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Research Article

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Effects of Alternating Cycle Lengths on Refractoriness of the His-Purkinje System

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Abstract. The effects of alternating cycle lengths (bigeminal rhythm) on His-Purkinje system refractoriness were studied in 14 patients using His bundle and right bundle recordings. Programmed atrial stimulation at constant cycle length (method I) was scanned using the atrial extrastimulus technique (A2) and compared with an atrial cycle length of identical duration coupled to A2 on alternate beats (method II). The results showed that (*a*) despite shorter cycle length of the His-Purkinje system with method II due to effect of A2 on atrioventricular nodal conduction (699±90 vs. 743±87 ms, $P < 0.001$), the relative refractory period of the His-Purkinje system was always longer with method II (463±52 vs. 440±43 ms, $P < 0.001$). A similar increase also occurred in effective refractory period of the His-Purkinje system; (*b*) while functional right bundle branch block occurred in eight patients and functional left bundle branch block in two patients with method I, functional right bundle branch block occurred in all 14 patients and left bundle branch block in seven patients with method II; (*c*) in two patients where both functional right and left bundle branch block occurred with method I this never was manifest at identical degree of abbreviation of His-Purkinje system cycle length but was manifest at identical abbreviation in each of seven patients with method II; (*d*) site of conduction delay and/or block during functional right bundle branch block was always proximal, i.e., between the His bundle and right bundle recordings with both methods. During method II this resulted in shortening of the subsequent right bundle cycle length relative to the subsequent His bundle (and of necessity left bundle) cycle length.

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The finding of increased His-Purkinje system refractoriness despite shorter preceding cycle length of the His-Purkinje system during atrial bigeminy has never been previously described and suggests that classical concepts of His-Purkinje system behavior may require revision in this setting. Secondly, during atrial bigeminy the occurrence of alternating functional bundle branch block cannot be accounted for solely by the degree of abbreviation of His-Purkinje system cycle length, but may be explained by a relative shortening of the next ipsilateral bundle branch cycle length in the bundle manifesting block.

Introduction

Functional conduction delay or block within the His-Purkinje system (HPS)[1] during propagation of premature atrial (A2) impulses is a well recognized phenomenon that may manifest as right (R) or left bundle branch block (LBBB) or bilateral block within the HPS (1–13). These patterns of aberrant ventricular conduction (VAb) or block within the HPS are contingent upon HPS refractory period properties, which are known to be cycle length (CL) dependent, varying directly with CL preceding A2 (4, 9, 13). Previous studies have also shown that different degrees of CL abbreviation are necessary for manifestation of RBBB vs. LBBB and this has been attributed to differences in refractoriness between the RBB and LBB (3).

Considering these concepts, there exists in the literature an unexplained paradox concerning VAb. In a report by Cohen et al. (4) on VAb in man, the authors suggested that an atrial bigeminal rhythm, whereby A2 was coupled to every sinus beat, increased the likelihood of VAb by producing a prolonged CL following A2. Interestingly, it was also observed that such a pacing method produced alternating patterns of VAb with each

1. *Abbreviations used in this paper:* AV, atrioventricular; BB, bundle branch; BBB, BB block; BCL, basic CL; CL, cycle length; ECG, electrocardiogram; ERP, effective refractory period; FRP, functional refractory period; HB, His bundle; HV, His-ventricular; HPS, His-Purkinje system; LBBB, left BBB; RB, right bundle; RB-V, RB-ventricular; RBBB, right BBB; RRP, relative refractory period; VAb, aberrant ventricular conduction.

A2 (i.e., RBBB alternating with LBBB) despite identical A2 coupling intervals. This would be a distinctly unusual finding, if indeed, the HPS CL changes were also identical. While the role of changes in the cycle lengths of the bundle branches (BBs) during alternate patterns of VAb have been postulated (5, 11) this has never previously been demonstrated in man.

In an attempt to explain this phenomenon of alternating patterns of functional BBB, the present study used both His bundle (HB) and right bundle (RB) recordings and a programmed atrial stimulation specifically designed protocol to permit identical CL sequences. Moreover, in view of recent findings concerning the effect of abrupt alterations in atrial CL on refractoriness of the HPS (14), this study attempted to determine if the alternating CL changes per se, rather than prolonged CL (as occurs with A2 coupled to sinus beats), contributes to the increased likelihood of VAb.

Methods

Electrophysiologic studies were performed in the unsedated, postabsorptive state after obtaining signed consent to the explained procedure (15). Using local anesthesia and fluoroscopic guidance, multipolar electrode catheters were percutaneously introduced and positioned in the high right atrium, the atrioventricular (AV) junction, and the right ventricle. The simultaneous recordings from the HB and RB were obtained either with a single quadripolar catheter (interelectrode distance 1 cm) or with two separate electrode catheters. All intracardiac electrograms (filtered at 30–500 Hz), three surface electrocardiographic leads (I, II, and VI), and time lines were simultaneously displayed on a multi-channel oscilloscope and recorded on magnetic tape for later reproduction. Electrical stimulation was performed with a digital stimulator capable of delivering rectangular impulses of variable voltage and duration with total current < 3 mA. In none of the patients was the magnitude or duration of the electrical impulse altered during the study. All equipment was grounded and an isolation unit was used for electrical stimulation.

Patients were studied with the conventional technique (method I) of atrial premature stimulation (A2) wherein the A2 was introduced after a series of six atrial beats of predetermined constant cycle length (A1A1). Scanning of constant atrial cycle length with A2 was initiated at relatively long A1A2 intervals which were outside the relative refractory period (RRP) of the HPS and were decreased by 10 ms intervals until functional refractory period (FRP) of the AV node or atrial refractoriness was encountered.

With method II premature atrial stimulation was coupled to an atrial CL of identical duration as used in method I, however, every other beat was premature producing a bigeminal rhythm. This method used the same initial six-beat atrial drive of constant CL and began the alternating CL pattern with A2 of method I. As shown in Fig. 1 this results in the designation of the CL in method II as A2A1, which equals A1A1 in method I. This CL was scanned with A2 in a similar fashion as during method I.

Definition of terms (14, 16, 17). The His-ventricular (HV) and RB-ventricular (RB-V) intervals were measured from the onset of respective deflections to the earliest detectable ventricular activity, whether recorded on surface electrocardiogram (ECG) or one of the local electrograms. RRP of the HPS is the longest H1H2 interval at which H2 conducts to the ventricles with a longer HV interval than the basic drive beat or with a QRS showing a definite BBB pattern. Effective refractory period (ERP) of the HPS is the longest H1H2 interval at which H2 does not propagate to the ventricles.

