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M H Lehmann, … , J Dongas, M Akhtar

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Research Article

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Electrophysiologic Mechanisms of Functional Bundle Branch Block at Onset of Induced Orthodromic Tachycardia in the Wolff-Parkinson-White Syndrome

Role of Stimulation Method

Michael H. Lehmann, Stephen Denker, Rehan Mahmud, Patrick Tchou, John Dongas, and Masood Akhtar
Natalie and Norman Soref and Family Electrophysiology Laboratory, University of Wisconsin-Milwaukee Clinical Campus, Mount Sinai Medical Center, Milwaukee, Wisconsin 53233

Abstract

The mechanisms of aberrant conduction at the onset of induced orthodromic tachycardia in the Wolff-Parkinson-White syndrome were analyzed in 20 consecutive patients in whom this tachycardia was initiated by the atrial (A_2) and/or right ventricular (V_2) extrastimulus techniques. Of 13 patients in whom orthodromic tachycardia was induced by the A_2 method, functional right bundle branch block occurred at tachycardia onset in four (31%) and left bundle aberrancy in two (15%), one of whom also manifested right bundle aberrancy. The occurrence of bundle branch block at the onset of tachycardia was linked to aberrant conduction of the initiating A_2 impulse which, in turn, was associated with attainment of relatively short His_1His_2 intervals within the tachycardia initiation zone. Aberrant conduction of A_2 was also more common in patients without manifest preexcitation. In contrast, of 14 patients in whom orthodromic tachycardia was induced by the V_2 method, left bundle aberrancy occurred at the onset of tachycardia in 11 (79%), one of whom manifested right bundle branch block as well. Left bundle aberrancy was more likely to occur when the interval from the initiating V_2 (or macro-reentrant V_3) impulse to the first anterograde His deflection was <300 ms. This suggests that left bundle aberrancy at the onset of orthodromic tachycardia induced by the V_2 method results from concealed retrograde penetration of the His-Purkinje system, with the left bundle being last to recover. Our findings provide the conceptual basis for a physiologic approach to the deliberate induction of specific types of aberrant conduction at onset of orthodromic tachycardia in patients with Wolff-Parkinson-White syndrome.

Introduction

Accessory pathway localization is an important objective during electrophysiologic assessment of patients with Wolff-Parkinson-White syndrome.[1] Of special value in this regard is the quantitation of changes in ventriculoatrial conduction time attending the occurrence of particular types of functional (fx)[2] bundle branch block (BBB) during orthodromic tachycardia (OT) (1, 2). Unfortunately, little is known regarding the means by which one may deliberately induce specific fx BBB patterns during OT using programmed stimulation, other than exhaustive trial and error (2). Moreover, the relationship (if any) between particular mechanisms of OT initiation and the occurrence of aberrant conduction during OT has not been systematically investigated.

To gain insight into this problem, we elected to focus upon the determinants of aberrant conduction at onset of OT initiated by either atrial or right ventricular extrastimulation (A_2 and V_2 methods, respectively). The effects of these induction techniques, in particular, can be precisely related to the events at onset of induced OT. Moreover, these two stimulation methods initiate OT by entirely different mechanisms: induction of OT by A_2 requires anterograde block in the accessory pathway with effective propagation along the atrioventricular node and His-Purkinje system (3). In contrast, V_2 generally starts OT by inducing bilateral retrograde infra-His block (4) (causing concealed conduction in the His-Purkinje system [5]) with concomitant retrograde atrial activation occurring via the bypass tract.

We conjectured that, in view of these disparate modes of input to the normal pathway during OT induction, refractoriness of the His-Purkinje system should be differentially encountered and manifested with the A_2 and V_2 methods. This hypothesis was evaluated using a beat-by-beat analysis in 20 consecutive patients with accessory pathways and OT induced by the A_2 and/or V_2 techniques. Our findings suggest that a rational physiologically oriented approach to the deliberate induction of fx BBB at OT onset is indeed feasible.

Methods

Electrophysiologic studies were performed in the nonsedated, postabsorptive state after informed consent was obtained and after discontinuation of antiarrhythmic drugs for at least 48 h. Under local anesthesia, three or more multipolar electrode catheters were introduced percutaneously via peripheral veins and, with fluoroscopic guidance, positioned in the high right atrium, coronary sinus, right ventricular apex (or outflow tract), and the region of the His (and right) bundle. The catheters were used to record local bipolar intracardiac electrograms and to perform programmed stimulation. All intracardiac electrograms, surface electrocardiographic leads I, II, and V_1, and time lines were simultaneously displayed on a multichannel oscilloscope and recorded on an FM tape

Address reprint requests to Dr. Lehmann at his current address: Division of Cardiology, Harper Hospital, 3990 John R, Detroit, Michigan 48201.

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1. For the purpose of this article, we will apply the eponym "Wolff-Parkinson-White syndrome" to all patients with accessory atrioventricular pathways, whether or not there is manifest ventricular preexcitation.

2. *Abbreviations used in this paper:* A_2 method, atrial extrastimulus method; AH, atrio-His; BBB, bundle branch block; fx, functional; HV, His-ventricular; LBBB, left bundle branch block; OT, orthodromic tachycardia; RBBB, right bundle branch block; V_2 method, right ventricular extrastimulus method; VA, ventriculoatrial; VH, ventricular-His.

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recorder for subsequent reproduction at a paper speed of 100 mm/s (Electronics for Medicine model VR-16 [Pleasantville, NY]). Intracardiac stimulation was performed with a digital stimulator (Bloom Associates, Reading, PA) capable of delivering rectangular impulses of 5-V amplitude and 2-ms duration.

The stimulation protocol included incremental atrial and ventricular pacing as well as A_2 and V_2 methods (at one or more basic cycle lengths) to assess anterograde and retrograde conduction and refractory periods, and to induce OT as previously described (6). Accessory pathway localization was accomplished using standard criteria (7), including quantitation of the change in ventriculoatrial (VA) interval (measured on the His bundle electrogram) accompanying the occurrence of sustained (≥three beats of) fx BBB during OT (2).

