Electrophysiologic Characteristics of Sudden QRS Axis Deviation during Orthodromic Tachycardia

Role of Functional Fascicular Block in Localization of Accessory Pathway

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Abstract

We analyzed the effect of functional fascicular block (FFB) on ventriculoatrial conduction time (VACT) during orthodromic tachycardia (OT) in 32 patients with single accessory pathway (AP) of the Kent bundle type. The location of AP was left free wall (LFW-AP) in 21 patients, left posteroseptal in 6, right free wall in 2, and right anteroseptal in 3. FFB either alone or in combination with functional left or right bundle branch block (LBBB or RBBB) occurred predominantly at the onset of OT and was initiated with ventricular extrastimulus technique more often than with atrial extrastimulation. In patients with LFW-AP, isolated functional left anterior fascicular block (LAFB) produced significant prolongation in VACT (15–35 ms). A similar magnitude of VACT increase (20–35 ms) was also observed when LAFB was associated with RBBB. Although 25–45-ms prolongation in VACT occurred with functional LBBB and normal axis, an additional 20–55-ms VACT increase was seen when LAFB accompanied LBBB. Functional LAFB, alone or in combination with bundle branch block, however, did not prolong VACT in patients with other AP locations. Furthermore, left posterior fascicular block did not produce prolongation of VACT in any of the cases.

It is concluded that in patients with the Wolff-Parkinson-White syndrome, evaluation of VACT during functional LAFB provides important information regarding AP localization and a clear separation of LFW-AP from all other locations.

Introduction

Aberrant intraventricular conduction during supraventricular tachycardia is a relatively common electrophysiologic phenomenon which in the form of bundle branch block has been extensively investigated. The effect of functional bundle branch block on cycle length of orthodromic tachycardia (OT)[1] and ventriculoatrial conduction time (VACT) has been

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1. *Abbreviations used in this paper:* AP, accessory pathway; FFB, functional fascicular block; LAFB, left anterior fascicular block;

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of special interest because of its usefulness in the localization of accessory pathways (1–5). Although a sudden shift of QRS axis due to functional fascicular block (FFB) during OT is not uncommon, the electrophysiologic characteristics of such a phenomenon and its role in accessory pathway localization remain unexplored.

In order to gain insight into this problem, we performed a beat-by-beat analysis of electrophysiologic recordings during OT in patients who exhibited FFB with or without concomitant functional bundle branch block. It is the purpose of this report to provide new information concerning the occurrence of FFB and its impact on localization of accessory pathways.

Methods

Electrophysiologic study. All antiarrhythmic medications were discontinued for at least five half-lives before studies. Electrophysiologic evaluations were carried out in a nonsedated, postabsorptive state after obtaining informed consent. After local anesthesia, three or more multipolar electrode catheters were introduced percutaneously via antecubital and femoral veins and positioned under fluoroscopic guidance in the high right atrium, coronary sinus, in the region(s) of the right bundle and/or the His bundle, and right ventricular apex. Surface ECG leads (I, II, and V_1), intracardiac electrograms, and time lines were simultaneously displayed on a multichannel oscilloscope and recorded on a magnetic tape for subsequent reproduction. Electrical stimulation was performed with a programmable digital stimulator (Bloom Associates, Ltd.). The induction of OT was repeatedly attempted by atrial and ventricular stimulation (incremental pacing and programmed premature extrastimulation). For each patient, VACT was determined during the following types of OT when they occurred: (*a*) narrow QRS complex with and without axis shift; (*b*) functional left bundle branch block (LBBB) with and without axis shift; (*c*) functional right bundle branch block (RBBB) with and without axis shift. It should be pointed out that the VACT measurements used in this study were obtained at constant OT cycle length. However, in patients where tachycardia exhibited cycle length changes, VACT comparison between different types of tachycardia was performed at comparable OT cycle lengths ($\Delta < 10$ ms).

Patient population. Of 58 consecutive patients with single-accessory pathway (AP) of Kent bundle type, 32 cases who demonstrated at least one type of OT (i.e., narrow QRS, functional LBBB, or functional RBBB with and without shift of frontal plane QRS axis) form the basis of this report. Essential clinical and electrophysiologic data on these patients are depicted in Table I. There were 19 males and 13 females with age of 9–64 yr (mean 31 yr). By using previously reported criteria (6), the accessory pathways were localized to the left free wall (LFW) in 21 patients, left posteroseptal (LPS) in 6, right free wall (RFW) in 2, and right anteroseptal (RAS) in 3. In 16 patients (Nos. 1, 6, 7, 10, 11,

LBBB, left bundle branch block; LFW, left free wall; LPFB, left posterior fascicular block; LPS, left posteroseptal; OT, orthodromic tachycardia; RAS, right anteroseptal; RBBB, right bundle branch block; RFW, right free wall; VACT, ventriculoatrial conduction time.

