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Research Article

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His-Purkinje Conduction during Retrograde Stress

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ABSTRACT The pattern of retrograde His-Purkinje conduction was evaluated in 28 patients using ventricular extrastimuli. In each patient progressive prolongations of His-Purkinje conduction (S_2H_2) which appeared as ventricular extrastimuli were induced at closer coupling intervals (S_1S_2). There was an inverse linear relationship of S_2H_2 to S_1S_2 which was cycle length-dependent: i.e., at any S_1S_2 interval the resultant S_2H_2 was less at shorter drive cycle lengths. The degree of S_2H_2 delay varied widely (from 30 to 340 ms) and was unrelated to the presence of bundle branch block, H-V intervals, or capability of ventriculoatrial conduction. Prolongation of S_2H_2 was independent of intraventricular (muscle) conduction delay; such delay was usually absent at most, and occasionally all, S_1S_2 coupling intervals during which S_2H_2 was lengthening. Furthermore, in two patients both left and right ventricles were activated before the timed depolarization of the His bundle occurred, demonstrating that under the stress of extrastimuli, the impulse conducts through ventricular muscle with less delay than through the His-Purkinje system. We conclude that the His-Purkinje system typically displays slow conduction response to ventricular stress. The site of this conduction delay is probably at the distal "gate."

INTRODUCTION

Evaluation of His-Purkinje conduction in response to atrial extrastimuli is limited by atrio-ventricular (A-V)[1] nodal and atrial refractoriness. In the vast majority of patients these two parameters prevent the His-Purkinje system from being adequately stressed by atrial extrastimuli. During the evaluation of ventricular refractoriness and retrograde conduction in man, we have regularly observed progressive conduction delay within the His-Purkinje system. The present study was therefore undertaken to analyze and quantitate systematically the response of the His-Purkinje system to retrograde stress.

METHODS

Retrograde conduction was analyzed in 40 patients referred to the Clinical Electrophysiology Laboratory at the Hospital of the University of Pennsylvania for the evaluation of cardiac arrhythmias. These studies were approved by the Committee Concerning Human Subjects in Research at the University of Pennsylvania. All patients were studied in the nonsedated, postabsorptive state after informed consent was obtained. Antiarrhythmic agents were withheld for 24 h before the study. No. 6 or 7 electrode catheters with a 1-cm interelectrode distance were percutaneously inserted through the femoral and (or) brachial veins and fluoroscopically positioned in the high right atrium, right ventricular apex, standard His-bundle position across the tricuspid valve, and in many patients, in the coronary sinus. In some patients a catheter was also placed in the left ventricle by direct retrograde arterial approach. Quadripolar catheters were generally used in the high right atrium, coronary sinus, and ventricles. The distal pair of electrodes were used for pacing, while the proximal pair were used to record local electrograms. These intracardiac electrograms were simultaneously displayed with multiple surface ECG leads. Stimulation of the heart was performed with a specially designed programmable stimulator (Bloom Associates Limited, Narberth, Pa.), which delivered impulses at one and one-half to two times diastolic threshold and 1 ms in duration. Each patient was paced at a basic cycle length from either the right or left ventricles and progressively premature ventricular extrastimuli were introduced at 10–20 ms intervals until ventricular refractoriness was reached. In 11 patients these studies were performed at two or more cycle lengths. All data were relayed through matching amplifiers and viewed on a switch beam oscilloscope (Electronics and Medicine, Inc., White Plains, N. Y.), and simultaneously recorded on a 14-channel Honeywell tape recorder (Honeywell, Inc., Minneapolis, Minn.). The data were later reproduced on photographic paper at speeds of 150–200 mm/s. Care was taken to assure that all electronic equipment was adequately grounded.

Definition of terms. S_1, V_1, H_1, A_1 represented stimulus artifact, ventricular, His-bundle, and atrial electrograms of the basic drive beats, respectively.

S_2, V_2, H_2, A_2 represented the stimulus artifact, ventricular, His-bundle, and atrial electrogram of the premature beats, respectively.

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[1] *Abbreviations used in this paper*: A-V, atrio-ventricular; BCL, basic drive cycle length; ERP-V, effective refractory period of the ventricle.

H_1 was obscured by the ventricular electrogram (V_1) during pacing in most patients and could only be identified in four cases. Based on previous experimental data (1) the S_1H_1 interval was assumed to be constant in those patients in whom H_1 was not visible. Thus, S_1H_2, a combination of a fixed S_1H_1 and S_2H_2, was used as an approximation of the minimum retrograde His-Purkinje response to two consecutive beats whenever H_1H_2 was not recorded.

The effective refractory period of the ventricle was defined as the longest S_1S_2 that failed to evoke V_2.

The retrograde effective refractory period of the His-Purkinje system was taken as the longest V_1V_2 at which V_2 blocks within the His-Purkinje system. Determination of this parameter requires identification of H_2 before the occurrence of block.

Prolongation of intraventricular (muscle) conduction was evaluated using S_2V_2 from either right (21 patients) or left (2 patients) ventricular electrograms or from the ventricular electrogram in the His-bundle recording (28 patients).

The antegrade functional refractory period of the His-Purkinje system is defined as the shortest V_1V_2 resulting from any H_1H_2.

The retrograde functional refractory period of the His-Purkinje system is defined as the shortest S_1H_2 or H_1H_2 resulting from any S_1S_2 or V_1V_2.

RESULTS

In 28 of 40 patients, an H_2 deflection was recorded thereby allowing retrograde His-Purkinje conduction to be analyzed. These 28 patients form the basis of this report (Table I). 11 of the 28 had no clinical cardiac disease, whereas the remaining 17 had a variety of cardiac disorders of which atherosclerotic heart disease was most common (6 patients). The 12 patients in whom no retrograde H_2 was seen were similar in age, sex, and cardiac diagnosis to the remaining 28 patients; however, there was a higher percentage of bundle branch block and (or) H-V prolongation (8/12 vs. 11/28).

In 24 of the 28 patients, H_2 was not observed until a critically short S_1S_2 interval was reached. Further decrease in S_1S_2 intervals resulted in progressive lengthening of S_2H_2 (Fig. 1). The increase in S_2H_2 intervals assumed an inverse linear relationship to the decreasing S_1S_2 intervals (Fig. 2). The slope of progressive S_2H_2 prolongation, which was calculated by measuring the change in S_2H_2 per 10-ms decrement in S_1S_2 ($\Delta S_2H_2/\Delta S_1S_2$ in Table I), was virtually unchanged at all coupling intervals. Inasmuch as, in most patients (22/28), the increments in S_2H_2 approximated the decrements in S_1S_2 (slope 0.8–1.2), a relatively fixed S_1H_2 interval resulted in these cases (Fig. 3).

In the four patients in whom H_1 (i.e., retrograde His deflection during ventricular pacing) was observed, no change in S_2H_2 was noted at long coupling intervals. Further shortening of S_1S_2 resulted in gradual prolongation of S_2H_2 with H_2 becoming "buried" within the ventricular electrogram before its reappearance after V_2 at very close coupling intervals (Fig. 4). Once H_2 had appeared after V_2, further prolongation of S_2H_2 appeared linear as in the other cases. As anticipated, the S_1H_2 curves in these cases showed an initial decreasing slope followed by a flattening out of the curve. Thus, in these four cases the curves appeared similar to antegrade A-V nodal refractory curves, but all of His-Purkinje conduction (that portion of the curve obscured by the ventricular electrogram) could not be analyzed.

Cycle length responsiveness of retrograde His-Purkinje delay. In 11 patients ventricular stimulation was performed at two or more cycle lengths. In each case the S_2H_2 delay was less at any given S_1S_2 at shorter drive cycle lengths (Fig. 5). The slopes of S_2H_2 increments were similar at the different drive cycle lengths resulting in parallel S_2H_2 and S_1H_2 vs. S_1S_2 curves (Fig. 5 and Fig. 6).

Relationship of intraventricular conduction delay to S_2H_2 prolongation. The onset and degree of S_2H_2 prolongation was unrelated to intraventricular conduction delay (S_2V_2) as recorded in local right (21 patients) and (or) left (2 patients) ventricular electrograms or in the ventricular electrogram in the His bundle recording (28 patients). Intraventricular conduction delay (latency) was noted in 22 patients but only occurred after marked S_2H_2 prolongation had already appeared (Fig. 1). The degree of intraventricular conduction delay ranged from 5 to 40 ms and was always less than the increment of S_2H_2 prolongation at any coupling interval (Fig. 1). Furthermore, the presence of latency did not alter the slope of S_2H_2.

In two patients both right and left ventricular electrograms were simultaneously recorded during ventricular stimulation. In each case the electrogram from the unstimulated ventricle appeared before the inscription of the retrograde His-bundle deflection at all S_1S_2 intervals (Fig. 4). Therefore, biventricular depolarization occurred irrespective of the presence or degree of S_2H_2 delay. Thus, transeptal conduction was faster than conduction over the His-Purkinje system.

Ventricular and retrograde His-Purkinje refractoriness. In 24 of 28 patients, H_2 was present at the shortest S_1S_2 resulting in ventricular depolarization. Thus, the effective refractory period of the ventricle was greater than or equal to the retrograde effective refractory period of the His-Purkinje system. In four cases the retrograde effective refractory period of the His-Purkinje system exceeded the effective refractory period of the ventricle. Only one patient demonstrated a retrograde gap (2).

Comparison of the antegrade and retrograde functional refractory periods of the His-Purkinje system could be made in 19 patients (Table I). The retrograde functional refractory period (S_1H_2 or H_1H_2) exceeded antegrade functional refractory period in 16

TABLE I
Patient Data

Patient	Age	Sex	ECG	Cardiac diagnosis	AH	HV	BCL (S_1S_1)	Range of S_1S_2 producing S_2H_2	Range of S_2H_2	$\frac{\Delta S_2H_2}{\Delta S_1S_2}$	Retrograde ERP of HPS	Retrograde FRP of HPS	Antegrade FRP of HPS	ERP-V	VA conduction
1	18	M	WPW B	NHD	110	50	600	320–275	140–240	2.2	275	460	460	195	+
2	69	F	LVH	HCVD	85	65	550	350–220	130–240	0.8	—	470		210	+
3	70	M	AF	ASHD	—	55	600	400–220	112–250	0.8	220	500	—	200	−
4	68	F	RBBB LAH 1°AVB	NHD	125	80	600	400–260	135–295	1.1	250	520	430	200	−
5	62	M	IVCD 1°AVB LAE	CM	90	115	600	390–310	160–235	0.8	300	535		250	−
6	54	F	WNL	NHD	80	50	600	400–290	140–180	0.4	—	465		280	+
7	45	M	LBBB LAE	ASHD	82	68	600	340–220	145–275	1.1	—	480	380	210	−
8	56	M	RBBB	HCVD	65	50	600	600–230*	90–280	0.5	—	500	390	220	+
9	62	M	WNL	NHD	75	55	600	350–210	110–270	1.1	—	450	400	200	+
10	43	M	WNL	Sarcoidosis	100	50	650	650–230*	45–210	0.4	—	440	540	220	+
11	30	F	WNL	NHD	72	45	600	300–220	138–215	1.0	—	435	360	210	+
12	62	M	WNL	ASHD	85	45	600	280–190	200–310	1.2	—	475	445	180	−
13	54	M	RBBB LPH	AS	114	78	600	390–230	166–328	1.0	—	555	510	220	−
14	53	F	A-V Dis SB JR	SSS	—	48	550	380–240	150–270	0.8	—	490	—	230	+
15	67	F	IMI	ASHD	85	45	600	280–230	180–230	1.0	—	460	—	220	+
16	72	M	IVCD	ASHD	75	50	600	330–230	175–260	0.9	—	470	440	220	−
17	57	M	LVH	HCVD	90	60	600	420–280	165–275	0.8	—	520	360	270	+
18	52	F	Short PR IACD SVT	NHD	50	45	600 800	320–240 380–260	155–235 150–270	1.0 1.0	— —	480 520	370 420	230 250	+ +
19	54	F	WNL	NHD	80	40	600 800	320–240 380–280	130–200 90–180	0.9 0.9	— —	410 460	— 510	230 270	− −
20	41	M	RBBB	ASHD	65	65	450 600	360–200 400–220	135–190 125–220	0.3 0.5	— —	385 430		180	+ +
21	49	M	RBBB LPH	CM	100	80	600 700 800	300–230 340–240 350–260	145–230 145–220 135–240	1.2 0.8 1.2	— — —	445 480 485	— 425 —	220 230 240	− − −
22	53	M	WNL	NHD	85	40	500 700	365–255 400–290	180–275 175–295	0.9 1.2	— —	520 570	—	250 280	+
23	18	M	3°AVB RBBB	Corrected Transpos. VSD, PS S/P surg.	—	35	800 1000	800–280* 1000–260*	30–265 30–310	0.4 0.4	280 260	500 520	— —	230 240	+ +
24	72	M	WNL	NHD	95	50	500RV 500LV 700RV 700LV	280–190 250–200 300–230 300–230	130–220 120–160 150–210 150–290	1.0 0.8 0.8 0.6	— — — —	395 365 400 400	345 — — —	180 200 220 220	− − − −
25	35	M	IVCD	Mitral prolapse	68	88	700 800	355–220 380–240	155–310 175–340	0.9 1.1	— —	530 540	450 —	210 200	+ +
26	18	M	WNL	NHD	85	42	600RV 600LV 760RV 700LV	340–250 600–250* 380–280 700–250*	150–246 60–240 155–305 60–290	1.1 0.4 1.5 0.4	— — — —	495 475 540 495	410 — — —	245 240 260 240	+ + + +
27	72	F	RBBB LAH	Amyloidosis	60	70	500 700	260–200 320–250	180–225 170–220	0.8 0.7	— —	425 470	— 350	190 240	+ +
28	19	M	WNL	NHD	75	55	550 750	380–240 420–290	110–220 140–260	0.7 0.9	— —	450 530	— 460	230 295	+ +

Abbreviations used in this table: AF, atrial fibrillation; AS, aortic stenosis; ASHD, atherosclerotic heart disease; A-V Dis, atrio-ventricular dissociation; AVB, atrioventricular block; CM, cardiomyopathy; ERP, effective refractory period; ERP-V, effective refractory period of the ventricle; FRP, functional refractory period; HCVD, hypertensive cardiovascular disease; HPS, His-Purkinje system; IACD, intra-atrial conduction defect; IMI, inferior myocardial infarction; IVCD, intra-ventricular conduction defect; LAE, left atrial enlargement; LAH, left anterior hemiblock; LBBB, left bundle branch block; LPH, left posterior hemiblock; LVH, left ventricular hypertrophy; NHD, no heart disease; PS, pulmonic stenosis; RBBB, right bundle branch block; SB, sinus bradycardia; SSS, sick sinus syndrome; SVT, supraventricular tachycardia; VA, ventriculo-atrial; VSD, ventricular septal defect; WNL, within normal limits; WPW, Wolff-Parkinson-White.
* Retrograde His deflection (H_1).

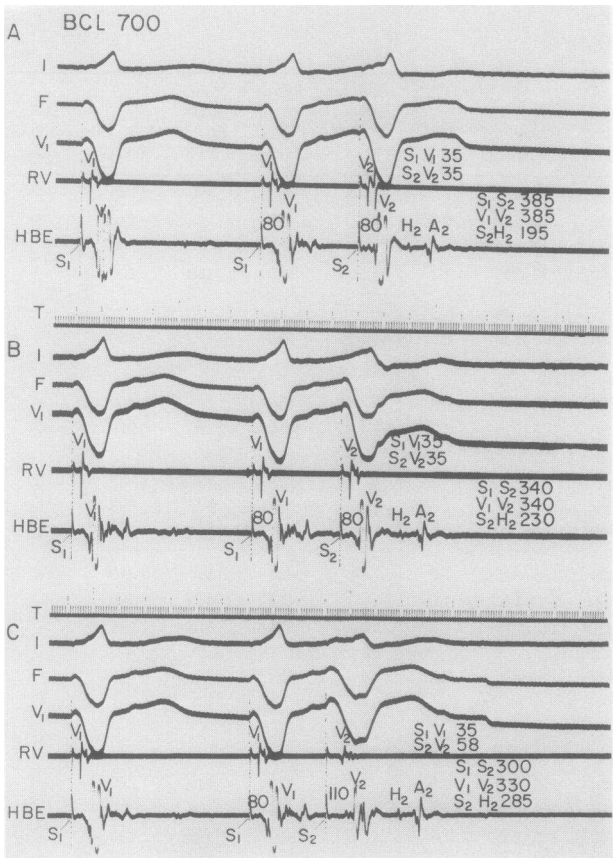

FIGURE 1 Effect of ventricular extrastimuli on His-Purkinje conduction. The three panels are each organized from top to bottom as follows: surface ECG leads I, avF (F), V_1, right ventricular (RV) and His bundle electrograms (HBE), and time lines (T). The right ventricular electrogram is being paced (S_1S_2) at a basic drive cycle length (BCL) of 700 ms. Premature ventricular extrastimuli (S_2) are delivered at progressively closer coupling intervals (S_1S_2) in panels A–C resulting in gradual prolongation of His-Purkinje conduction time (S_2H_2). Note that S_2H_2 prolongation occurs in the absence of local ventricular muscle delay ($S_1V_1 = S_2V_2$) in panels A and B. In panel C the presence of local ventricular delay in the right ventricular and His bundle electrograms of 23 and 30 ms does not entirely account for continued S_2H_2 delay of 55 ms.

patients; in 2 patients minimum antegrade output was greater; and in 1 patient they were identical.

Relationship of antegrade and retrograde conduction. The degree of S_2H_2 prolongation showed no consistent relationship to the presence of intraventricular conduction defects, H-V intervals, or the presence or absence of ventriculo-atrial conduction (Table I).

DISCUSSION

Our data in 28 patients suggest that the normal response of the His-Purkinje system to ventricular premature stimulation is gradual conduction delay recog-

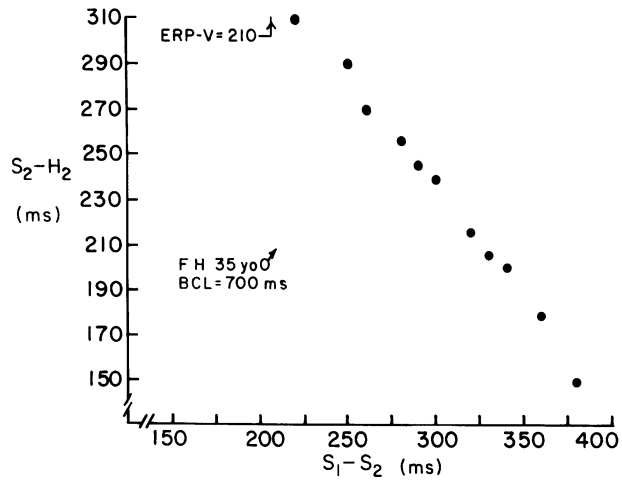

FIGURE 2 Relationship of coupling interval of ventricular extrastimuli to the degree of slowing of His-Purkinje conduction. The coupling interval of ventricular extrastimuli (S_1S_2) is plotted against resultant His-Purkinje conduction time (S_2H_2) during a BCL of 700 ms. There is an inverse linear relationship of S_2H_2 to S_1S_2 with the increment in S_2H_2 approximating the decrement of S_1S_2. ERP-V, effective refractory period of the ventricle.

nized by S_2H_2 prolongation. Progressive increase of S_2H_2 occurred as the coupling intervals of the premature stimuli were decreased. The amount of this delay was variable ranging from 30 to 330 ms. The appearance of S_2H_2 prolongation was noted in 80% of our patients and appears less common in patients with bundle branch block and (or) H-V prolongation; significant infranodal conduction disturbances were noted in 8/12 patients in whom no retrograde H_2 deflection was noted and in only 11/28 patients manifesting a retrograde H_2 during stimulation.

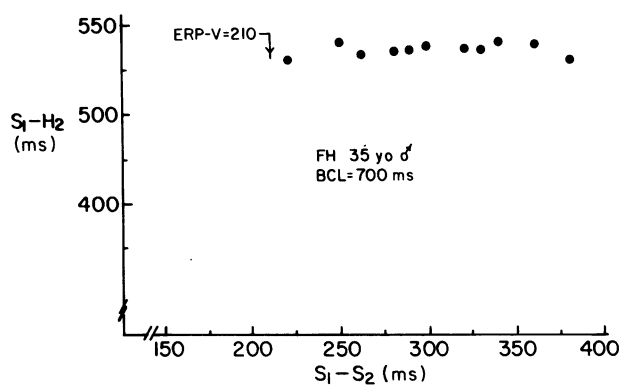

FIGURE 3 Relationship of minimal His-Purkinje output to coupling interval of ventricular extrastimuli. The coupling interval of ventricular extrastimuli (S_1S_2) is plotted on the abscissa and the minimal His-Purkinje response (S_1H_2) on the ordinate. Because the increment of H_2H_2 equaled the decrement of S_1S_2 (Fig. 2) the resultant S_1H_2 remained fixed, producing a flat curve.

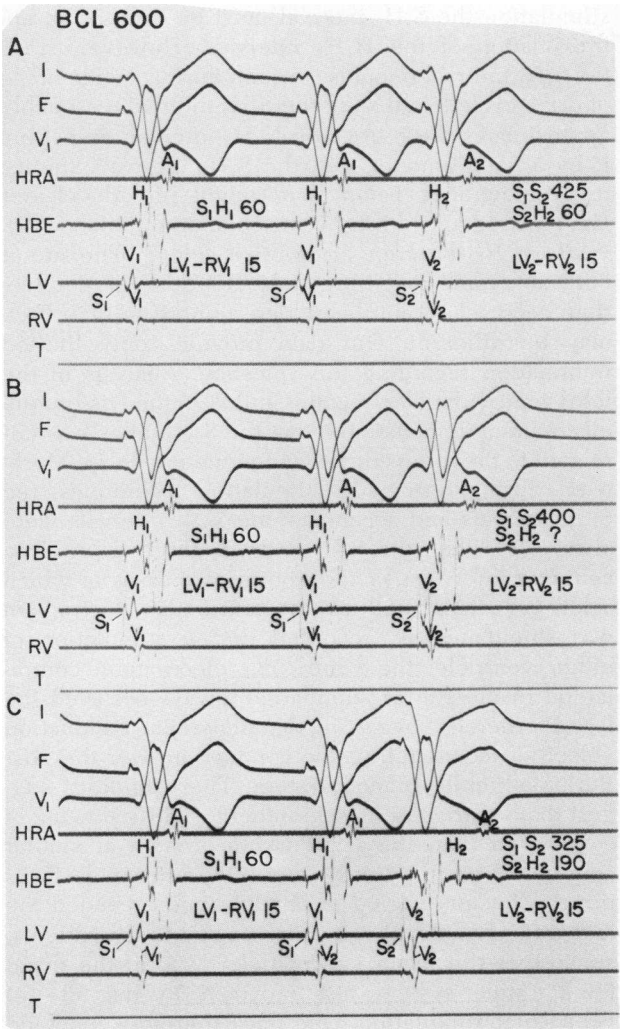

FIGURE 4 Site of His-Purkinje conduction delay during premature stimulation. The three panels are each organized from top to bottom as follows: surface leads I, avF (F), and V_1, a high right atrial electrogram (HRA), His bundle electrogram (HBE), left ventricular electrogram (LV), right ventricular electrogram (RV), and time lines (T). The left ventricular electrogram is being paced (S_1S_1) at a BCL of 600 ms. Note that a retrograde His deflection (H_1) can be seen during the basic drive beats, and that retrograde His-Purkinje conduction during these beats (S_1H_1 = 60 ms) exceeds local ventricular and transseptal conduction time (LV_1–RV_1 = 15 ms). In panels A–C progressively premature ventricular extra-stimuli (S_2) are introduced. In panel A at a coupling interval (S_1S_2) of 425 ms, no retrograde His-Purkinje delay (S_2H_2) is seen. At closer coupling intervals (panels B and C) S_2H_2 prolongs without concomittant local ventricular conduction delay.

The response of the His-Purkinje system to premature ventricular stimulation was cycle length dependent; at any given S_1S_2 interval, the resulting S_2H_2 interval was less at shorter drive cycle lengths. Once H_2 was seen, the pattern of S_2H_2 prolongation appeared linear at all cycle lengths studied giving

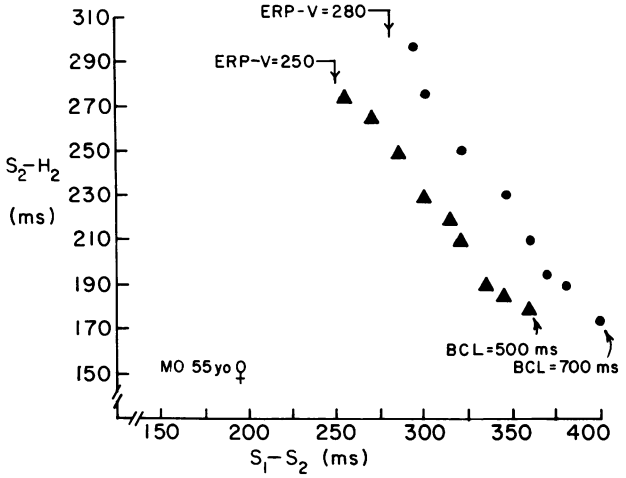

FIGURE 5 Cycle length responsiveness of retrograde His-Purkinje conduction delay. This figure is organized as in Fig. 2. Retrograde His-Purkinje conduction (S_2H_2) in response to variably coupled ventricular extrastimuli (S_1S_2) is compared at two BCL; 700 ms (●) and 500 ms (▲). At every S_1S_2 the resultant S_2H_2 is longer at a BCL of 700 ms. The ERP-V is also longer at a BCL of 700 ms. The slopes of S_2H_2 are similar at both cycle lengths resulting in parallel curves.

rise to fixed slopes ($\Delta S_2H_2/\Delta S_1S_2$). The amount of S_2H_2 prolongation bore a direct relationship to the decrement in S_1S_2 intervals resulting in flat S_1H_2 curves in 80% of patients.

In the four patients in whom a His deflection was seen at the basic drive cycle length (H_1), the S_2H_2 slowly increased prior to disappearing on the QRS; subsequent S_2H_2 prolongation was linear. In these four patients the S_1H_2 curves progressively decreased before they flattened out at closer S_1S_2 intervals. This

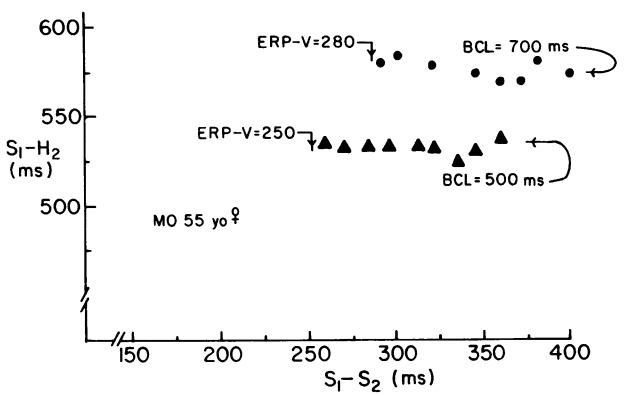

FIGURE 6 Effect of cycle length on minimal His-Purkinje output. This figure is organized as in Fig. 3. The studies were carried out at a BCL of 700 ms (●) and 500 ms (▲). Inasmuch as the slope of retrograde His-Purkinje delay are parallel at different cycle lengths (Fig. 5), the curves of resultant minimal outputs (S_1H_2) are also parallel. The shorter the BCL the less the minimal output.

pattern is similar to antegrade A-V nodal refractory curves. It is likely that this would have been the usual pattern, if H_1 were noted in all patients and the entire refractory curve could be plotted. Thus, while the curve of S_2H_2 increase appeared linear, it may have been an artifact of the technique which allowed observation of only a small portion of the refractory curve.

Prior investigations. Previous studies in both animals and humans have demonstrated that the His-Purkinje system does not behave in an "all-or-nothing" fashion but undergoes progressive slowing of conduction, in a similar fashion to the A-V node (1–7). In fact, several clinical observations of infra-nodal Wenckebach have been noted in man (1). The ability to demonstrate such slow conduction in the His-Purkinje system in man during antegrade stimulation has been limited by the refractoriness of the A-V node and (or) the atrium. Refractoriness in these tissues limits the prematurity at which atrial impulses can reach the His-Purkinje system, thereby preventing adequate stressing of the infra-nodal structures. In the animal laboratory, where direct His-bundle stimulation and recording can be performed more readily, slowing of His-Purkinje conduction is routinely observed (2). Prior work in man (3–7) has demonstrated the occurrence of V-H prolongation in response to coupled ventricular stimulation; however, neither the degree nor the pattern of this His-Purkinje conduction delay has been analyzed. However, a linear increase in S_2H_2, after H_2 appears beyond the QRS, has been noted (3). Evaluation of retrograde refractoriness by Akhtar et al. (3) demonstrated no consistency in the relationship between antegrade and retrograde His-Purkinje refractoriness, although exact comparisons could not be made due to the limitations of the technique (see below).

Limitations of the technique. Although ventricular stimulation allows one to readily observe the behavior of the His-Purkinje system in response to premature stimuli, it is clear that one cannot compare responses to ventricular stimulation with those produced by comparable antegrade stimulation. Inasmuch as the His deflection is usually hidden within the QRS during ventricular pacing (3, 6), not even basal V-H and H-V intervals can be compared. Even in the few patients in whom a retrograde His deflection can be seen during ventricular pacing, the site at which the stimulated impulse enters the His-Purkinje system during retrograde conduction is different than the site of exit during antegrade studies. Thus, even in the presence of basal S_1H_1 intervals, comparisons of antegrade and retrograde infra-nodal conduction are not meaningful. It is equally impossible to accurately compare antegrade and retrograde refractoriness of the His-Purkinje system because of the requirement for the observation of H_1H_2 intervals. During retrograde stimulation the S_1H_2 interval must be used as an approximation of the H_1H_2 intervals. However, since the S_1H_1 interval remains constant during a wide range of drive cycle lengths (2), the S_1H_2 interval reasonably approximates the retrograde H_1H_2 interval, exceeding it by a fixed amount, i.e., the S_1H_1 interval. Studies using electrodes 1-mm apart might provide clearer His electrograms during ventricular stimulation (4, 7).

Site of His-Purkinje conduction delay. The site of both antegrade and retrograde His-Purkinje conduction delays has not been determined and, in fact, may be different. Our data provides only limited information regarding this question. Analysis of the local ventricular electrograms and V_2 in the His-bundle electrogram demonstrated that the S_2H_2 delay was unrelated to the intraventricular conduction delay. Moreover, during retrograde stimulation techniques, the S_1S_2 intervals and accompanying V_1V_2 intervals were always less than the S_1H_2 or H_1H_2 intervals at close coupling intervals. In addition, in two cases in which a left ventricular and a right ventricular electrogram was simultaneously recorded during stimulation of either ventricle, the ventricular electrogram contralateral to the site of stimulation always occurred before H_2, thereby suggesting that transseptal conduction velocity exceeds retrograde conduction over the His-Purkinje-bundle branch system. These findings suggest that ventricular muscle undergoes less slowing of conduction than the His-Purkinje system at similar coupling intervals. Furthermore, the retrograde functional refractory period of His-Purkinje exceeded the antegrade functional refractory period in 16 patients, suggesting the major site of delay was in the distal His-Purkinje system just proximal to the site of ventricular stimulation. One must therefore conclude that the site of S_2H_2 delay must be between the Purkinje-myocardial junction and the His bundle itself.

Prior work in animals using microelectrode techniques has demonstrated that the most distal regions of the His-Purkinje system have the longest refractory periods (8–11). This area of maximum refractoriness has been termed the "distal gate" and is located just proximal to the Purkinje-myocardial junction. It seems logical, therefore, that the site of S_2H_2 delay noted during ventricular stimulation occurs in this region. Such confinement of these delays to the gate during retrograde stumulation has been demonstrated in animal preparations (8–11). