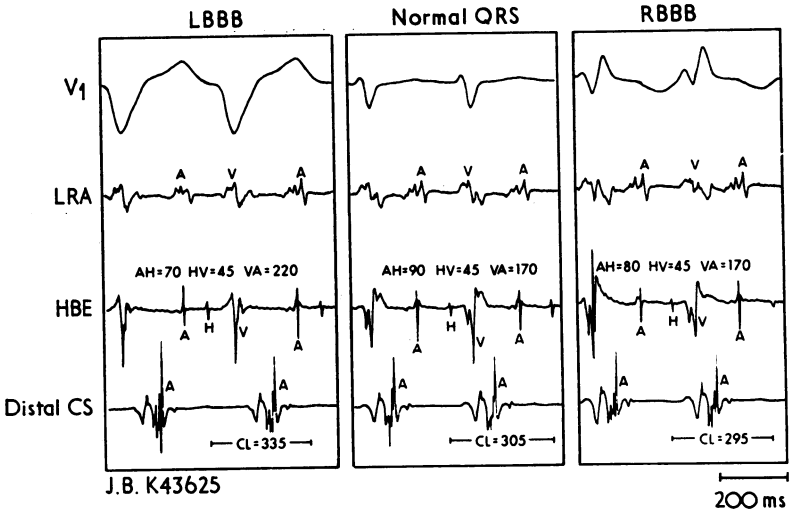

FIG. 1 *Electrophysiological studies during reciprocating tachycardia in a patient with a left free wall accessory pathway. From top to bottom are standard lead V1 (V1), and electrograms from the low lateral right atrium (LRA), His bundle (HBE), and distal coronary sinus (Distal CS). During reciprocating tachycardia with left bundle-branch block aberration, ventriculo-atrial conduction time increased by 50 ms over that observed during reciprocating tachycardia with normal intraventricular conduction and right bundle-branch block aberration. Note that local ventriculo-atrial interval measured in CS does not change with QRS configuration changes. Cycle length discrepancies are caused by variation in AH and HV intervals. Fig. 3, 4, and 5 are labelled similarly.*

TABLE 2 *Bundle-branch block during reciprocating tachycardia in patients with septal accessory pathways*

Case No.	*Normal intra-ventricular conduction*			*RBBB*			*LBBB*		
	CL	*HV*	*VA*	*CL*	*HV*	*VA*	*CL*	*HV*	*VA*
19	275	50	150	275	50	150	Did not occur		
20	280	40	190	280	40	190	Did not occur		
21	270	40	135	290	40	155	270	40	135
22	260	50	130	Did not occur			275	50	145

ventriculo-atrial interval may provide evidence for the existence of bypass tracts with unidirectional retrograde (ventriculo-atrial) conduction properties (Coumel and Attuel, 1974; Neuss *et al.*, 1975; Zipes, DeJoseph, and Rothbaum, 1974; Spurrell, Krikler, and Sowton, 1974). Fig. 2 is the 12 lead electrocardiogram from Case 15 (Table 1). There was no evidence for ventricular pre-excitation at rest or after pacing at multiple atrial sites. Fig. 3 shows simultaneous surface and intracardiac electrograms recorded during reciprocating tachycardia in this patient. The left panel shows tachycardia with left bundle-branch block and cycle length 385 ms. The right panel shows the same paroxysm after spontaneous reversion to reciprocating tachycardia with normal intraventricular conduction. There was a simultaneous decrease in the cycle length of reciprocating tachycardia to 350 ms. Measurement of the respective intervals within the cardiac cycle showed that shortening of the cycle length was entirely at the expense of the ventriculo-atrial interval. These observations, presented in detail elsewhere (Tonkin *et al.*, 1975b), support the conclusion that a left free wall accessory pathway with antegrade block participated in the reciprocating tachycardia. This patient has subsequently undergone epicardial mapping and operation which confirmed the location of the pathway.