Table I. Baseline Clinical and Electrophysiologic Data

Patient no.	Age	Sex	MP	AP location	OT-CL NQRS	Ventriculoatrial conduction time							
						NQRS NA	NQRS LAFB	NQRS LPFB	LBBB NA	LBBB LAFB	RBBB NA	RBBB LAFB	RBBB LPFB
	yr					*ms*							
1	14	M	+	LFW	310	120	145	120	160	200	120	—	120
2	16	M	−	LFW	360	135	155	135	180	200	135	—	135
3	37	M	−	LFW	310	140	—	—	185	215	140	165	—
4	51	F	+	LFW	320	115	—	—	—	—	115	135	—
5	47	F	+	LFW	300	120	140	— .	—	185	—	—	—
6	35	M	+	LFW	300	115	—	—	145	170	—	—	—
7	27	M	+	LFW	315	110	130	—	—	—	110	—	110
8	19	F	+	LFW	310	140	155	—	—	—	—	—	140
9	44	M	−	LFW	320	120	145	—	—	170	120	145	—
10	21	F	+	LFW	350	105	—	—	135	165	—	—	105
11	34	F	+	LFW	305	125	—	—	155	180	125	175	125
12	55	F	−	LFW	240	120	—	120	165	200	120	—	120
13	23	M	+	LFW	325	115	140	—	140	165	—	—	115
14	22	F	+	LFW	320	130	—	130	170	210	130	—	130
15	26	M	+	LFW	270	120	—	—	150	175	120	—	120
16	26	M	−	LFW	300	115	150	—	—	190	115	150	115
17	20	M	−	LFW	275	140	160	—	180	235	—	—	140
18	21	M	−	LFW	280	125	155	—	—	180	125	155	—
19	64	M	+	LFW	—	—	—	—	—	—	135	170	—
20	40	F	−	LFW	285	105	130	—	150	170	105	—	—
21	33	M	+	LFW	260	105	125	—	—	180	—	—	105
22	43	F	+	LPS	350	105	105	105	—	—	—	—	—
23	26	M	+	LPS	370	110	110	—	—	135	110	—	110
24	17	M	+	LPS	300	115	115	115	140	140	115	115	115
25	28	M	+	LPS	330	115	115	—	—	135	115	—	—
26	57	F	+	LPS	340	90	—	90	105	105	90	90	—
27	18	M	−	LPS	280	110	110	110	130	130	—	—	110
28	11	F	+	RFW	270	130	130	—	130	130	175	—	175
29	23	M	+	RFW	305	135	135	—	135	—	170	170	—
30	9	F	+	RAS	280	95	95	—	95	—	115	115	—
31	35	M	+	RAS	300	110	110	110	110	110	130	130	—
32	22	F	+	RAS	295	105	105	—	—	—	125	125	—

Abbreviations: AP, accessory pathway; CL, cycle length; LAFB, left anterior fascicular block; LBBB, left bundle branch block; LFW, left free wall; LPFB, left posterior fascicular block; LPS, left posteroseptal; MP, manifest preexcitation; NA, normal axis; NQRS, narrow QRS complex; OT, orthodromic tachycardia; RAS, right anteroseptal; RBBB, right bundle branch block; RFW, right free wall.

14, 15, 16, 20, 21, 23, 24, 27, 29, 31, 32) who underwent epicardial cryosurgery of their accessory pathways, the location of these pathways during epicardial mapping was the same as had been determined preoperatively. Manifest preexcitation during sinus rhythm was present in 23 patients, and the remaining nine patients had unidirectional accessory pathways capable of conducting only in the retrograde direction. Absence of structural heart disease was confirmed by clinical examination and noninvasive cardiac investigations in all patients.

Definition of terms. A complete list of definitions that are used in this type of investigation has been previously published (7). Only those pertinent to the present study are defined below. VACT during OT was measured from the earliest onset of ventricular activation (on either intracardiac recordings or surface ECG) to the onset of atrial activation on the His bundle electrogram recording. Definition of functional LBBB and RBBB has been previously published (8). Functional left anterior fascicular block (LAFB) was defined as sudden shift of the QRS axis toward the left (frontal plane QRS axis of $-30°$ or less, i.e., R < S in ECG lead II). Functional left posterior fascicular block (LPFB)

was defined as sudden shift of the QRS axis toward the right (frontal plane QRS axis of $+90°$ or greater, i.e., R < S in ECG lead I). Isolated FFB was diagnosed when the QRS duration of the complex with axis shift was equal to or less than 110 ms.

Statistical analysis. All data are expressed as mean±SD. Statistical comparison was done using Student's t test for paired values. Unpaired data were compared by analysis of variance. A probability (P) value < 0.05 was considered significant.

Results

In all patients, initiation and termination of OT was repeatedly attempted. FFB most commonly occurred at the onset or within the first several cycles after OT initiation. The electrophysiologic characteristics of functional LAFB and LPFB, for each type of OT (narrow QRS or functional bundle branch block), will be presented separately.
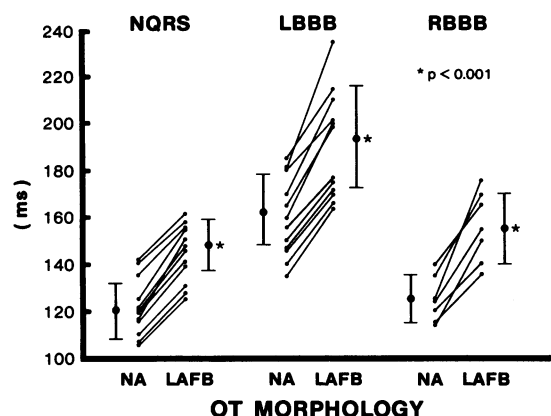
Figure 1. VACT changes due to LAFB in patients with LFW-AP. The magnitude of VACT prolongation for three types of OT in patients with LFW-AP is depicted. The occurrence of functional LAFB during narrow QRS (NQRS) OT prolongs VACT over a range of 15–35 ms (mean 23.5±5.8 ms). Concomitant occurrence of LAFB with functional LBBB or functional RBBB results in VACT prolongation of 31.8±10.1 ms (range 20–55 ms) and 31.4±11.1 ms (range 20–35 ms), respectively.