Patient characteristics. Base-line clinical and electrophysiologic data of the 20 patients studied are indicated in Table I. There were 11 males and 9 females ranging in age from 8 to 59 yr. No patients had evidence of structural heart disease. Accessory pathways were left-sided in 14 patients, paraseptal in 5, and right-sided in 1. Manifest preexcitation during sinus rhythm (or atrial pacing) was present in 14 with the remainder demonstrating accessory pathway conduction only in the retrograde direction. The OT cycle lengths ranged from 250 to 410 ms with a mean of 328±48 ms. During OT all patients were capable of conducting with narrow QRS complexes (≤90 ms in duration) associated with His–ventricular (HV) intervals ranging from 35 to 60 ms.

Definitions. OT onset refers to the first induced ventricular complex (regardless of stimulation technique) resulting exclusively from reentrant excitation of the atria and His bundle (anterogradely). It follows from this working definition that when OT is induced by the A_2 method, the ventricular response to the paced A_2 *precedes* the first OT complex.

Complete right (R) and left (L) BBB were defined according to accepted electrocardiograph (ECG) criteria (8).

Incomplete LBBB was considered present when (*a*) QRS duration was prolonged (versus narrow-QRS OT) to a value <120 ms with loss of septal Q wave in lead I, and/or (*b*) the frontal axis was shifted leftward (to −30 or beyond often with a deeper S wave in V_1) or, rarely, rightward (to +90 or beyond but without evidence of terminal delay). This definition was used to include all forms of manifest left bundle conduction delay short of complete LBBB.

Incomplete RBBB was defined by the appearance of terminal rightward conduction delay with QRS prolongation to a value <120 ms.

When OT was induced by the V_2 method, differentiation of anterograde from retrograde origin of the (emergent) His deflection following V_2 was accomplished using previously published criteria (4). It was possible, therefore, to distinguish also between fx complete LBBB (at OT onset) and macro-reentrant V_3 induced by V_2 (9).

VH interval (in cases of OT induced with the V_2 method) was measured from the onset of the initiating V_2 (or V_3) electrogram to the onset of the anterograde His deflection immediately preceding the first beat of OT. This parameter (which equals VA + atrio–His[AH] intervals) was considered an estimate of the time elapsed from concealed retrograde penetration of the His–Purkinje system by the initiating right ventricular impulse to the onset of initial reentrant anterograde His activation.

Statistical analysis. Measured values are expressed as mean±standard deviation. The unpaired *t* test was utilized to compare differences in means. Statistical significance was defined as $P < 0.05$.

Results

For reasons stated above, only findings pertinent to OT initiation by the A_2 or V_2 methods form the basis of this report. Moreover, for each patient, only data obtained at the basic cycle length associated with the widest aberrant complex at OT onset are presented so as to simplify analysis of the results.

Table I. Base-line Clinical and Electrophysiologic Data

Patient no.	Age (*yr*)/sex	Location of AP	Manifest preexcitation	Characteristics of OT						Induction method	
				CL	QRS	Axis	AH	HV	VA	A_2	V_2
				ms	*ms*		*ms*	*ms*	*ms*		
1	30/F	Left FW	+	370	80	N1	210	50	110	+	−
2	17/F	Left FW	−	410	90	N1	235	50	125	+	−
3	32/M	Left FW	+	385	80	N1	210	55	120	+	−
4	59/M	Left FW	+	340	70	N1	135	55	150	+	−
5	8/F	Right FW	+	250	80	N1	85	40	125	+	−
6	42/F	Left FW	−	360	80	N1	200	40	120	+	−
7	16/M	Left FW	−	310	90	N1	145	45	120	+	+
8	43/M	Paraseptal	+	350	70	N1	185	50	115	+	+
9	17/M	Left FW	−	340	90	N1	150	50	140	+	+
10	43/F	Paraseptal	+	290	80	N1	155	35	100	+	+
11	18/M	Paraseptal	−	260	90	N1	105	45	110	+	+
12	30/F	Left FW	+	270	80	N1	110	40	120	+	+
13	21/F	Left FW	+	365	90	N1	205	40	120	+	+
14	40/F	Paraseptal	+	260	80	N1	130	40	90	−	+
15	29/F	Paraseptal	+	290	80	N1	140	50	100	−	+
16	56/M	Left FW	+	390	80	N1	225	45	120	−	+
17	20/M	Left FW	+	380	80	N1	210	50	120	−	+
18	44/M	Left FW	−	320	90	N1	140	50	130	−	+
19	23/M	Left FW	+	300	90	N1	135	40	125	−	+
20	24/M	Left FW	+	325	80	N1	145	60	120	−	+

Abbreviations: AP, accessory pathway; CL, cycle length; VA, ventriculoatrial conduction time (measured on the His bundle electrogram); FW, free wall; N1, normal.

Comparative incidence of fx BBB at OT onset with the A_2 and V_2 methods. As Table I indicates, OT was induced by the A_2 method alone in six patients (nos. 1–6), by both the A_2 and V_2 methods in seven (nos. 7–13) and by the V_2 method alone in seven (nos. 14–20).

Fig. 1 shows the distribution of widest aberrant complexes observed in each patient at OT onset, according to morphologic type and induction method. It is apparent that, with the V_2 method, not only was the incidence of fx BBB in general greater than with the A_2 method (79% vs. 38%) but, more specifically, fx LBBB (complete or incomplete) was also considerably more frequent (79% vs. 15% with A_2). In contrast, fx RBBB was relatively more common at onset of OT initiated by the A_2 than by the V_2 method (31% vs. 7%).

Tables II and III list the actual electrophysiologic characteristics of the widest aberrant complexes (for each individual patient) at onset of OT induced by the A_2 and V_2 methods, respectively. It may be appreciated that both the predominance of fx LBBB at OT onset with the V_2 method and the greater incidence of fx RBBB with the A_2 method also applied to the subgroup of patients (nos. 7–13) in whom OT was inducible by both methods (with identical basic cycle lengths used in six of the seven cases). Complete RBBB and LBBB were both observed at OT onset in patient 6 with the A_2 method and in patient 9 with the V_2 method. Our electrophysiologic findings will now be considered in further detail.