Patient population. All patients included in this study were in sinus rhythm with normal intraventricular conduction on resting ECG, had normal atrium-His and HV intervals, and manifested VAb with atrial premature stimulation during one or both methods of stimulation described above. The 14 consecutive patients (nine males, five females) were 23–68 years old (mean 45±13) and were studied for various reasons, predominantly for palpitations, syncope, or ventricular tachychardia. The underlying structural heart disease was arteriosclerotic in six, mitral valve prolapse in one, valvular in one, while the remaining had no clinically evident heart disease. No patient in this series was taking cardioactive medications within 48–72 h before the study. In nine patients, satisfactory RB recordings were obtained (16, 17) and used to determine the site of functional delay and/or block along the H-RB axis (17).

Statistical analysis. Mean values are expressed with corresponding standard deviations. Comparison of mean values was done using the paired t test. Statistical significance was defined at the 5% level.

Results

The electrophysiologic data are listed in Tables I–III. For the 14 patients in this study the atrial basic cycle length (BCL) used was 743±87 ms (range 650–900 ms). The HV interval ranged from 40 to 55 msec (mean 48±5 ms). In the nine patients (Nos. 1–9) where RB recordings were obtained, the H-RB ranged from 20 to 35 ms (mean 27±6) and the RB-V ranged from 10 to 30 ms (mean 21±7).

Effect of alternating changes in atrial CLs on H-H CL. Since the atrial drive was constant during method I, A1A1 equaled H1H1. However, during method II, the atrial BCL (designated A2A1) was always longer than H2H1 due to the effect on AV nodal conduction of A2 (Table I, Fig. 1) (14). Therefore, the H-H interval coupled to A2 was longer in method I than in method II (743±87 vs. 699±90 ms, $P < 0.001$). For a given atrial BCL and A2 during method II, the subsequent H2H1 intervals and H1H2 intervals remained constant. These effects are shown in Fig. 1 *A* where the atrial bigeminal rhythm is preceded by a constant atrial drive, (the last three beats of which are the initial complexes in this panel). Note that at constant atrial CL A1A1 = H1H1, but during atrial bigeminy A2A1 > H2H1 due to A2H2 delay.

Effect of alternating changes in CL on HPS refractoriness (Table I). An increase in HPS refractoriness occurred in all patients with method II compared with method I despite the shorter H-H CL coupled to A2 with method II described above. This is illustrated in Figs. 2 and 3, which show the onset of functional RBBB, LBBB, and bilateral block within the HPS with method I compared with method II. The data for the same patient is plotted for both methods in Fig. 4, which graphically depicts the longer RRP and ERP HPS with method II. While RRP HPS was encountered in all patients with method II, in six patients RRP HPS was less than FRP AV node with method I, and therefore, was not encountered with this method. During method I the FRP AV node in these patients, however, was less