Functional LAFB

OT with narrow QRS complex. Isolated functional LAFB occurred in 2 patients with atrial stimulation (Nos. 6 and 18) and in 18 patients with ventricular stimulation (Nos. 1, 2, 5, 7–9, 13, 20, 22–25, 27–32) at the onset of OT. Of these 20 patients, four (Nos. 16–18, 28) also exhibited the same type of aberrancy during subsequent OT complexes. Two additional patients (Nos. 17 and 21) who did not demonstrate functional LAFB at the initiation of OT, developed this type of aberrancy only during subsequent complexes. The occurrence of functional bundle branch or fascicular block during subsequent complexes was related to sudden cycle length (H-H) shortening, albeit small in most instances (9, 10).

In 12 patients with LFW-AP isolated functional LAFB consistently prolonged VACT (Figs. 1 and 2) by 15–35 ms (mean 23.5±5.8 ms, *P* < 0.001). In eight of these patients whose functional LAFB occurred during multiple episodes of induced OT, the magnitude of the VACT prolongation was the same. Furthermore, in three patients who demonstrated this

type of aberrancy at the OT onset as well as during subsequent OT beat (Fig. 2), the magnitude of VACT prolongation in each patient remained unchanged. In patients with other AP locations (i.e., LPS, RFW, and RAW), isolated LAFB did not produce any VACT prolongation (Fig. 3A).

OT with functional LBBB. Functional LBBB-LAFB occurred at the onset of OT with atrial stimulation in 4 patients (Nos. 11, 14, 16, 18) and ventricular stimulation in 14 patients (Nos. 1, 2, 5, 6, 9, 13, 20, 21, 23–27, 31). Six additional patients (Nos. 3, 10, 12, 15, 17, 28) demonstrated this type of aberrancy during conduction of subsequent OT complexes (Fig. 2). Functional LBBB with normal axis which occurred in a total of 19 patients was infrequent at OT onset (Nos. 10, 29–31 with ventricular stimulation and Nos. 15 and 17 with atrial stimulation), and common during subsequent complexes (Nos. 1–3, 6, 11–14, 20, 24, 26–28) of the tachycardia. Of note, in 6 of 33 patients who did not exhibit inducible OT with functional LBBB, LAFB in isolated form (Nos. 7, 8, 22, 32) or in combination with RBBB (Nos. 4, 19, 32) was attained.

As shown in Figs. 1 and 4, in patients with LFW-AP, functional LBBB with normal axis (Nos. 1–3, 6, 10–15, 17, 20) increased VACT by 36.4±7.4 ms (range 25–45 ms) over OT complexes with narrow QRS and normal axis. However, the occurrence of combined functional LBBB-LAFB in the same patients resulted in 68.2±14.9 ms (range 50–95 ms) VACT prolongation. Of note, at the onset of OT and during subsequent OT complexes, the VACT increase due to LBBB-LAFB was the same (Fig. 4). In three patients with LPS-AP (Nos. 24, 26, 27) who demonstrated functional LBBB with and without functional LAFB, VACT prolongation (15–25 ms) was independent of the presence of LAFB (Fig. 3, *B* and *C*). Functional LBBB-LAFB did not produce any VACT prolongation in patients with RFW-AP or RAW-AP.

Complete resolution of functional LBBB-LAFB aberrancy resulted in narrow QRS and normal axis in 12 patients, whereas partial resolution resulted in functional LBBB with normal axis in six patients or isolated functional LAFB in five patients. In the remaining one patient, functional LBBB-LAFB persisted until the OT was terminated.

OT with functional RBBB. 22 patients exhibited functional RBBB with normal axis during OT. Of these, 12 patients also developed functional RBBB-LAFB occurring either at onset of
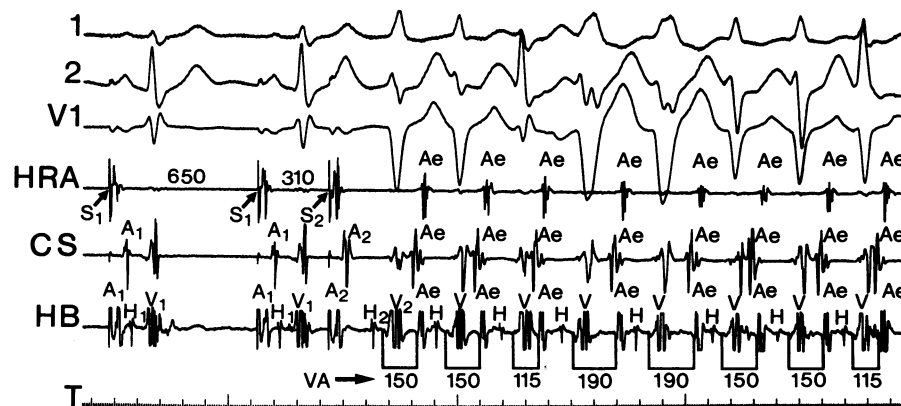
Figure 2. VACT prolongation due to functional LAFB (concealed LFW-AP, patient 16). Tracings from top to bottom are: surface ECG leads (1,2,V₁), high right atrium (HRA), coronary sinus (CS), His bundle electrogram (HB), and time line (T). A similar format is used in subsequent tracings. The site of stimulation is high right atrium and the basic drive cycle length is 650 ms. At S₁S₂ coupling interval of 310 ms, OT is initiated. Note that A₂ conduction occurs with isolated functional LAFB morphology and is followed by the same type of aberrancy during conduction of the first OT complex. These two complexes are associated with