Aberrant conduction at OT onset with the A_2 method. As shown in Table II, five patients (nos. 5–7, 9, and 11) exhibited fx BBB at onset of OT initiated by A_2, associated with an altered axis in four (nos. 6, 7, 9, and 11) and HV interval prolongation in all (range 10–135 ms). By the second beat of OT (not tabulated), aberrant conduction usually resolved (accompanied by shortening of the HV interval) except in two patients (nos. 6 and 11) in whom fx RBBB was sustained and one (no. 7) in whom infra-His block developed.

Table II also lists characteristics of the QRS complex in response to A_2 when the latter induced fx BBB at OT onset. Note

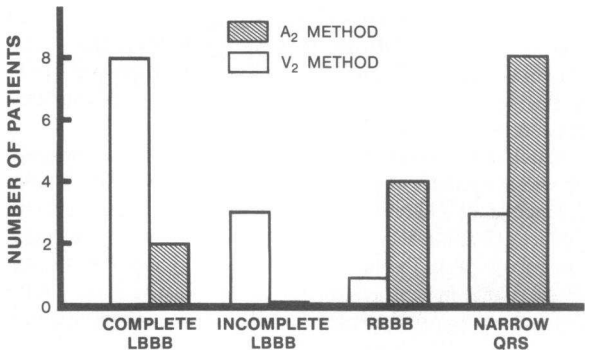

Figure 1. Comparative distribution of QRS morphologies at onset of OT induced by the A_2 and V_2 methods. Only the widest QRS complexes of a given morphology for each patient are tallied. The total number of patients here exceeds 27 (the number of those induced by A_2 plus those induced by V_2) because two patients had both complete RBBB and LBBB.

that in all such cases the same type of fx BBB occurring at OT onset was already present during conduction of A_2. In two patients (nos. 5 and 7) incomplete RBBB during propagation of A_2 progressed to complete RBBB at OT onset. Table II reveals further than the HV interval shortened by 10–60 ms during the first beat of OT (versus a longer value during the preceding complex) in five of six instances of aberrancy, with the remaining case (patient 7) manifesting progressive HV prolongation during fx 3:2 Wenckebach block in the His–Purkinje system (6, 10).

We examined a number of variables to assess their potential contribution to aberrant conduction of the A_2 impulse, because the latter occurrence appeared to be a prerequisite for development of fx BBB during the subsequent beat (i.e., at OT onset). The ratio H_1H_2/H_1H_1 (where H_1H_1 = basic cycle length) was used as a measure of the degree of prematurity of a given H_1H_2 input to the His–Purkinje system. This parameter could be measured within the OT initiation zone in all patients from Table

*Table II. Electrophysiologic Data in Patients with OT Induced by the A_2 Method**

Patient no.	BCL	Widest aberrant complex at OT onset						Preceding complex in response to A_2 when aberrant conduction present at OT onset					
		Morphology	QRS (Δ)	Axis	HV (Δ)	VA (Δ)	HH‡	Morphology	QRS (Δ)	Axis	HV (Δ)	VA (Δ)	HH‡
	ms		*ms*		*ms*	*ms*	*ms*		*ms*	*ms*	*ms*	*ms*	*ms*
1	600	Narrow	80 (0)	N1	50 (0)	110 (0)	—	—	—	—	—	—	—
2	600	Narrow	90 (0)	N1	50 (0)	125 (0)	—	—	—	—	—	—	—
3	700	Narrow	80 (0)	N1	55 (0)	120 (0)	—	—	—	—	—	—	—
4	700	Narrow	70 (0)	N1	55 (0)	150 (0)	—	—	—	—	—	—	—
5	600	CRBBB	130 (+50)	N1	90 (+50)	175 (+50)	330	IRBBB	90 (+10)	N1	100 (+60)	140 (+15)	290
6	600	CRBBB§	120 (+40)	RAD	95 (+55)	130 (+10)	300	CRBBB	120 (+40)	N1	105 (+65)	120 (0)	305
		CLBBB	130 (+50)	LAD	50 (+10)	185 (+65)	310	CLBBB	130 (+50)	LAD	110 (+70)	185 (+65)	380
7	600	CRBBB	130 (+40)	LAD	180 (+135)	160 (+40)	350	IRBBB	105 (+15)	N1	130 (+85)	120 (0)	350
8	600	Narrow	70 (0)	N1	50 (0)	115 (0)	—	—	—	—	—	—	—
9	600	CLBBB	135 (+45)	LAD	70 (+20)	210 (+70)	390	CLBBB	135 (+45)	LAD	110 (+60)	210 (+70)	410
10	600	Narrow	80 (0)	N1	35 (0)	100 (0)	—	—	—	—	—	—	—
11	500	CRBBB§	120 (+30)	RAD	55 (+10)	110 (0)	280	CRBBB	120 (+30)	N1	95 (+50)	110 (0)	290
12	450	Narrow	80 (0)	N1	40 (0)	120 (0)	—	—	—	—	—	—	—
13	600	Narrow	90 (0)	N1	40 (0)	120 (0)	—	—	—	—	—	—	—

Abbreviations: BCL, basic cycle length; OT, orthodromic tachycardia; CRBBB, complete right bundle branch block; IRBBB, incomplete right bundle branch block; CLBBB, complete left bundle branch block; ILBBB, incomplete left bundle branch block; LAD, left axis deviation; RAD, right axis deviation. * Numbers in parentheses indicate differences from values of corresponding parameters in Table I. ‡ Refers to HH interval immediately preceding the subsequent beat; tabulated only for complexes showing aberrancy. § Sustained aberrant conduction.