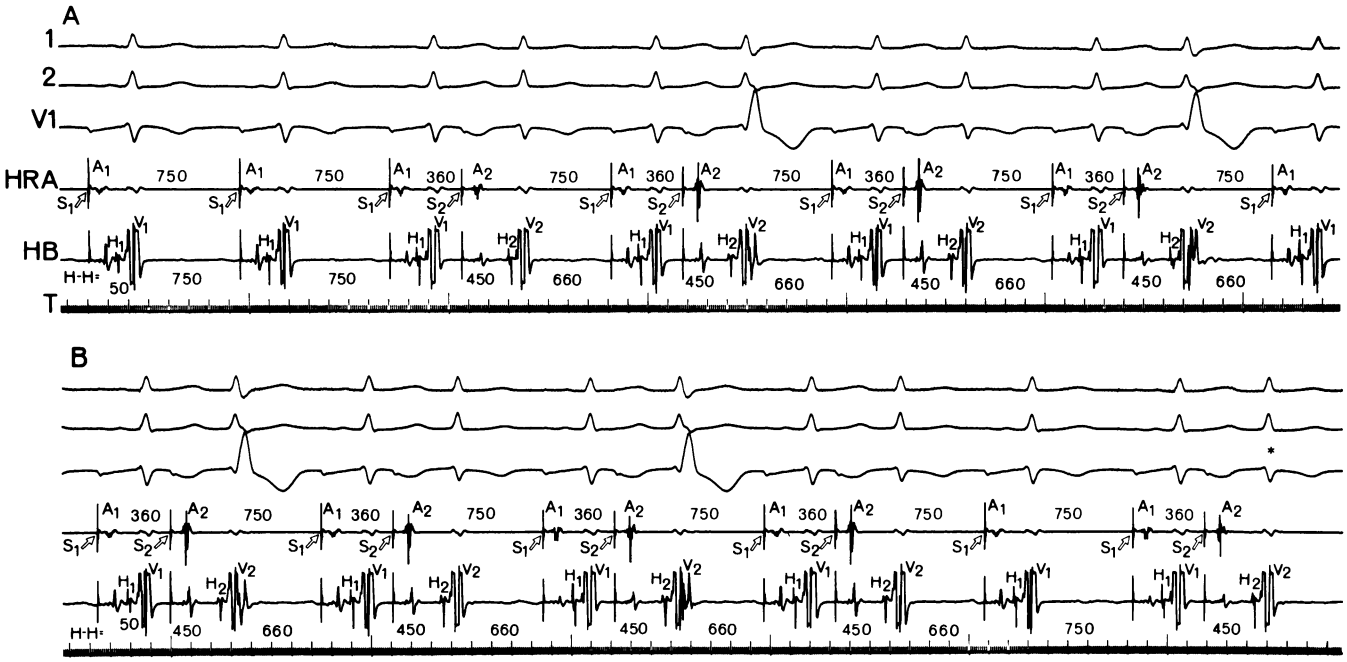


Figure 1. The tracings from patient No. 10 illustrate the pacing methods used (*A*), the effect of alternating changes in atrial CL on the subsequent "long" H-H interval, the increase in RRP HPS with this method despite shorter "long" H-H interval preceding A2, and the alternation of RBBB pattern VAb with no VAb at identical H1H2 intervals. Note the last three beats of the constant atrial CL drive (first three beats of *A*) have a constant H-H CL equal to atrial drive of 750 ms (method I). An A1A2 of 360 ms produces an H1H2 of 450 ms and impulse propagation with essentially normal intraventricular conduction. Subsequent alternating long-short atrial CLs with identical intervals (method II) result in a long H-H interval of only 660 ms, a decrease of 90 ms compared with constant CL. This occurs due to increase in AV nodal conduction time with A2. Nonetheless, at identical H1H2, VAb with RBBB occurs with the next A2. (Note that the long CLs during alternating CLs are labeled A2A1, H2H1, V2V1.) However, with the next long-short CL sequence VAb is abolished despite identical H2H1 and H1H2 where previously functional RBBB occurred. Alternation of no VAb and functional RBBB with alternate A2 persists until in *B* (continuous with *A*) the alternating CL of method II is changed and two atrial CLs of 750 ms are programmed and coupled to the identical A2 of 360 ms (last three CLs in *B*). Note again that despite the again longer H-H interval of 750 ms no VAb occurs. The beat indicated by asterisk depicts the next expected occurrence of functional RBBB with A2 during alternating long-short CLs, which has been abolished by two beats of constant CL. 1, 2, V1, Surface ECG leads; HRA, High right atrial electrogram; HB, His bundle electrogram; T, time lines. All measurements are in milliseconds.

than the RRP HPS during method II. For all 14 patients the RRP HPS increased from 440±43 ms with method I to 463±52 ms ($P < 0.001$) with method II (using FRP AV node values where RRP HPS was not encountered). The ERP HPS was manifest in four patients with both methods. For each of these cases ERP HPS was longer with method II compared with method I and the mean values increased from 435±23 to 454±20 ms ($P < 0.025$) (compare Fig. 5 *D* to Fig. 6 *B*).

Effect of alternating changes in CL on patterns of VAb (Tables I and II). All patients who manifested RBBB and/or LBBB VAb with method I also manifested the same pattern VAb with method II. However, while eight patients had RBBB and two had LBBB with method I, 14 had RBBB and seven had LBBB with method II. Remarkably, while two patients had both RBBB and LBBB with method I, this occurred in seven with method II. Moreover, while it was never observed that RBBB and LBBB occurred at the same H1H2 with method I, RBBB and LBBB occurred at identical H1H2 in all seven patients with method II. Examples of this phenomenon are shown in Fig. 3 *C* and Fig. 6, *A* and *C*.

Relation between patterns of VAb and HPS conduction (Tables I–III). VAb and HPS conduction could be evaluated in terms of HV conduction in all patients and H-RB and RB-V conduction in nine patients (Nos. 1–9) where RB recordings were available. With method I, 4/8 patients during RBBB and 2/2 during LBBB manifested HV prolongation compared with sinus beats (Table I). Different magnitudes of HV prolongation during RBBB and LBBB occurred in 2/2 patients where both patterns of VAb occurred (Fig. 2, *C* and *D*). With method II, 3/14 patients during RBBB and 7/7 patients during LBBB manifested HV prolongation (Table I). During this latter method the occurrence of alternating RBBB and LBBB at identical H1H2 (7 patients) was associated with HV prolongation with both patterns of VAb (in 3 patients) or during LBBB solely in the

Table I. HPS Refractoriness and Conduction during Methods I and II

	Method I						Method II			
	BCL		Type of block HPS (longest H1H2)			FRP	BCL	Type of block HPS (Longest H1H2)		
Patient	A1A1 = H1H1	HV	RBBB (HV)	LBBB (HV)	Bilateral	AVN	A2A1/H2H1	RBBB (HV)	LBBB (HV)	Bilateral
No.										
1	700	50	480 (70)	—	455	440	700/680	520 (50)	520 (70)	
							660			470
2	800	45	—	—	—	490	800/750	510 (45)	—	—
3	700	55	420 (70)	—	—	410	700/640	430 (55)	430 (70)	—
4	650	55	—	—	—	380	650/620	385 (70)	385 (80)	—
5	700	50	—	—	—	370	700/610	400 (50)	—	—
6	600	45	370 (45)	—	—	340	600/580	405 (45)	—	—
7	800	45	—	—	—	420	800/760	440 (45)	—	—
8	700	45	450 (60)	—	420	390	700/670	465 (45)	465 (60)	
							670			
							670	425 (90)	425 (60)	455
9	700	45	450 (45)	420 (125)	410	410	700/680	480 (45)	—	
							670	450 (45)	450 (105)	
							670			420
10	750	50	—	—	—	420	750/660	450 (50)	—	—
11	700	40	390 (80)	380 (100)	—	380	700/640	430 (40)	430 (70)	—
12	800	50	—	—	—	480	800/780	570 (50)	—	—
							770	520 (60)	520 (80)	
13	900	40	480 (40)	—	455	445	900/850	495 (40)	—	
							850			465
14	900	50	460 (50)	—	—	425	900/875	495 (50)	—	—

AVN, AV node. All electrophysiologic data are given in milliseconds.

remaining patients (Tables I and II). In the three cases with HV prolongation during both patterns, HV prolongation with LBBB was 10–20 ms longer compared with RBBB. However, in one patient where conduction resumed at shorter H1H2 after BBH (gap within HPS) (10) HV prolongation with RBBB exceeded that with LBBB (Fig. 6, *A* and *C*). In nine cases where RB recordings were available (Table III) the RB potential disappeared in all patients manifesting RBBB, indicating conduction delay and/or block proximal to or within the area of RB recording (17) with both methods I and II. An example of this is shown in Fig. 2 *B* for method I and Fig. 3 *A* for method II. During LBBB with HV prolongation the RB-V intervals were comparable to sinus beats, again suggesting presence of conduction delay proximal to RB recording site. This is demonstrated in Fig. 2 *D* and Fig. 3 *C*.