VACT prolongation of 35 ms (as compared to VACT during last OT complex with narrow QRS-normal axis). Note also that the same magnitude of VACT change occurs during conduction of fifth and sixth complexes with isolated functional LAFB. It should also be appreciated that functional LBBB-LAFB (third and fourth OT complexes) is associated with 75-ms VACT lengthening.
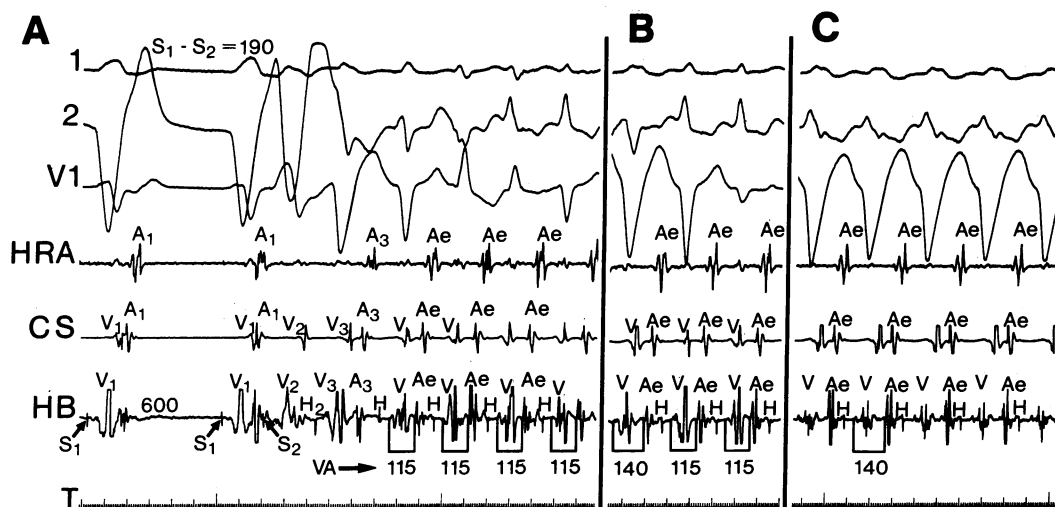
Figure 3. VACT response to functional LAFB in the presence of LPS-AP (patient 24). Stimulation site is right ventricular apex and the basic drive cycle length is 600 ms. At premature coupling interval of 190 ms (*A*), a bundle branch reentrant beat (V_3) (11) is induced which in turn blocks retrogradely in the His-Purkinje system, allowing initiation of OT (12). The first OT complex conducts with isolated functional LAFB which is followed by functional RBBB-LAFB. Note that during these two aberrant morphologies, VACT remains the same as during narrow QRS with normal axis (fourth OT complex). In *B*, functional LBBB-LAFB is associated with 25-ms VACT prolongation which is the same as during functional LBBB with normal axis (*C*).

OT (Nos. 4, 9, 18 with atrial stimulation and Nos. 3, 24, 26, 29, 30–32 with ventricular stimulation) or during conduction of subsequent OT complexes (Nos. 11 and 16). Additionally, one patient (No. 19) who consistently exhibited RBBB morphology during OT, demonstrated superimposed functional LAFB at OT onset initiated by ventricular stimulation.

In patients with LFW-AP, functional RBBB with normal axis did not have any effect on VACT, however, the occurrence of combined functional RBBB-LAFB (Figs. 2 and 5) resulted in 20–35-ms VACT prolongation (31.4 ± 11.1 ms, $P < 0.001$). Similarly, one patient (No. 19) with persistent RBBB during OT (probably preexisting), demonstrated 35-ms VACT prolongation due to superimposed functional LAFB. In three patients (Nos. 9, 16, 18) who manifested both isolated functional LAFB and functional RBBB-LAFB morphologies, identical magnitudes of VACT prolongation were observed during the two types of aberrancy. Patients with LPS-AP (Nos. 24 and 26) did not have any VACT prolongation during functional RBBB with and without LAFB (Fig. 3). In patients with

RFW-AP and RAW-AP (Nos. 29–32) who exhibited functional RBBB with and without LAFB, VACT prolongation (20–35 ms) was independent of the presence of LAFB. The resolution of functional RBBB-LAFB was complete in four patients resulting in narrow QRS-normal axis morphology and incomplete in 8 patients resulting in functional RBBB with normal axis (Figs. 3 and 5).