*Table III. Electrophysiologic Data in Patients with OT Induced by the V_2 Method**

Patient no.	BCL	Widest aberrant complex at onset of OT initiated by V_2						Widest aberrant complex at onset of OT initiated by V_3					
		Morphology	QRS (Δ)	Axis	HV (Δ)	VA (Δ)	HH‡	Morphology	QRS (Δ)	Axis	HV (Δ)	VA (Δ)	HH‡
	ms		*ms*		*ms*	*ms*	*ms*		*ms*		*ms*	*ms*	*ms*
7	600	CLBBB	130 (+40)	LAD	95 (+50)	170 (+50)	370	—	—	—	—	—	—
8	600	ILBBB	100 (+30)	LAD	60 (+10)	115 (0)	340	—	—	—	—	—	—
9	600	CLBBB	135 (+45)	LAD	100 (+50)	200 (+60)	410	—	—	—	—	—	—
		CRBBB	120 (+30)	RAD	50 (0)	140 (0)	370	—	—	—	—	—	—
10	500	—	—	—	—	—	—	CLBBB§	130 (+50)	N1	45 (+10)	100 (0)	310
11	500	—	—	—	—	—	—	CLBBB§	150 (+60)	N1 (L)‖	60 (+15)	135 (+25)	275
12	450	—	—	—	—	—	—	Narrow	80 (0)	N1	40 (0)	120 (0)	290
13	600	—	—	—	—	—	—	Narrow	90 (0)	N1	40 (0)	120 (0)	370
14	600	—	—	—	—	—	—	CLBBB§	130 (+50)	LAD	70 (+30)	90 (0)	260
15	600	CLBBB§	130 (+50)	LAD	100 (+50)	100 (0)	310	—	—	—	—	—	—
16	700	Narrow	80 (0)	N1	45 (0)	120 (0)	380	—	—	—	—	—	—
17	700	ILBBB	90 (+10)	LAD	65 (+15)	150 (+30)	430	—	—	—	—	—	—
18	700	ILBBB	100 (+10)	LAD	70 (+20)	140 (+10)	360	—	—	—	—	—	—
19	600	CLBBB	150 (+60)	LAD	50 (+10)	180 (+55)	345	Narrow	90 (0)	N1	40 (0)	125 (0)	290
20	700	CLBBB	140 (+60)	LAD	80 (+20)	160 (+40)	360	Narrow	80 (0)	N1	60 (0)	120 (0)	330

Abbreviations are the same as in Table II. * Numbers in parenthesis indicate differences from values of corresponding parameters in Table I. ‡ Refers to HH interval immediately preceding the subsequent beat. § Sustained aberrant conduction. ‖ Normal axis, but more leftward than during narrow QRS OT.

II except for two (nos. 3 and 4) in whom the H_1 deflection was obscured by a delta wave. The mean maximum H_1H_2/H_1H_1 value in patients who developed fx BBB during propagation of the A_2 impulse was smaller than the mean minimum attainable value measured in those without aberrant condition (0.59±0.05, range 0.52–0.64, vs. 0.69±0.07, range 0.59–0.78, respectively; $P < 0.02$). A similar relationship obtained when absolute H_1H_2 intervals were compared (i.e., mean maximum H_1H_2 in patients with aberrancy was 341±37 ms vs. mean minimum value of 401±63 ms in those without aberrancy, $P < 0.05$).

Of note, manifest preexcitation was more common (88%) in the group that did not develop fx BBB than in those patients who demonstrated aberrant conduction (only 20%). Moreover, in all cases of manifest preexcitation the minimum H_1H_2 value associated with induction of OT was attained at the longest A_1A_2 coupling interval within the OT initiation zone. This interval, of course, coincided with the anterograde effective refractory period of the accessory pathway, which happened to be relatively short, ranging from 230 to 310 ms at the basic cycle length utilized. Finally, it should be mentioned that mean OT cycle length and mean basic cycle length (during the A_2 method) were not significantly different in the group exhibiting aberrant conduction compared to those patients who did not.

Aberrant conduction at OT onset with the V_2 method. As Table III indicates, in patients with OT induced by the V_2 method, the initiating impulse consisted of V_2 alone in seven patients (nos. 7–9 and 15–18), macro-reentrant V_3 alone in five (nos. 10–14), and either V_2 or V_3 (at different coupling intervals) in two (nos. 19 and 20). The site of retrograde block of the initiating impulse was determined using previously published criteria (4), and was located below the His bundle in all cases.

Aberrant conduction at OT onset was observed in 11 patients (fx LBBB in patients 7–11, 14, 15, and 17–20 and fx RBBB in patient 9). This was usually associated with axis deviation (leftward in all with fx LBBB, except for patient 10, and rightward in the one case of fx RBBB—patient 9). The HV interval pro-

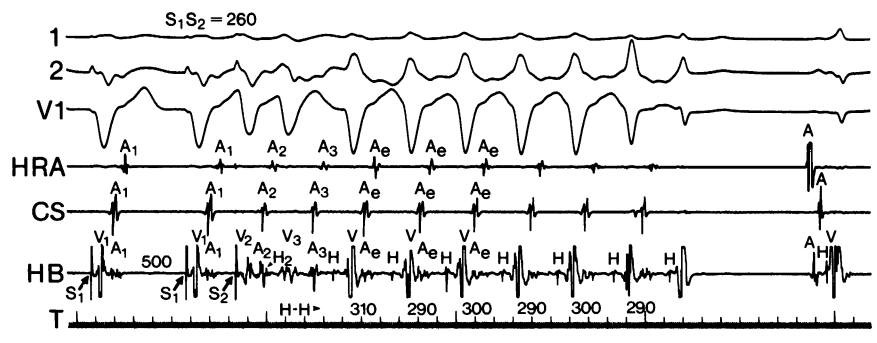

Figure 2. Induction by the V_2 method of fx sustained LBBB at OT onset in a patient (no. 10) with a paraseptal accessory pathway. Tracings from top to bottom are ECG leads 1, 2, V_1, high right atrium, coronary sinus, and His bundle electrograms, followed by time line. The basic cycle length (S_1S_1 or V_1V_1) is 500 ms. At an S_1S_2 coupling interval of 260 ms, a macro-reentrant beat (V_3) is induced (9, 11) which in turn retrogradely blocks bilaterally in the His–Purkinje system, permitting initiation of OT (4). Fx complete LBBB, which is observed at OT onset, sustains for another four beats before spontaneously resolving over the subsequent two beats, despite minimal changes in HH cycle length. When the QRS fully normalizes, there is associated VV shortening (because of less HV delay) and the reentrant wavefront blocks retrogradely in the accessory pathway, thereby terminating the tachycardia. It should be mentioned that the VA_e interval during sustained LBBB (100 ms, unlabeled) is identical to that measured (elsewhere) during narrow QRS OT. HRA, high right atrium; CS, coronary sinus; HB, His bundle; T, time line; A, atrial electrogram; A_e, atrial echo; V, ventricular electrogram; S, stimulus artifact.

longed by 25±16 ms (range 10–50 ms) in all cases of fx LBBB, but remained unchanged in the single instance of fx RBBB. Sustained fx LBBB beginning at OT onset was observed in four patients (nos. 10, 11, 14, and 15). An example from patient 10, in whom sustained LBBB resolved spontaneously after several beats, is shown in Fig. 2.