Relationship between H1H1 (H2H1) CL and RB1RB1 (and RB2RB1) CL and VAb (Table III). During method I using constant BCL, H1H1 equaled to RB1RB1. However, during method II H2H1 equaled RB2RB1 only where the "long" CL followed no VAb. Because of the proximal site of conduction delay during VAb as mentioned above, the "long" CL following VAb had a shorter RB-RB CL compared with H-H CL, although the exact shortening in RB-RB CL could not be determined following RBBB. The following relationships were therefore seen: (*a*) where RBBB alternated with no VAb (Fig. 3 *A*), the H2H1 CLs (long H-H CLs) were identical preceding premature beats conducted normally and with RBBB pattern, however RB2RB1 was longer preceding RBBB compared with no VAb (Table III, Fig. 3 *A*); (*b*) where RBBB alternated with LBBB, the H2H1 CLs preceding R and LBBB were identical (Fig. 3 *C*, Fig. 6, *A*

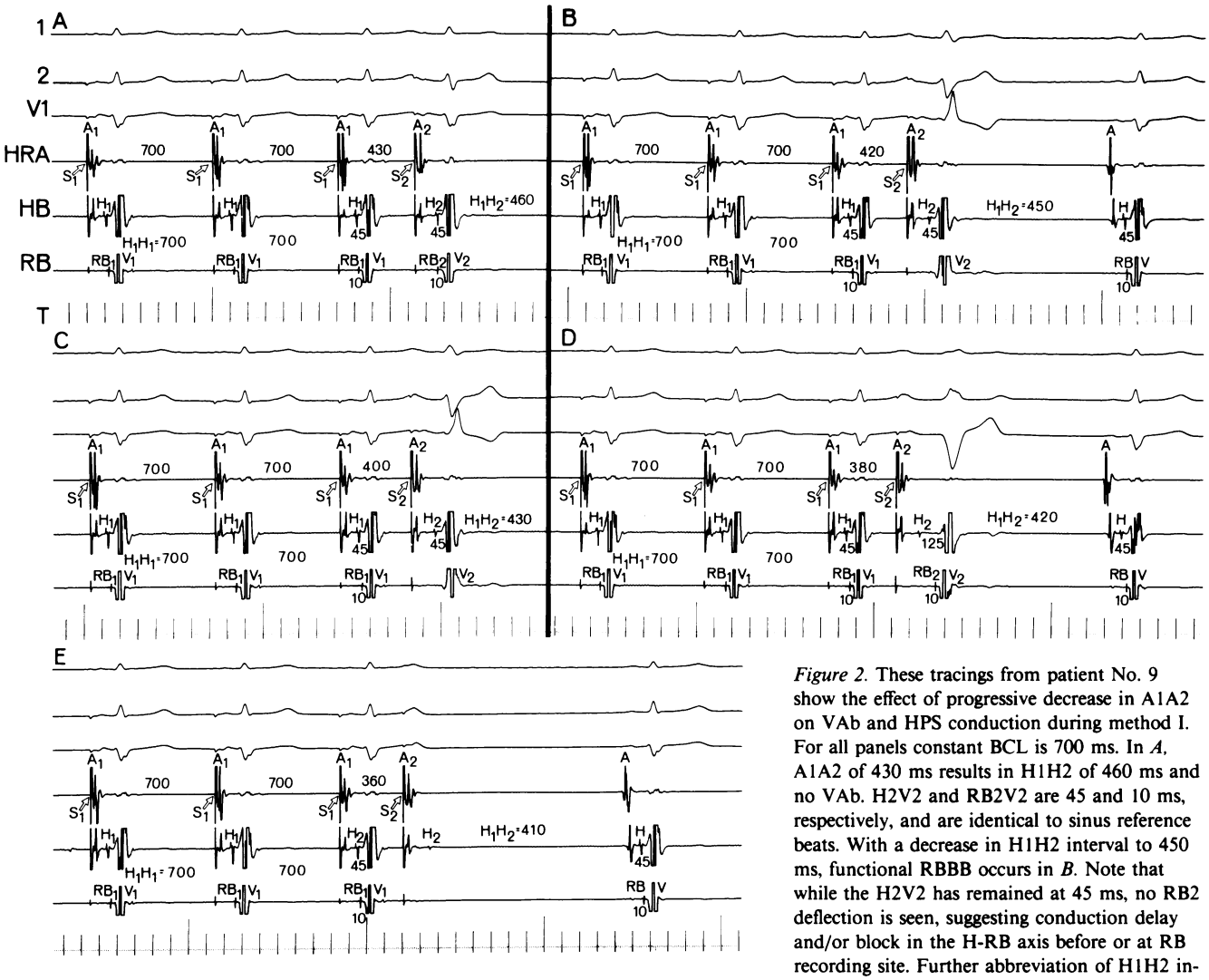
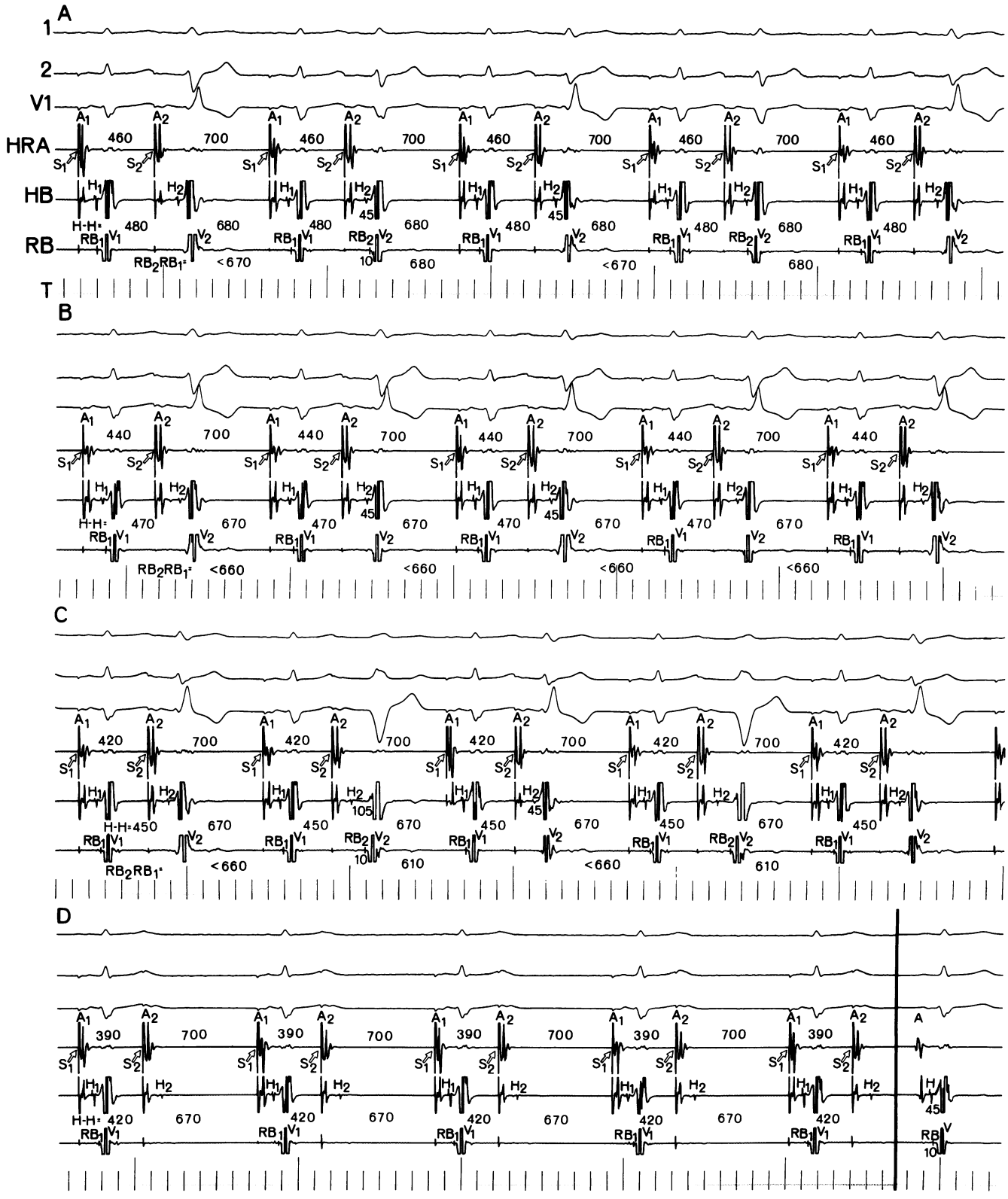