We contemplated the OT cycle length variation as a possible alternative explanation for VACT prolongation. Therefore, in every patient who exhibited OT cycle length variation, the VACT was compared between the shortest and the longest retrograde AP input (i.e., V-V cycle length) preceding each type of OT. It was noted that despite significant statistical cycle length difference ($P < 0.001$) during each group of OT (narrow QRS-normal axis, 26.4 ± 9.6 ms; isolated LAFB, 24.5 ± 9.8 ms; isolated RBBB, 24.2 ± 9.3 ms; RBBB-LAFB, 25.4 ± 9.3 ms; isolated LBBB, 23.7 ± 7.6 ms; LBBB-LAFB, 25.9 ± 6.9 ms), VACT remained unchanged. Furthermore, comparing the V-V cycle length preceding functional LAFB (isolated or combined with
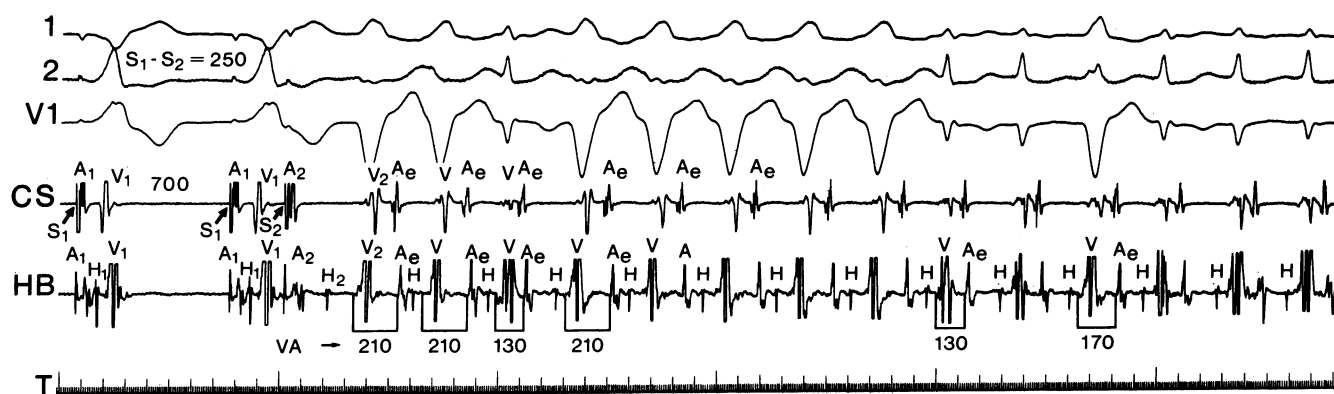
Figure 4. VACT prolongation due to LBBB-LAFB (manifest LFW-AP, patient 14). The site of stimulation is CS and the basic drive cycle length is 700 ms. At premature coupling interval of 250 ms, OT is initiated. The A_2, the first and the third through the seventh OT complexes are conducted with functional LBBB-LAFB morphology. Note that compared to complexes with narrow QRS and normal axis, combined functional LBBB-LAFB increases VACT for 80 ms. However, subsequently a single beat of functional LBBB with normal axis occurs which is associated with only 40-ms VACT prolongation.
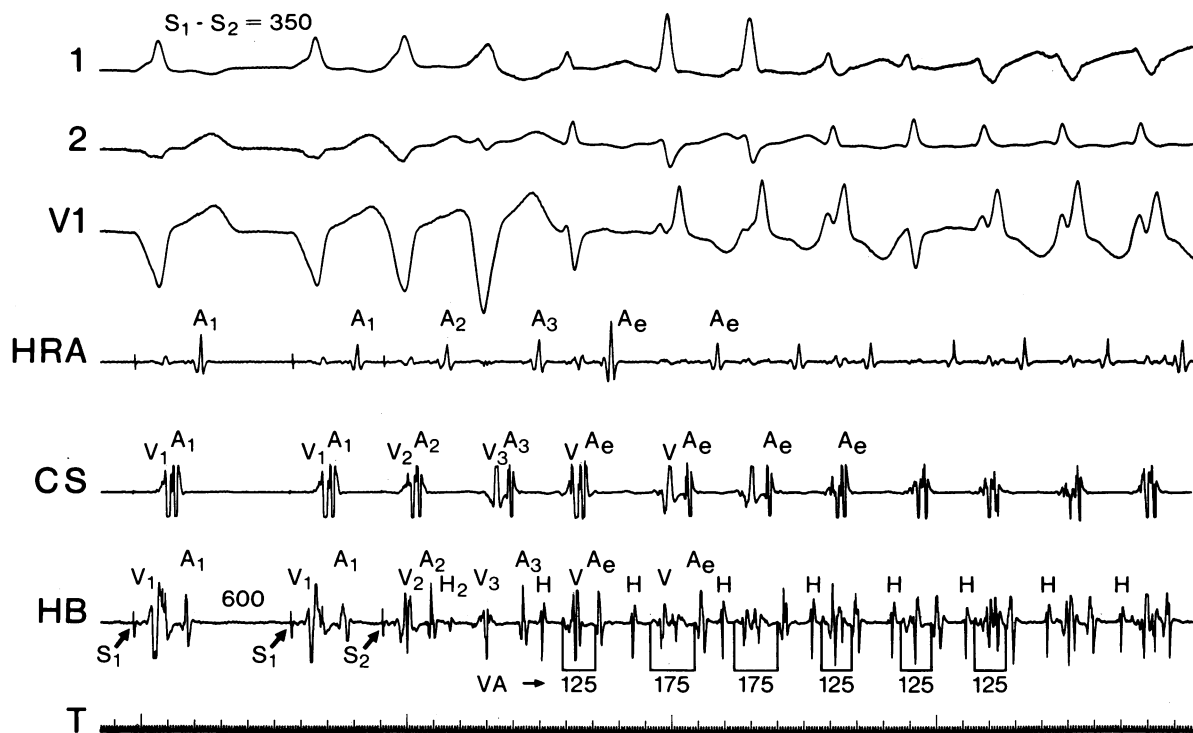
Figure 5. VACT prolongation due to RBBB-LAFB (patient 11). The stimulation site is right ventricular apex and basic cycle length (S_1S_1) is 600 ms. At coupling (S_1S_2) interval of 350 ms, a bundle branch reentrant beat (V_3) is induced which in turn blocks retrogradely in the His-Purkinje system allowing initiation of OT. The first OT complex with narrow QRS-normal axis morphology is followed by complexes showing functional RBBB-LAFB morphology. The beats with RBBB-LAFB have VACT values of 175 ms as compared to 125 ms during narrow QRS complexes. Incomplete resolution of functional RBBB-LAFB results in functional RBBB normal axis and VACT becomes normal. Note that VACT during functional RBBB-LPFB (last three OT complexes) also remains similar to those during narrow QRS complexes.

bundle branch block) between patients with LFW-AP and those with other AP locations did not show any significant statistical difference.

Functional LPFB

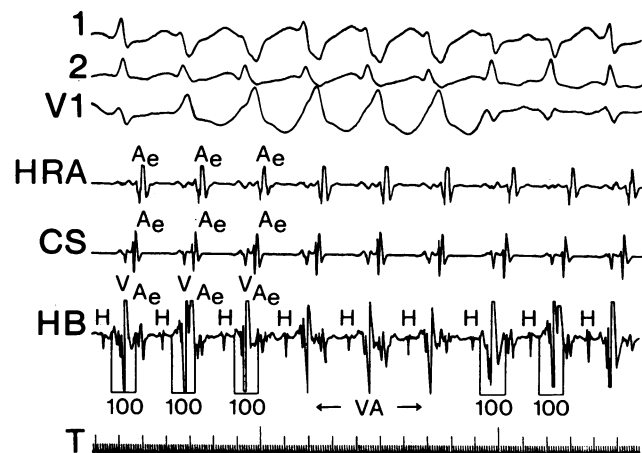Functional LPFB occurred either in isolated form or in combination with functional RBBB (Fig. 6) but, at no time was

Figure 6. Functional LPFB in the presence of LPS-AP (patient 27). VACT during narrow QRS-normal axis OT beats (first and last complexes) is 110 ms which remains unchanged during isolated functional LPFB (two complexes before last) or combined RBBB-LPFB (second through sixth complexes).

associated with functional LBBB in this series. The occurrence of isolated functional LPFB was at OT onset in seven patients (Nos. 1, 2, 12, 22, 26, 31 with ventricular stimulation and No. 14 with atrial stimulation) and during subsequent OT complexes in two patients (Nos. 24 and 27). Functional RBBB-LPFB aberrancy occurred either at OT onset (Nos. 10–12, 14, 27 with atrial stimulation and No. 8 with ventricular stimulation) or during subsequent OT complexes (Nos. 1, 2, 7, 13, 15–17, 21, 23, 24, 28). It should be mentioned that the resolution of functional RBBB-LPFB was either complete, resulting in narrow QRS with normal axis (five patients) or incomplete, resulting in functional RBBB with normal axis (nine patients) or isolated functional LPFB (three patients). The occurrence of functional LPFB, alone or in combination with RBBB, had no effect on VACT in any of the patients.

Sustenance of FFB

Although isolated FFB manifested as a single-beat phenomenon in the majority of patients, occasionally, two to four consecutive OT complexes showing the same type of aberrancy were observed: 3 of 22 patients (Nos. 16, 18, 21) with isolated functional LAFB (Fig. 2) and 1 of 9 patients (No. 27) with isolated functional LPFB (Fig. 6). The sustenance of combined FFB and bundle branch block, however, was observed more frequently: functional LBBB-LAFB (Fig. 4) in 15 of 24 patients (Nos. 1, 2, 6, 9–11, 13–17, 20, 21, 26, 27), functional RBBB-LPFB (Fig. 6) in 7 of 17 patients (Nos. 1, 10, 11, 14–16, 27), and functional RBBB-LAFB (Fig. 5) in 3 of 12 patients (Nos. 9, 11, 18). Of particular note, sustained functional LBBB

in any given patient was solely associated with one type of axis (normal or leftward) although isolated beats showing both types of aberrancies were seen. It should be pointed out that sustenance of isolated FFB (LAFB or LPFB), functional RBBB-LPFB, and functional RBBB-LAFB was only observed when these types of aberrant conduction occurred either at the onset of OT induced by atrial stimulation or during antegrade propagation of subsequent OT complexes. In contrast, sustenance of functional LBBB-LAFB was independent of initiation method of aberrant conduction, (A_2, V_2, or subsequent OT complexes) although as presented earlier LBBB-LAFB was more frequently induced with ventricular stimulation.

Discussion

Results of this study indicate that at the onset of OT, the occurrence of FFB, especially LAFB, in isolated form or combined with functional bundle branch block is a common phenomenon. Ventricular stimulation was by far, the most common method of OT initiation which generated functional LAFB at OT onset: 18 of 20 patients (90%) with isolated functional LAFB, 14 of 18 patients (77.8%) with functional LBBB-LAFB, and 7 of 10 patients (70%) with functional RBBB-LAFB. The exact reason for a higher incidence of functional LAFB at OT onset during right ventricular stimulation is not completely clear. However, mechanisms similar to those which have been previously offered to explain frequent occurrence of functional LBBB at OT onset during ventricular stimulation (8) may also be operative in these cases as well. The less frequent occurrence of isolated FFB during atrial stimulation may be due to the longer functional refractory period of the AV node and/or the relative refractory period of the proximal portion of the left bundle system as compared to the left anterior fascicle in these cases (13, 14).

Effect of fascicular block on VACT. The most important and clinically useful observation during this study relates to the effect of FFB on VACT. In patients with LFW-AP, the occurrence of isolated functional LAFB or functional RBBB-LAFB during OT was associated with significant VACT prolongation. Thus, one could reasonably conclude that in the presence of these aberrancies, the antegrade impulse must reach the AP by traveling through the left ventricular myocardium in the distribution of left anterior fascicle. This slower intramyocardial impulse propagation, as previously shown by epicardial mapping (15), results in delayed activation of the anterolateral left ventricle and, hence, prolongation of the VACT in these patients. Furthermore, these data demonstrate that although functional LBBB with normal axis produced remarkable VACT prolongation, the occurrence of functional LBBB-LAFB resulted in even greater magnitudes of VACT prolongation. A possible explanation for this observation is as follows: in the presence of functional LBBB with normal axis the antegrade impulse propagates to the left ventricle by traversing the septum and reaching the LBB distal to the site of block. From this point on the impulse could rapidly spread antegradely along the distribution of fascicles in a somewhat normal fashion. The VACT prolongation during this type of aberrancy is therefore, primarily due to transseptal conduction time (30–60 ms) (16). In the presence of functional LBBB-LAFB on the other hand, the transseptal impulse has to propagate through a larger area of the left ventricular myocardium before reaching the anterobasal portion of the left ventricle

and hence the AP (17). Thus, greater magnitude of VACT prolongation in this type of aberrancy is due to the further conduction delay generated by intramyocardial propagation of the impulse through the left ventricle in addition to the transseptal conduction time.

If the activation of the posterior paraseptal area was provided by the left posterior fascicle (18), one might expect some degree of VACT prolongation during OT with functional LPFB aberrancy in patients with the LPS-AP. However, our results were in marked contrast to that expectation, inasmuch as we did not observe any VACT changes associated with functional LPFB (in isolated form or combined with functional RBBB) in this subgroup of patients. This rather unexpected finding might be accounted for by the fact that the left ventricular activation in the region of the LPS-AP insertion may be provided by the left septal fascicle (18–20). Further work is obviously needed to more clearly delineate the electrophysiologic characteristics of functional left septal fascicular block (as an isolated form or combined with other types of aberrant conduction) and thereby, to highlight its role in the intraventricular conduction delay.

Sustenance of FFB. In the course of this study we noted that FFB only sustained when it: (*a*) occurred during propagation of atrial impulses (i.e., A_2 or subsequent OT beats) or (*b*) combined with functional LBBB. In order to explain this phenomenon one needs to address the site of initial FFB, and also the route by which the retrograde penetration of the antegradely blocked fascicle is provided. It has been shown in animals (21–23) and been suggested in humans (11, 24) that the site of functional RBBB is usually located proximally during atrial stimulation and distally during ventricular stimulation. It is conceivable that the site of FFB would also depend upon the site where the impulse originates; i.e., proximal FFB during propagation of atrial impulses (Fig. 7, *a* and *b*) and distal FFB during ventricular stimulation (Fig. 7, *d–f*). Therefore, as shown in Fig. 7, a proximal FFB could allow deeper retrograde penetration of the fascicle via its counterpart (interfascicular activation). Consequently, by the arrival of the next antegrade impulse, the fascicle would still be refractory from its prior retrograde excitation. Repetition of this process could maintain the FFB for several cycles. When a functional block concomitantly occurs in both left anterior and posterior fascicles by an atrial impulse (Fig. 7 *c*), or during ventricular stimulation (Fig. 7, *d, g, h*), propagation of the antegrade impulse would assume a combined LBBB-LAFB morphology. Once this aberrancy is initiated, delayed retrograde penetration of the two fascicles via transseptal activation (linking phenomenon) (25) could maintain functional LBBB-LAFB, regardless of the site of block.