Failure to define which segment of the cardiac cycle accounts for the increase in cycle length may give misleading information. Fig. 4 was taken from the records of a patient with frequent tachycardia whose 12 lead electrocardiogram showed intermittent ventricular pre-excitation. The left panel shows tachycardia with left bundle-branch block and cycle length 395 ms. The right panel shows tachycardia with normal QRS and cycle length 365 ms. This observation might suggest that a left-sided accessory pathway participated in the tachycardia (Coumel and Attuel, 1974). However, measurement of the timing of intracardiac events showed that the 30 ms change in cycle length accompanying left bundle-branch block was entirely accounted for by an increase in the HV interval from 65 to 95 ms, while the ventriculo-atrial interval remained unchanged (135 ms). This prolongation of the HV interval, therefore, became a pivotal piece of evidence showing that the distal conduction system participated in the reciprocating tachycardia

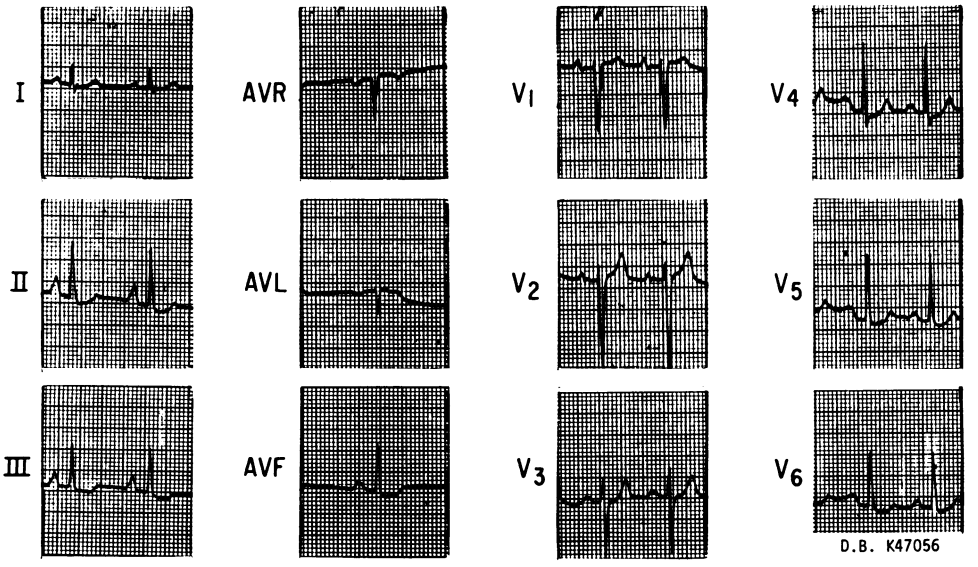

FIG. 2 *Twelve lead electrocardiogram showing no pre-excitation in a patient with an accessory pathway which sustained only retrograde conduction.*

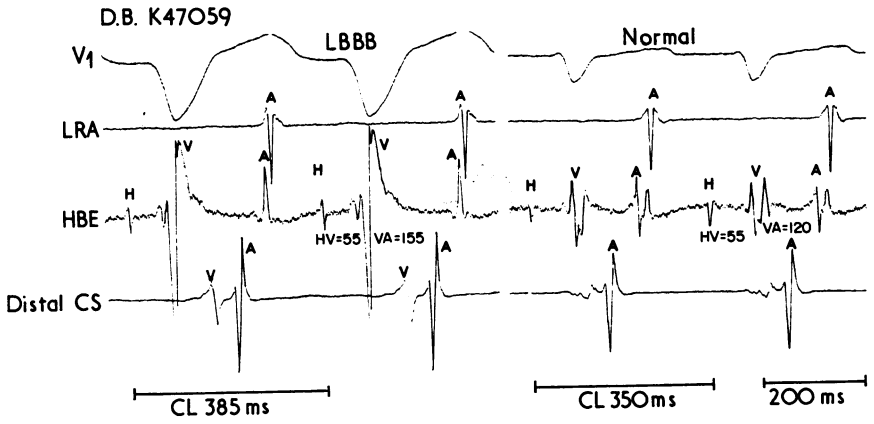

FIG. 3 *Increase in cycle length of reciprocating tachycardia from 350 ms to 385 ms during left bundle-branch block aberration was caused by prolongation of the ventriculo-atrial interval, suggesting the presence of left ventricular accessory pathway. This is from the same patient whose electrocardiogram is shown in Fig. 2.*