Figure 2. These tracings from patient No. 9 show the effect of progressive decrease in A1A2 on VAb and HPS conduction during method I. For all panels constant BCL is 700 ms. In *A*, A1A2 of 430 ms results in H1H2 of 460 ms and no VAb. H2V2 and RB2V2 are 45 and 10 ms, respectively, and are identical to sinus reference beats. With a decrease in H1H2 interval to 450 ms, functional RBBB occurs in *B*. Note that while the H2V2 has remained at 45 ms, no RB2 deflection is seen, suggesting conduction delay and/or block in the H-RB axis before or at RB recording site. Further abbreviation of H1H2 interval shows onset of functional LBBB at 420 ms (*D*). Note that this is associated with H2V2 of 125 ms, but RB2V2 is equal to sinus reference beats. This suggests bilateral delay, or delay and block along the H-RB and H-LB axis, respectively. Again, delay in H-RB axis appears to be proximal. *E* shows the occurrence of bilateral block below the HB but proximal to the RB (i.e., no RB2 deflection) with further H1H2 shortening. 1, 2, V1, surface ECG leads; HRA, high right atrial electrogram; HB, His bundle electrogram; RB, right bundle electrogram; T, time lines.

and *C*), however RB2RB1 CL preceding RBBB was greater than RB2RB1 CL preceding LBBB where greater magnitude HV prolongation occurred with RBBB (Fig. 6 *C*). When greater magnitude of HV prolongation occurred with LBBB (as was usually the case) RB2RB1 CL preceding RBBB was greater than RB2RB1 CL preceding LBBB as long as the difference between magnitudes of HV prolongation associated with RBBB and LBBB was less than RB-V interval during LBBB (Fig. 6 *A*); where this difference was greater than RB-V interval it could not be definitely stated that RB2RB1 CL preceding RBBB was greater than RB2RB1 CL preceding LBBB (Fig. 3 *C*). Nonetheless, if a similar pattern of conduction delay and/or block observed in the H-RB axis occurs in the H-LB axis, as previous studies suggest (3, 18, 19) then the least one can assume during alternating RBBB and LBBB is that RB2RB1 is greater than LB2LB1 preceding RBBB, and LB2LB1 is greater than RB2RB1 preceding LBBB.

Discussion

Alternating functional RBBB and LBBB is a commonly observed phenomenon during atrial premature stimulation coupled to every sinus beat. Whereas it has been postulated that this phe-


A
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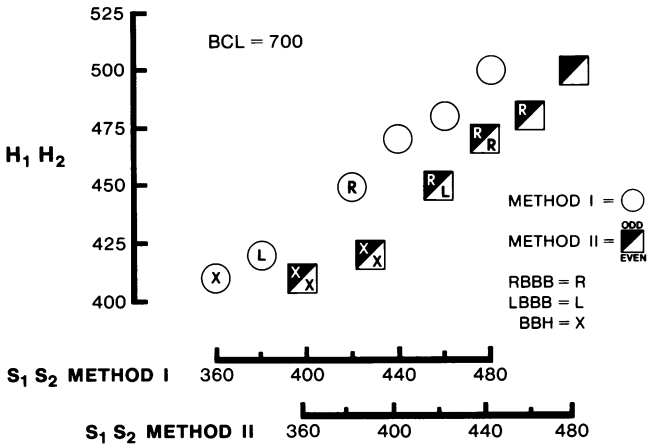

Figure 4. A plot of the data from patient No. 9 (same patient as in Figs. 2 and 3). For the most part the legends are self explanatory. However, it should be noted that the absence of letters in circles or squares indicate normal intraventricular conduction. For method II since different patterns of VAb can occur at identical H1H2, the black half of each square represents all odd numbered A2 (i.e., 1st, 3rd, 5th) while the white half represents all even numbered A2 (i.e., 2nd, 4th, 6th). Note that compared with method I, method II results in onset of functional RBBB, functional LBBB, and block below His (BBH) at longer H1H2. Also during method I, normal intraventricular conduction, functional RBBB, and functional LBBB occur at different H1H2 intervals, however, this is not the case with method II.

nomenon may be related to changes in BB CL (5), the underlying electrophysiologic mechanism has never been systematically investigated or explained. Earlier studies (4) on VAb in man have suggested that such a bigeminal rhythm can increase the likelihood of VAb. This was attributed to the longer attainable CL such a method produces with sinus escape beats following each A2. However, the effect of alternating CLs on HPS refractoriness has never been previously examined.

The present study attempted to specifically address these issues using programmed atrial stimulation whereby the identical "long" atrial CL coupled to A2 was scanned with A2 during a conventional method of constant CL (method I) and during atrial bigeminal rhythm (method II). The findings demonstrate: (*a*) an increase in HPS refractoriness with method II compared with method I despite the fact that the H-H cycle length preceding A2 is shorter with method II compared with method I; and (*b*) during atrial bigeminal rhythm alternating patterns of VAb (i.e., RBBB alternating with LBBB) can occur at identical degrees of HPS CL abbreviation, as can VAb alternating with normal intraventricular conduction (i.e., RBBB alternating with no VAb). These results could not have been predicted by classical electrophysiologic concepts of HPS behavior whereby HPS refractoriness is CL-dependent varying directly with CL (4, 9, 13, 20).

The present finding of increased HPS refractoriness with atrial bigeminal rhythm compared with constant CL is consistent, however, with our previously reported observation of increased HPS refractoriness with abrupt short-to-long CL changes (14, 21, 22). Results from these studies suggested that the HPS responds in a dynamic fashion to the rate and direction of CL change and not solely to the absolute duration of preceding CL or cumulative duration of preceding CLs. This dynamic nature of the HPS response is again seen in the present study where alternating long-short CL changes are used compared with the previous study using a basic drive of constant CL abruptly increased or decreased for one beat before A2. Remarkably, the appearance of VAb during alternation of atrial CL and disappearance after two constant atrial CL (Fig. 1) attests to the rapidity with which these dynamic changes occur. Conceivably, alternating CL changes produce greater rates of CL change and may have greater effect on HPS refractoriness than a single comparable CL change (22). While this hypothesis cannot be conclusively proven it is nonetheless evident that such CL changes have a pronounced effect on HPS behavior that needs to be considered when CLs are not constant.

The second finding of alternating patterns of VAb at identical degrees of HPS CL abbreviation is perhaps even more fascinating and has never before been described. Any proposed explanation for this phenomenon will have to explain the various patterns

Figure 3. These tracings show the effect of alternating atrial CLs (method II) on HPS refractoriness and conduction in the same patient as in Fig. 2. The same "long" atrial CL of 700 ms is used as in Fig. 2. Note, however, that due to effect of A2 on AV nodal conduction the H2H1 (long H-H CL) is decreased (to 680 ms in *A* and to 670 ms in *B–D*). Despite the shorter H2H1 interval the onset of functional RBBB occurs at an H1H2 of 480 ms (*A*) compared with 450 ms during constant CL (Fig. 2). While the H2V2 during functional RBBB remains at equal to reference sinus beats, the RB2 deflection is not present during functional RBBB similar to what occurred during method I. Note the occurrence of alternation of functional RBBB with no VAb with every other A2 despite identical H2H1 and H1H2. However, due to delayed RB depolarization during functional RBBB, the RB2RB1 CL preceding A2 with no VAb is expected to be shorter than the RB2RB1 CL preceding A2 with functional RBBB. (The "<" value is measured from onset of V2 to next RB1). In *B*, further abbreviation of H1H2 results in functional RBBB with each A2. Again the H2V2 is equal to reference sinus beats, suggesting no conduction delay along the contralateral BB, and the RB2 deflection is not present. Abbreviation of H1H2 to 450 ms (*C*) produces alternating functional RBBB and LBBB. Note the occurrence of functional LBBB at an H1H2 30 ms longer compared with constant CL (Fig. 2 *D*). Similar to the occurrence in *A* of functional RBBB alternating with no VAb with identical H2H1 and H1H2 intervals, *C* shows functional RBBB and LBBB at identical H2H1 and H1H2 intervals. During functional LBBB there is HV prolongation with no change in RB2V2 interval compared with reference beat (in *D*) and that with functional RBBB there is no change in H2V2 interval but RB2 deflection is not present compared with reference beat. In *D* bilateral block below HB occurs at an H1H2 of 420 ms, 10 ms longer than during method I (Fig. 2 *E*).

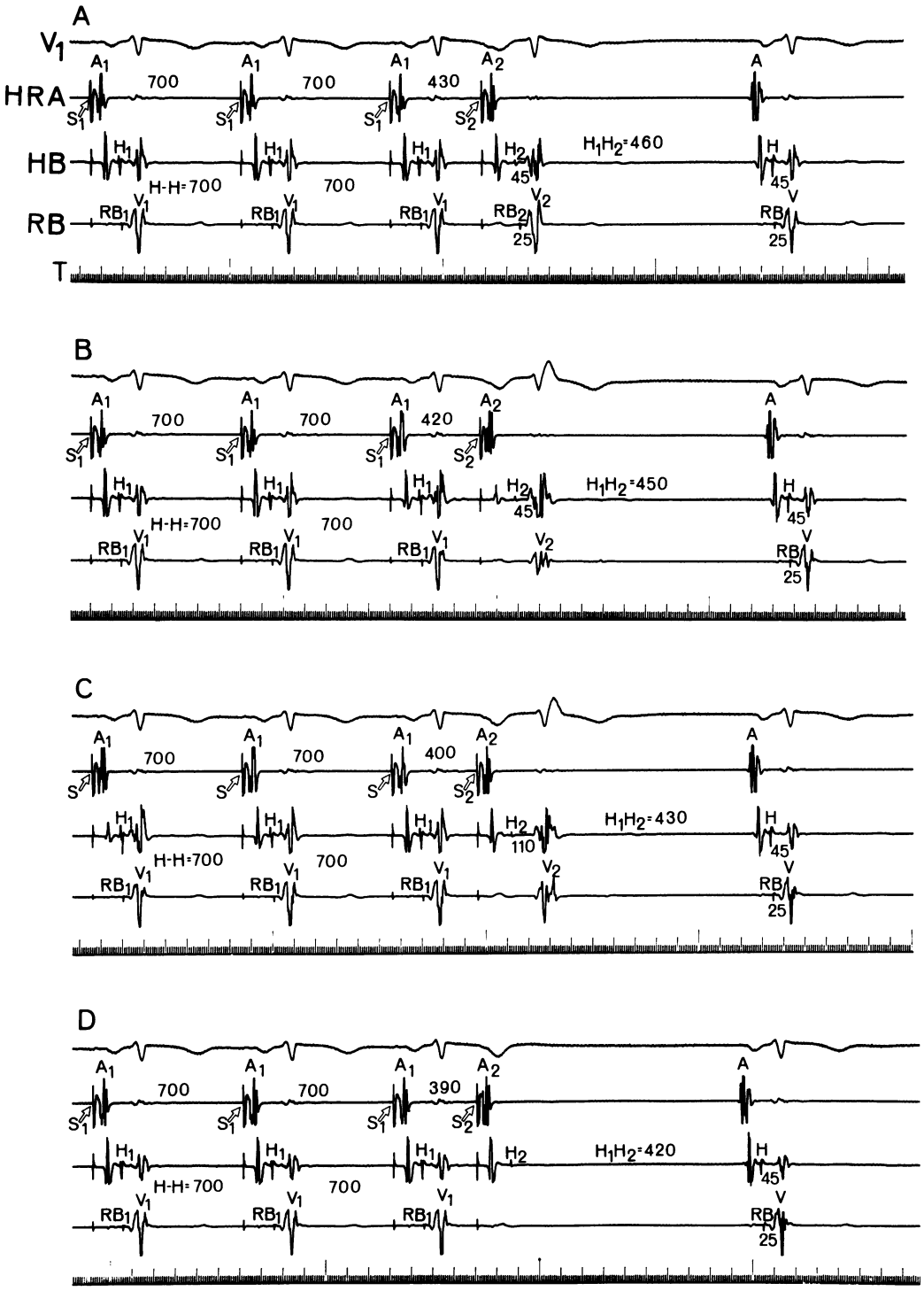

Figure 5. Illustration of method I from patient No. 8. For all panels BCL is 700 ms and HV and RB are 45 and 25 ms, respectively, on reference sinus beats. *A* shows no VAb at H1H2 of 460 ms. With a decrease by 10 ms in H1H2 interval functional RBBB occurs (*B*). Note that H2V2 has remained at 45 ms, but RB2 potential is no longer present. Further decrease in H1H2 to 430 ms results in functional RBBB with increase in H2V2 to 110 ms. Again no RB2 potential is evident (*C*). In *D* bilateral BB below HB occurs at an H1H2 of 420 ms.

of VAb noted during this study, i.e., RBBB alternating with no VAb, RBBB alternating with RBBB, RBBB alternating with LBBB, and BBH alternating with BBH. It will also need to account for the patterns of HV conduction delays observed with VAb and why certain patterns of VAb, i.e., LBBB alternating with LBBB were not evident.

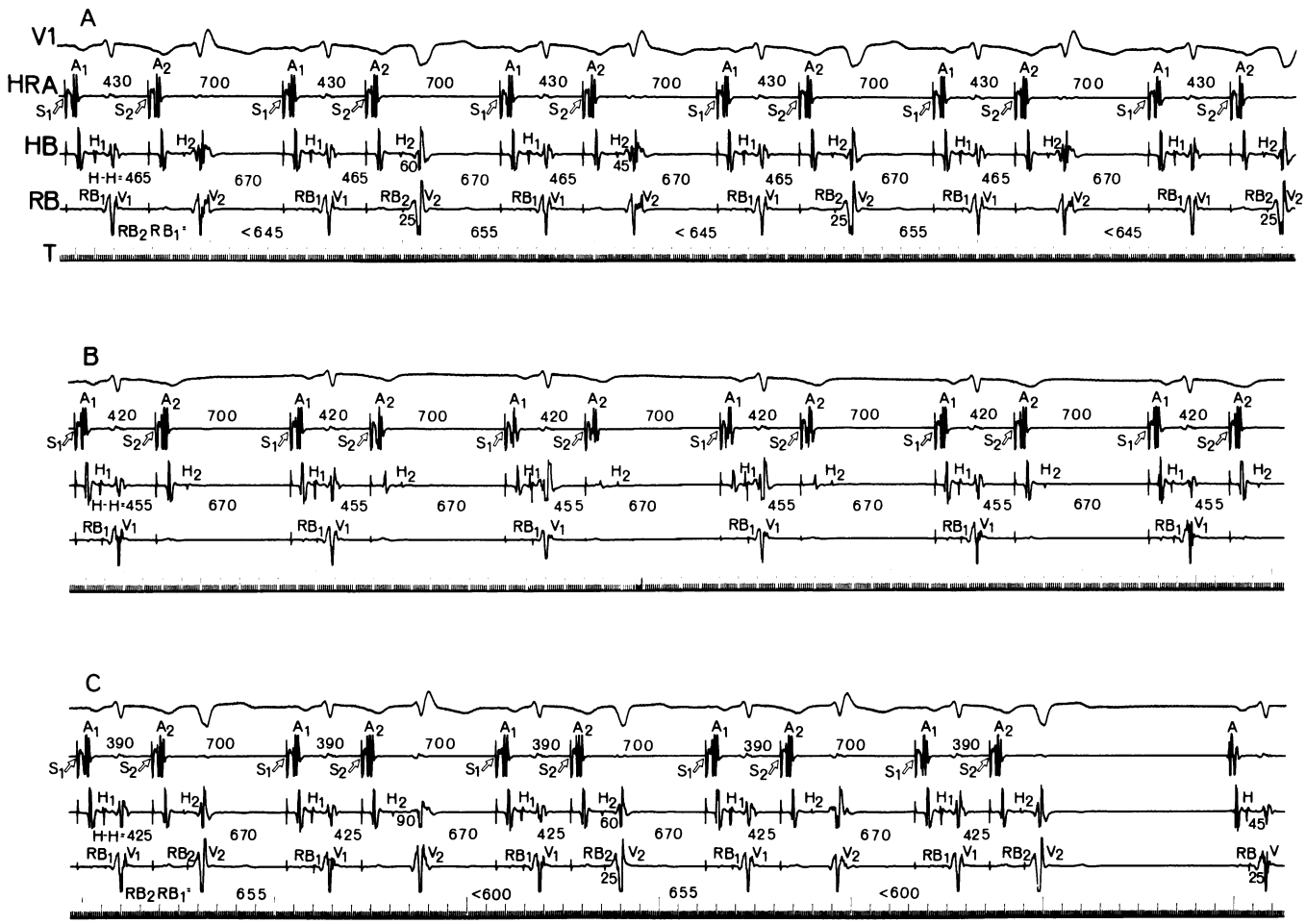

Figure 6. These tracings are from the same patient as in Fig. 5 but depict method II. Note the shorter "long" H-H CL with this method compared with method I in Fig. 5 despite the identical long atrial CL. In *A* at an H1H2 of 465 ms both functional RBBB and LBBB occur in an alternating pattern. This is a longer coupling interval than onset of functional RBBB during method I. Moreover, functional LBBB was never manifest during method I. Note that during functional RBBB the HV interval remains at 45 ms but no RB potential is recorded, whereas during functional LBBB the HV interval is 60 ms and the RB-V interval is equal to the reference sinus beat (*C*) at 25 ms. In *B* bilateral BBB below HB occurs at an H1H2 of 455 ms compared with 420 ms during method I (Fig. 5 *D*). With further H1H2 abbreviation HPS conduction again occurs with alternating functional RBBB and LBBB at identical H1H2. However, there is now HV prolongation with both functional RBBB and LBBB (compared with *A*) and prolongation is greater with functional RBBB. Again note the absent of RB potential during functional RBBB and the same RB-V interval during functional LBBB compared with the reference beat.

Results from this study as well as previous reports indicate that the site of conduction delay and/or block during VAb with A2 is usually between H and BB recording sites, probably in the proximal BBs (3, 17, 23, 24), although the present study cannot exclude a site within the HB (with longitudinal dissociation (25–28). Studies in man have suggested (17, 29–32), and canine studies by Moe et al. (3) have shown that where A2 results in functional RBBB, RB could be depolarized retrogradely via transeptal activation from impulse propagated by LB. There is therefore a significant delay in time of depolarization distal to the site of block (30 ms in canine hearts). This then sets the stage for a similar decrease in CL at this site within the HPS with the next A1 where every other beat is "long" as in the present study. Such a CL change could shorten refractoriness at this site, abolishing VAb with the next A2. The reverse sequence of CL changes at the initial site of block would then occur, i.e., a relative increase in CL at this site within the HPS with the next A1 resulting in an increase in refractoriness and manifestation of VAb with the next A2. Such an effect would result in a persistent pattern of RBBB alternating with no VAb

Table II. Patterns of VAb on Alternate A2 during Method II (Longest H1H2)

Patient	NL/NL	R/NL	R/R	R/L	BBH/BBH	Onset of HV prolongation
No.						
1	530	—	—	520	470	520
2	520	510	—	—	—	—
3	445	—	—	430	—	430
4	395	—	—	385	—	385
5	405	400	—	—	—	—
6	415	405	355	—	—	—
7	450	440	—	—	—	—
8	470	—	—	465	455	465
9	500	480	470	450	420	450
10	460	450	—	—	—	—
11	450	—	—	430	—	430
12	580	570	—	520	—	520
13	510	495	—	—	465	—
14	500	495	—	—	—	—

All electrophysiologic data are given in milliseconds. Values (other than for NL/NL) are given for onset of a given pattern of VAb. NL/NL; normal intraventricular conduction (NL) alternating with NL; R/NL, RBBB alternating with NL; R/R, RBBB alternating with RBBB; R/L, RBBB alternating with LBBB; BBH/BBH, Block below His bundle (BBH) alternating with BBH.

as was seen in this study. Where A1A2 (and H1H2) is further abbreviated, the shortening in CL described above following VAb with RBBB may not be sufficient to abolish refractoriness and RBBB may occur with each A2.

While more complex, alternating RBBB and LBBB VAb may be similarly explained. In such instances refractoriness in both BBs (or H-BB axes) is encountered with A2 in the form of conduction delay or block in the BB. In the event of block the BB manifesting the block is depolarized retrogradely from the contralateral BB and therefore significantly shortens the next BB2BB1 CL compared with the contralateral BB. Ipsilateral BB CL shortening will also take place if A2 simply shows delay rather than block within the BB distal to the site where delay starts. The resulting decrease in refractoriness of the ipsilateral BB favors the occurrence of contralateral BBB with the next A2. In a sense, alternating A2 unmasks refractoriness in the contralateral BB and probably explains the higher incidence of both R and LBBB occurring in the same patient where VAb manifests with HV prolongation with this method compared with constant CL. Such changes in BB CL should produce alternating patterns of functional BBB ad infinitum as was observed in this study.

It was also observed that during alternating BBB the HV prolongation was either present during both RBBB and LBBB or only during LBBB. A possible explanation for this may relate to the differential effect on refractoriness of BB CL shortening following VAb at and/or distal to the site of block on H-RB

Table III. Effect of Alternating Changes in Atrial CL with VAb on Bundle Branch CL

			A2 (odd beats)						A2 (even beats)					
Patient	H-V	RB-V	Type block HPS	H2H1	H1H2	H2V2	RB2V2	RB2RB1	Type block HPS	H2H1	H1H2	H2V2	RB2V2	RB2RB1
No.														
1	50	15	RBBB	680	520	50	<0	660	LBBB	680	520	70	15	<665
2	45	10	RBBB	750	510	45	<0	750	None	750	510	45	10	<740
3	55	30	RBBB	640	430	55	<0	625	LBBB	640	430	70	30	<610
4	55	25	RBBB	620	385	70	<0	595	LBBB	620	385	80	25	<580
5	50	25	RBBB	610	400	50	<0	610	None	610	400	50	25	<585
6	45	20	RBBB	580	405	45	<0	580	None	580	405	45	20	<560
7	45	25	RBBB	760	440	45	<0	760	None	760	440	45	25	<735
8	45	25	RBBB	670	465	45	<0	655	LBBB	670	465	60	25	<645
			RBBB	670	425	90	<0	655	LBBB	670	425	60	25	<600
9	45	10	RBBB	680	480	45	<0	680	None	680	480	45	10	<670
			RBBB	670	450	45	<0	610	LBBB	670	450	105	10	<660

All electrophysiologic data are given in milliseconds.

vs. H-LB axis. Since HV interval is a measurement of the conduction time between onset of HB and ventricular depolarization the results of this study would suggest that a greater magnitude of conduction delay occurs along the H-RB axis with LBBB than occurs along the H-LB axis with RBBB. This is suggested by the longer HV interval associated with LBBB compared with RBBB except in one patient (No. 8) where further H1H2 shortening produced a gap phenomenon within the HPS (10). While a higher incidence of HV prolongation with LBBB compared with RBBB VAb has been previously observed during A2 technique with constant BCL (13), conditions in the present study are modified such that during VAb the subsequent ipsilateral BB CL is shortened. It would appear that at the H1H2 range where alternating functional RBBB and LBBB manifests, CL shortening along the H-RB axis following RBBB is not sufficient to abolish refractoriness along this axis (resulting in HV prolongation with subsequent LBBB). However, shortening of H-LB axis refractoriness following LBBB may or may not be sufficient (as indicated by the absence or presence of HV prolongation with subsequent RBBB). These findings are compatible with shorter refractoriness of the H-LB axis for the CLs tested in these patients and probably explains why LBBB VAb alone with every A2 was never observed (as compared with RBBB VAb). Instead further H1H2 shortening results in bilateral block within the HPS.

In conclusion, the observations of the present study suggest that during circumstances of alternating CL changes additional factors besides the H1H1 and H1H2 relationships must be evaluated to predict onset of conduction delay and/or block within the HPS as well as the particular patterns of VAb. These factors include the dynamic response of the HPS to abrupt alterations in CL and the effect of VAb on subsequent ipsilateral bundle branch cycle length.

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