Limitations of the study. Since FFB often lasted for a single or a few consecutive beats and was not always sustained, one might argue that VACT changes were mainly due to OT cycle length variations associated with aberrant conduction. This is unlikely to be the case as discussed below. First, it has been previously demonstrated that the VACT increase during the first and subsequent beats of OT showing the same type of functional bundle branch block (ipsilateral to AP location) is of similar degree (8). This study again reaffirms that the magnitude of VACT prolongation due to LAFB (with or without bundle branch block) at OT onset is also the same as those during the subsequent OT beats, irrespective of OT cycle length changes. It appears therefore, that isolated beats with a
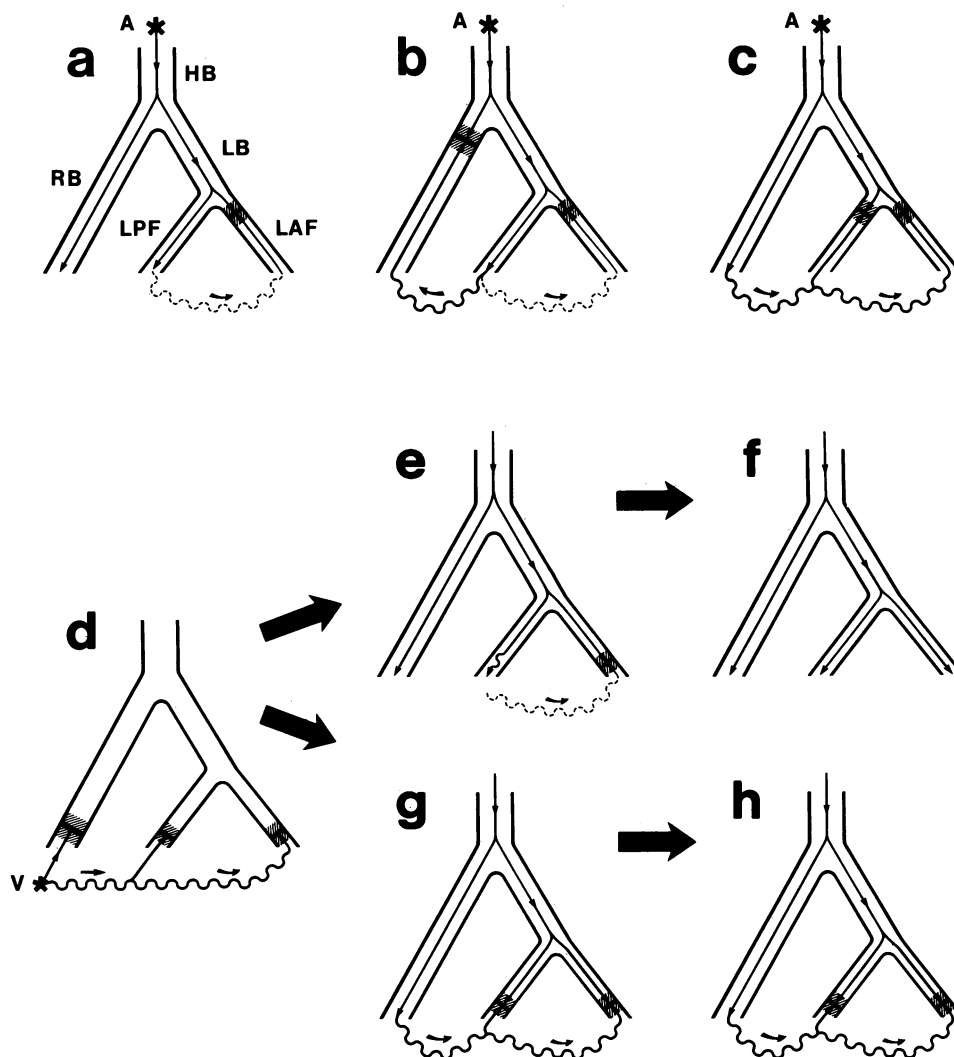
Figure 7. Schematic representation of the proposed mechanism of sustained functional fascicular block during antegrade and retrograde propagation of impulses. (*a*) An atrial impulse (paced or spontaneous) propagating over the His bundle (*HB*), left and right bundles (*LB* and *RB*) with a proximal block in the left anterior fascicle (*LAF*). The antegrade impulse via left posterior fascicle (*LPF*) penetrates LAF retrogradely (interfascicular activation shown as a dotted undulating line) and renders LAF refractory to the next antegrade impulse. Hence, repetition of this process could sustain functional LAFB for several cycles. (*b*) The atrial impulse blocks in the proximal LAF and proximal RB (23, 24). The retrograde penetration would lead to similar events shown in *a* as far as LAF is concerned. (*c*) The antegrade impulse blocks in both LAF and LPF resulting in functional LBBB-LAFB. Retrograde penetration of LAF and LPF following transseptal conduction (shown as a solid undulating line) renders both fascicles refractory to the next antegrade impulse and therefore, this type of aberrancy sustains for several cycles. (*d*) A premature ventricular impulse retrogradely blocks in the distal RB and traverses the septum to reach LB system. Note that this transseptal impulse blocks distally in both LPF and LAF. Due to the anatomic location of these fascicles, penetration and, hence, recovery of LAF occurs

at a later time as compared to its posterior counterpart. Thus, during propagation of the next antegrade impulse, distal LAF could still be refractory (*e*). However, this distally located functional block in LAF may not allow sufficient penetration of LAF during interfascicular activation (in contrast to a proximally located block as shown in *a*). Consequently, by the time the next impulse arrives, LAF is no longer refractory (*f*). However, when an appropriately timed antegrade impulse encounters both LAF and LPF refractory from their prior retrograde concealment, functional LBBB-LAFB occurs (*g*). Delayed retrograde activation of LAF and LPF due to transseptal conduction results in delayed recovery during successive cycles and leads to the sustenance of this aberrancy (*h*).

given type of aberrant conduction provide the same information concerning AP localization as sustained aberrancy of the same type. This information can be clinically quite useful when AP localization is critical and sustained aberrancy is not attained. Second, if this argument were relevant one would expect to observe VACT changes at the beginning of OT regardless of AP location. This is clearly not the case since in this series despite comparable OT cycle lengths, VACT prolongation due to LAFB was only observed in patients with LFW-AP.

In conclusion, the results of this study convincingly demonstrate that the occurrence of functional LAFB during OT is a common phenomenon which can provide useful information for separation of LFW-AP from all other AP locations.

Acknowledgments

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