Retrograde concealed conduction in the His–Purkinje system at onset of OT induced by the V_2 method. In patients with OT induced by the V_2 method, we sought to determine whether extent of His–Purkinje system recovery, as a function of elapsed time after concealed retrograde impulse penetration, could play a role in the genesis of fx BBB at OT onset. The possible existence of such a relationship was investigated by plotting the ventricular–His (VH) intervals corresponding to the widest initial OT complexes for each patient in whom OT was induced by the V_2 method (excluding the single case of fx RBBB, discussed separately below).

The resulting graph is shown in Fig. 3. Note that the VH interval for narrow initial QRS complexes (351±42 ms) was significantly greater ($P < 0.01$) than the value of that parameter determined when either fx incomplete or complete LBBB was manifest at OT onset (262±18 and 238±43 ms, respectively). It follows from Table III that the HV interval at OT onset varied reciprocally with VH because the HV interval increased from 45±9 to 65±5 to 75±22 ms for narrow QRS, incomplete, and complete LBBB, respectively.

The relationship between VH interval and occurrence of fx LBBB was also examined within OT initiation zones of eight individual patients in whom OT was induced at more than one S_1S_2 coupling interval. In these cases the corresponding "VH zones" spanned a width ranging from 10 to 40 ms. In four of five patients manifesting complete LBBB at OT onset, this widest aberrantly conducted complex occurred at the lower limit of the VH zone, whereas fx incomplete LBBB or a narrow QRS complex was observed at the longest VH interval. An example of this relationship is shown in Fig. 4, taken from patient 9. Note the progression at OT onset in *A–C* from narrow QRS to incomplete and then complete fx LBBB (with associated progressive HV prolongation) as the VH interval decreases from longest to shortest value.

The single case of fx RBBB, which we observed (in patient 9) at the onset of OT initiated by V_2, deserves special mention. As shown in Fig. 5 *A*, the His-right bundle temporal relationship during fx RBBB is identical to that existing during OT. This finding confirms that the aberrantly appearing complex is truly of anterograde origin, rather than representing atypical bundle branch reentry (9, 11, 13), as indeed occurred in this patient when the S_1S_2 coupling interval was decreased by 10 ms (Fig. 5 *B*). The relationship between RBBB at OT onset and retrograde concealed conduction in the His–Purkinje system will be considered below in the Discussion.

Effect of aberrant conduction at OT onset upon the VA interval. In this study we were able to assess specifically the effect of aberrant conduction during a single beat (i.e., at OT onset) upon the VA interval. Complete BBB ipsilateral to a free wall accessory pathway was observed at OT onset in six patients (nos. 5–7, 9, 19, and 20). In these cases the associated (initial) VA interval was 55±11 ms (range 40–70 ms) greater than the corresponding value during narrow QRS OT. Such prolonged VA intervals at OT onset fell within 10 ms (mean −3±4 ms) of the values measured in five of the six patients during sustained ipsilateral fx BBB.

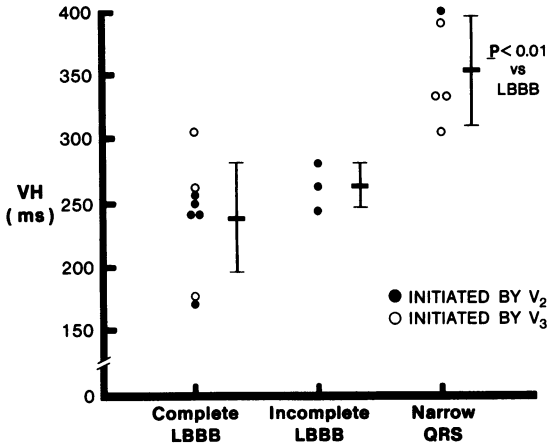

Figure 3. Plot of VH (i.e., V_2H or V_3H) intervals corresponding to morphology of widest QRS complexes at onset of OT initiated by the V_2 method. The total number of points plotted exceeds 14 (the number of patients with OT induced by the V_2 method) because in two cases OT could be initiated by either V_2 or V_3. The *P* value shown corresponds to the difference in VH for narrow QRS versus either complete or incomplete LBBB.

We considered the possibility that initial VA prolongation might have resulted in part from an incompletely recovered bypass tract rather than simply from delayed retrograde input to that tissue (owing to ipsilateral BBB). Such an interpretation appears unlikely inasmuch as the retrograde coupling interval (V_2V or V_3V) associated with fx complete BBB at OT onset greatly exceeded the retrograde effective refractory period of the accessory pathway, namely, by 65±29 ms (range 30–110 ms). This point may be illustrated (in patient 9) by comparing the V_2V interval in Fig. 3 *C* (corresponding to complete LBBB) with the V_1V_2 interval in Fig. 4 *B* (where the retrograde effective refractory period of the bypass tract is encountered).

At OT onset, fx complete BBB contralateral to a free wall bypass tract was observed in three patients (nos. 6, 7, and 9). The associated VA interval was prolonged by no more than 10 ms, except in one (patient 7) who exhibited fx bifascicular block (at onset of OT induced by A_2). Of the patients with a paraseptal pathway, both fx complete RBBB and LBBB occurred at OT onset in one (no. 11). In that case, the VA interval increased solely during complete LBBB (by 25 ms, an increment identical to that observed during sustained LBBB).

Discussion

Our results suggest that in patients with Wolff–Parkinson–White syndrome, the A_2 and V_2 methods each tend to promote specific types of aberrant conduction at onset of induced OT. In the series of patients that we studied, fx LBBB was readily induced by the V_2 method, whereas fx RBBB was more frequent with the A_2 method. Moreover, the overall incidence of aberrant conduction was greater with the V_2 method. Because random variation in patient selection and use of different basic cycle lengths can alter the actual incidences of particular types of aberrant conduction from one clinical series to another, we wish to place greater emphasis on the roles played by differences in electrophysiologic sequelae of the A_2 and V_2 methods.