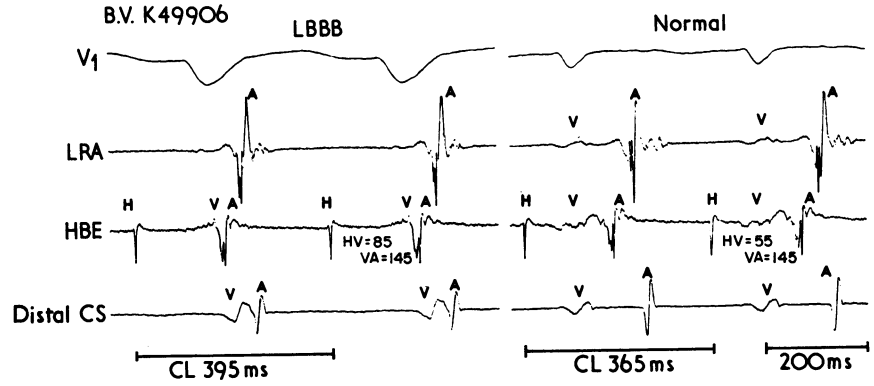

FIG. 4 *Increase in cycle length of reciprocating tachycardia during left bundle-branch block aberration caused by prolongation of HV interval. Failure to measure HV and ventriculo-atrial intervals would lead to false conclusion that there was a left lateral accessory pathway. The accessory pathway was in the septum.*

However, the absence of any change in the ventriculo-atrial interval argued against a left free wall accessory pathway. Atrial activation during reciprocating tachycardia and ventricular pacing began at the atrial septum and spread simultaneously to the left and right atria. This sequence is compatible with reciprocating tachycardia during either atrioventricular node re-entry or participation of a septal accessory pathway (Gallagher *et al.*, 1975; Gallagher *et al.*, 1976). It was, therefore, concluded that the reciprocating tachycardia was passing in an antegrade direction through the AV node and His-Purkinje system and returning by a septal accessory pathway to the atrium.

Fig. 5 shows portions of a single episode of reciprocating tachycardia from another patient who showed normal intraventricular conduction, left bundle-branch block aberration, and right bundle-branch block aberration. The cycle length was identical in all 3 forms of intraventricular conduction. However, during left bundle-branch block aberration the ventriculo-atrial interval increased by 35 ms and the AH interval decreased by the same amount. If cycle length only had been measured

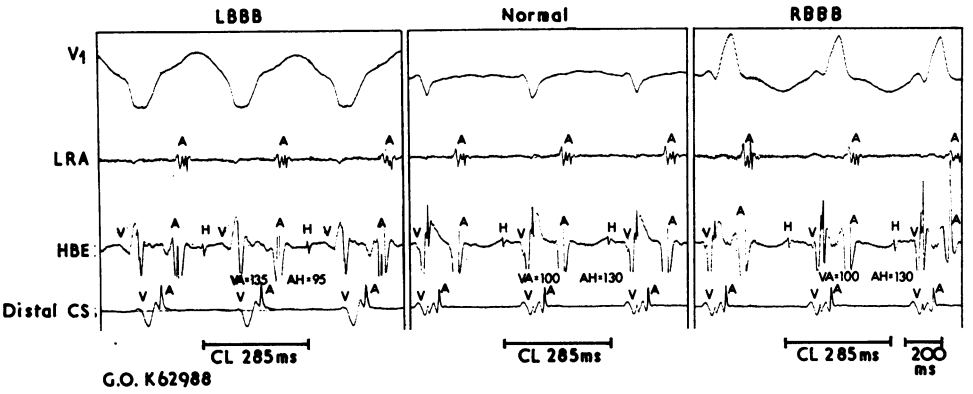

FIG. 5 *Failure of cycle length of reciprocating tachycardia to change during left bundle-branch block in a patient with a left free wall accessory pathway. Increase in ventriculo-atrial interval was compensated by identical decrease in AH interval. Lack of cycle length change by itself suggested AV nodal re-entry or participation of a septal accessory pathway as the mechanism for reciprocating tachycardia, but increase in ventriculo-atrial interval during left bundle-branch block confirms participation of a left free wall pathway.*

this tachycardia would have erroneously been diagnosed as being the result of AV node re-entry or of re-entry over a septal accessory pathway. The sequence of atrial activation during reciprocating tachycardia was again consistent with participation of a left free wall accessory pathway.

Discussion

As surgical treatment of the potentially fatal or medically uncontrollable arrhythmias which are associated with the Wolff-Parkinson-White syndrome becomes more common, correct interpretation of observations which localize accessory pathways will become more important. One such observation is the change in AH, HV, and ventriculo-atrial intervals in reciprocating tachycardia with aberrant conduction as compared with these measurements during reciprocating tachycardia with normal intraventricular conduction. These intervals may be useful in several ways:

(1) Evidence suggesting that an accessory pathway participates in reciprocating tachycardia

Prolongation of either the HV or ventriculo-atrial intervals in reciprocating tachycardia during aberrant intraventricular conduction supports the conclusion that the ventricular myocardium (then the accessory pathway) is a link in the re-entrant circuit. Prolongation of the ventriculo-atrial interval during reciprocating tachycardia caused by re-entry in the AV node is known to occur as the site of re-entry changes (Goldreyer and Bigger, 1971). If this prolongation occurred by coincidence simultaneously with the development of functional bundle-branch block, the result would mimic the events described here. Such a coincidence must be rare and far less common than if the ventriculo-atrial prolongation is caused by the functional bundle-branch block, which is the case in reciprocating tachycardia occurring over an accessory pathway.

(2) Differentiation between free wall and septal accessory pathways

The observation of changes in ventriculo-atrial interval with intermittent bundle-branch block in reciprocating tachycardia represents a potentially useful method for separating patients with septal accessory pathways from those with free wall accessory pathways. This separation is of importance because surgical interruption of accessory pathways in the septum is technically more difficult (Gallagher *et al.*, 1975). In the small group of patients reported here, ventriculo-atrial interval prolongation of 25 ms or more during bundle-branch block in reciprocating tachycardia occurred only in the presence of a free wall accessory pathway in the ventricle with bundle-branch block. Case 22 (Table 2) is of particular interest. Before operation, the accessory pathway was thought to be located just to the left crux. A dissection of this area failed to abolish pre-excitation. The septum was not dissected because of the risk of developing complete heart block. It was concluded that the accessory

pathway was either in the septum or in the epicardial fat behind the crux.

(3) Recognition of accessory pathways with antegrade block

There are unusual cases in which accessory pathways have unidirectional antegrade block but support retrograde conduction in reciprocating tachycardia (Coumel and Attuel, 1974; Neuss *et al.*, 1975; Zipes *et al.*, 1974; Spurrell *et al.*, 1974; Tonkin *et al.*, 1975b). In these cases, the observation that the ventriculo-atrial interval during reciprocating tachycardia prolongs with bundle-branch block may be regarded as proof of participation of the accessory pathway in the re-entry circuit.

To provide data that are diagnostically useful for these purposes, it is important to measure not only cycle length changes but also the AH, HV, and ventriculo-atrial intervals during reciprocating tachycardia. This is illustrated in Fig. 4 and 5. Casual inspection of cycle length change in Fig. 4 suggests the participation of a left free wall accessory pathway in reciprocating tachycardia. However, the ventriculo-atrial interval did not change when left bundle-branch block occurred. The sequence of atrial activation during ventricular pacing and reciprocating tachycardia confirmed the presence of a septal accessory pathway. Similarly, Fig. 5 would be misinterpreted if only the cycle length were measured. In reciprocating tachycardia with left bundle-branch block aberration the ventriculo-atrial interval increased by 35 ms. Simultaneously the AH interval decreased by 35 ms, perhaps because the increased duration of the ventricular segment of the tachycardia pathway allowed recovery of the partially refractory AV node.

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