Relation of the OT initiating mechanism with the A_2 method

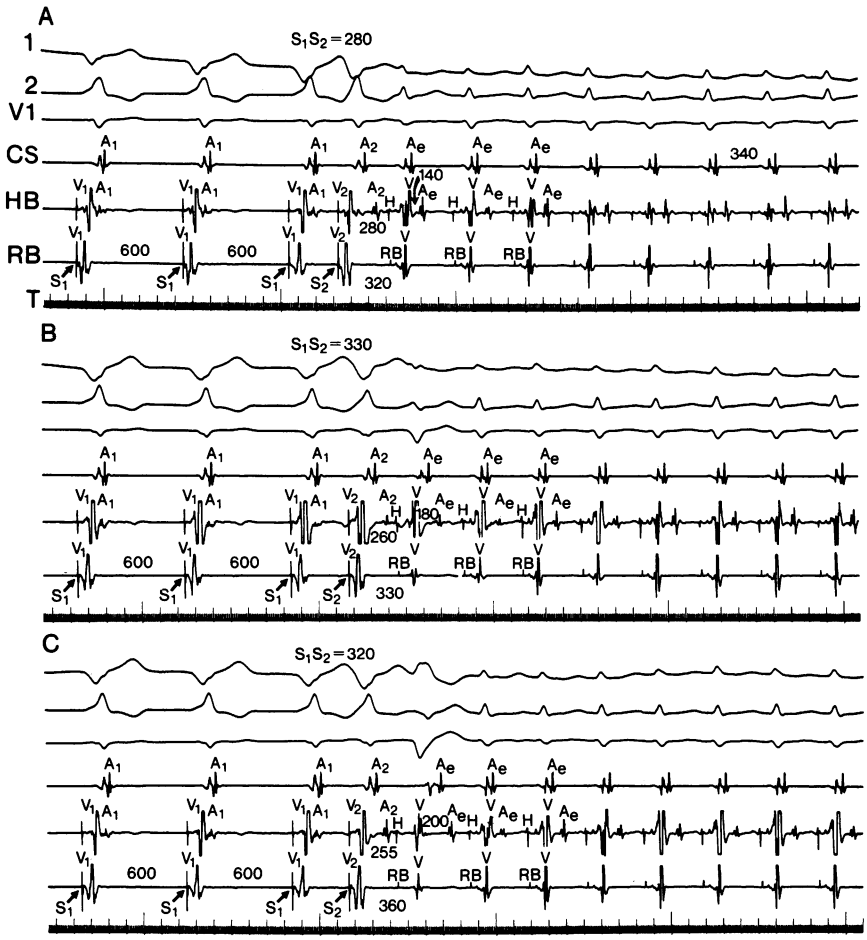

Figure 4. Induction of fx LBBB at onset of OT initiated by the V_2 method in a patient (no. 9) with a concealed left free wall accessory pathway. Tracings from top to bottom are ECG leads 1, 2, and V_1 followed by coronary sinus, His bundle and right bundle electrograms, and time line. In all three panels a ventricular extrastimulus (S_2 or V_2) initiates OT after the last beat of the basic ventricular drive which has a cycle length (S_1S_1 or V_1V_1) of 600 ms. The panels are arranged in order of increasing V_2H intervals. (*A*) At an S_1S_2 (or V_1V_2) coupling interval of 280 ms, the V_2 impulse initiates an OT with narrow initial QRS complex. This beat is associated with a V_2H interval of 280 ms and a VA_e interval of 140 ms. The HV and RB–V intervals at OT onset (45 and 25 ms, respectively, unlabeled) are identical to the values measured during sustained narrow QRS OT at a cycle length of 340 ms. Subtle QRS alternans, which may occur during OT in patients with Wolff–Parkinson–White syndrome (12), is also noted here. (*B*) At a longer S_1S_2 coupling interval of 330 ms OT is again initiated, but now fx incomplete LBBB is observed at OT onset. This occurs in association with a shorter V_2H interval of 260 ms and an increase in both the HV and RB–V intervals to 80 and 60 ms, respectively (both unlabeled). The initial VA_e interval also prolongs to 180 ms. (*C*) At a slightly shorter S_1S_2 coupling interval of 320 ms, fx complete LBBB is now apparent at OT onset whereas the V_2H interval has shortened further (versus *B*) to 255 ms. Concomitantly, the VA_e interval prolongs to 200 ms, a value only 10 ms less than that measured (elsewhere) during sustained fx LBBB. The initial HV and RB–V intervals have also increased to 100 ms and 80 ms, respectively (unlabeled). Preservation of the His–RB relationship that exists during narrow QRS OT ensures that the wide QRS complex at OT onset is of anterograde origin (and argues against the occurrence of a macro-reentrant V_3) (11). Moreover, the fact that in *B* and *C* both the HV and RB–V prolong by the same amount implies that, at least in this case, the site of coexistent conduction delay along the H–RB–V route is located distal to the RB recording site, presumably where retrograde concealed penetration of the RB by the V_2 impulse had occurred (13). This state of affairs is different from that which obtains during fx BBB induced by atrial premature stimulation where the site of associated right-sided conduction delay is usually located proximal to the RB recording site (14). It should also be evident from *A–C* that at the same time that greater degrees of fx LBBB are induced at OT onset, the V_2V interval progressively increases from 320 to 360 ms, making it most unlikely that the concomitant progressive VA_e prolongation can be attributed to greater retrograde input stress on the accessory pathway. Finally, it may be appreciated in *C* that fx complete LBBB at OT onset resolves by the second beat of OT in association with marked HH prolongation (to 420 ms, unlabeled) despite AH shortening relative to narrow QRS OT. RB, right bundle; other abbreviations as in Fig. 2.

to aberrant conduction at OT onset. For OT to be initiated by an atrial extrastimulus, a critical amount of anterograde delay in transmission of the A_2 impulse is required to permit recovery of the bypass tract (from the effects of concealed anterograde penetration) so that reentrant excitation of the atria can occur (3). To this end, slowing of impulse propagation solely in the atrioventricular node, at a sufficiently short A_1A_2 coupling interval, is often adequate (3, 6). Less commonly additional delays in anterograde His–Purkinje system conduction and/or disruption of the normal ventricular activation sequence are needed, as may be afforded by the occurrence of fx BBB, especially the ipsilateral type (6, 15, 16).

Whether or not fx BBB is a necessary condition for OT initiation by the A_2 method in a given patient, the occurrence of aberrant conduction will have similar determinants in all cases. Most important of these is the ability of the atrioventricular node, over a range of A_1A_2 coupling intervals, to deliver sufficiently short H_1H_2 inputs that will encroach upon the relative refractory period of the His–Purkinje system (17). In our cases of OT induced by the A_2 method, shorter H_1H_2 intervals (both absolute and relative to basic cycle length) were indeed found in patients who exhibited fx BBB during propagation of A_2 compared to those who did not.

Notably, we also found that a relatively short anterograde effective refractory period of the accessory pathway appeared to be an indirect (but potentially important) factor limiting the ocurrence of aberrant conduction during transmission of A_2 impulses that initiated OT. Such an impression is based upon observations in our patients with manifest preexcitation, all of whom had short anterograde accessory pathway refractory periods and most of whom did not exhibit fx BBB after A_2. In these cases, the minimum H_1H_2 interval associated with OT induction was found at the longest A_1A_2 coupling interval within the OT initiation zone. This finding will require confirmation

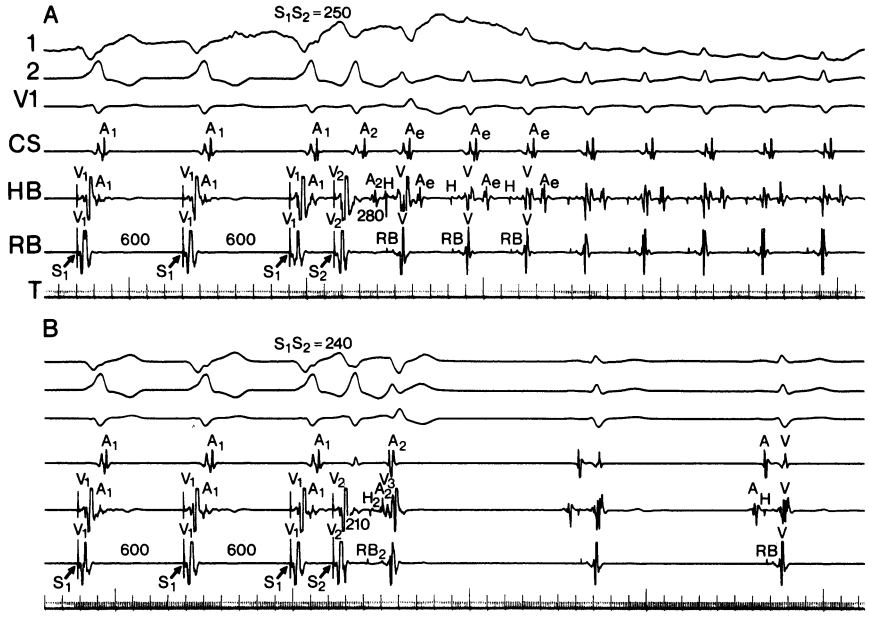

Figure 5. Induction of fx RBBB at onset of OT initiated by the V_2 method (same patient as in Fig. 4). Format, abbreviations, and basic cycle length are identical to those of Fig. 4. (*A*) At an S_1S_2 coupling interval of 250 ms (30 ms shorter than in Fig. 4 *A*) complete RBBB at onset of OT occurs with a V_2H interval of 280 ms and unaltered HV and RB–V intervals compared to narrow QRS OT (45 and 25 ms, respectively, unlabeled). The preserved His–RB relationship is consistent with a supraventricular site of impulse origin (and block in the RB below the RB recording site). (*B*) When S_1S_2 is shortened to 240 ms, the retrograde effective refractory period of the accessory pathway is encountered and macroreentry with a RBBB morphology ensues (9, 11). The latter phenomenon is associated with a resumption of retrograde conduction over the normal pathway despite bilateral His–Purkinje system block in *A* (gap phenomenon) (13). That His activation occurs retrogradely here (versus anterogradely in *A*) follows from postgap shortening of the V_2H interval (4) in *B* (to 210 ms) and from the alteration in the His-RB relationship (11, 13) so that the RB deflection now precedes the H by 20 ms. This clearly differs from the situation during narrow QRS OT (in *A*) or during sinus beats (in *B*). Finally, note that in contrast to the case of fx complete LBBB at OT onset (Fig. 4 *C*), there is no VA prolongation (relative to narrow QRS OT) associated with complete RBBB at OT onset (VA_e = 140 ms, unlabeled in *B*) in this patient with a left free wall accessory pathway.

in a larger series. Nonetheless, it implies that the atrioventricular nodal functional refractory period (and, hence, shorter H_1H_2 values) would have been encountered only at A_1A_2 coupling intervals exceeding the anterograde effective refractory period of the bypass tract (i.e., beyond the OT initiation zone).

When fx BBB did occur during propagation of A_2, we observed that the same type of aberrant conduction was sustained through the next beat (first OT complex), presumably through a linking-by-interference mechanism (18) involving transseptal retrograde invasion from the contralateral bundle (19). Because the A_2 method typically induces fx RBBB more commonly than fx LBBB (17), it is not surprising that such was also the case in our series during conduction of A_2 and, hence, at OT onset.

Relation of the OT initiating mechanism with the V_2 method to aberrant conduction at OT onset. The mechanism of OT initiation with the V_2 method is fundamentally different from that of its atrial counterpart. As depicted schematically in Fig. 6, during the basic drive (V_1, *upper left corner*) both the normal and the accessory pathways are activated retrogradely. We (4) have shown previously that in order for OT to be initiated, in most instances bilateral retrograde infra-His (rather than intranodal) block of the V_2 impulse (Fig. 6, *upper middle panel*) or the macro-reentrant V_3 impulse (Fig. 6, *lower left and middle panels*) must occur, with retrograde atrial activation proceeding exclusively via the accessory pathway. In either case, because ventricular activation during V_2 and V_3 (of the typical variety [9, 11]) originates from a right ventricular location, concealed retrograde penetration of the left bundle will be delayed relative to occurrence of similar concealment in the right bundle. Consequently, recovery of excitability in the left bundle will be completed at a later time than in the case of its right-sided counterpart.

In virtually all patients, therefore, a bias exists that clearly favors the development of fx LBBB when the initial reentrant impulse attempts to traverse the His–Purkinje system. More precisely, anterograde engagement of the left bundle will occur during its recovery process at a point determined by the arrival time of the anterograde impulse to the His–Purkinje system relative to the preceding retrograde penetration (i.e., the VH interval). This arrival time, in turn, is largely a function of anterograde atrioventricular nodal conduction (since VH = VA + AH, and VA is virtually fixed in any given patient).

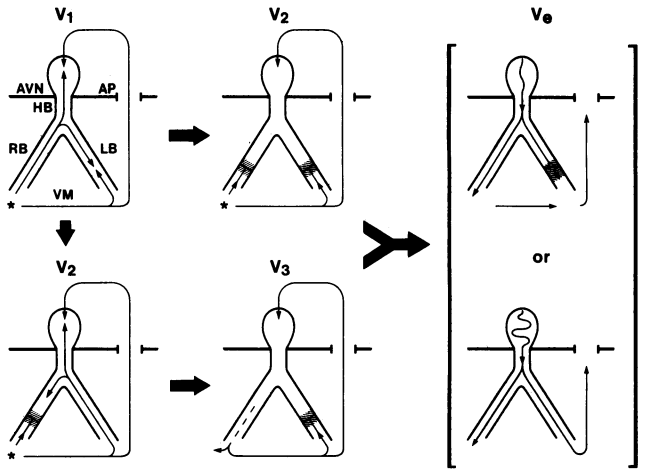

Figure 6. Schematic representation of mechanism responsible for facilitation of fx LBBB at onset of OT induced by the V_2 method. Paced stimuli (*asterisks*) that give rise to V_1 and V_2 are delivered to a right ventricular site. The location of the accessory pathway is arbitrary. Moreover, depicted sites of conduction delay or block in the His–Purkinje system (*shaded regions*) are purely schematic in nature and not intended to imply precise anatomic locations. See text for additional explanation. AVN, atrioventricular node; AP, accessory pathway; HB, His bundle; RB, right bundle; LB, left bundle; VM, ventricular myocardium; Ve, first ventricular echo of OT.

We were able to demonstrate that indeed the magnitude of the VH interval could be correlated with the occurrence of fx LBBB at onset of OT induced by the V_2 method. Over the range of basic cycle lengths utilized, fx LBBB was observed only when VH approached the vicinity of 300 ms or less, with complete (rather than incomplete) LBBB observed at the shortest VH intervals (Fig. 3). The reciprocal VH–HV relationship that we documented implies encroachment upon the relative refractory period of the right bundle, as well as left bundle, when the VH interval is short (Fig. 4). Ultimately, if VH were sufficiently short, bilateral infra-His block of the anterograde impulse would occur (5, 18).

Although not immediately apparent, it is possible to account also for the much less likely occurrence of fx RBBB at onset of OT induced with V_2 (Fig. 5 *A*) by invoking a mechanism analogous to that which appears responsible for the genesis of fx LBBB, as follows: at short coupling intervals during the V_2 technique, retrograde His activation usually occurs via the transseptal-left bundle route (with retrograde right bundle conduction either blocked or markedly slowed) (13). In a small subset of patients, however, the opposite situation may obtain at certain short coupling intervals so that retrograde block (or slow conduction) occurs in the left bundle while retrograde His activation proceeds via the right bundle route (13).

Under appropriate conditions this scenario could lead to macro-reentry with a RBBB morphology (11), as indeed was the case in Fig. 5 *B*. By extrapolation, in some instances of bilateral infra-His block of the V_2 impulse, retrograde block (and recovery) in the left bundle might actually precede occurrence of retrograde block (and recovery) in the right bundle. Hence, if a patient with a retrogradely functioning accessory pathway developed this type of bilateral retrograde His–Purkinje system block during the V_2 method, then fx R (not L) BBB would be facilitated at OT onset, as in Fig. 5 *A*.

Clinical implications. Our findings can be applied to the development of a rational approach to the deliberate induction of specific types of fx BBB using programmed stimulation in patients with Wolff–Parkinson–White Syndrome. Because aberrant conduction at onset of OT induced by the A_2 method appears largely determined by the occurrence (and type) of fx BBB during anterograde propagation of A_2, techniques that increase the "yield" of fx (usualy R) BBB during conduction of A_2 should also facilitate occurrence of (R)BBB at OT onset. Such techniques might include not only use of both long and short basic cycle lengths (the latter being perhaps more likely to promote fx LBBB [20]) but also maneuvers that prolong His–Purkinje system refractoriness, such as abrupt short-to-long cycle length changes (21) and bigeminal rhythms (22).

Because occurrence of bilateral retrograde His–Purkinje system block during the V_2 method permits initiation of OT at the same time that it facilitates fx LBBB at OT onset, all attempts to induce OT by this technique should be made. As in the case of the A_2 method, ventricular drives incorporating short-to-long cycle-length changes (23) or alternating long and short cycle lengths (24) might prove helpful. Bilateral fx retrograde His–Purkinje system block can also often be induced by a ventricular train consisting of two short cycles of equal duration (25). This technique can result in OT initiation (26) and facilitates fx LBBB at OT onset (18) by a mechanism similar to that which is operative with the V_2 method. If OT with only a narrow initial QRS is induced by the aforementioned ventricular stimulation techniques, then the VH interval is probably not sufficiently short. This situation might be remedied by administration of an agent such as atropine, which enhances atrioventricular nodal conduction (27).

Our findings also provide a mechanistic rationale for proposing a novel approach to the deliberate induction of fx RBBB at OT onset. By performing ventricular extrastimulation from the left ventricle, a mirror image of the events depicted schematically in Fig. 6 might very well obtain, so that at OT onset a bias would then exist favoring the occurrence of fx RBBB.

Finally, our data suggest that quantitation of changes in the VA interval associated with fx BBB at OT onset alone may aid in accessory pathway localization. This underscores the importance of being able deliberately to induce even a single aberrantly conducted beat at the onset of OT.

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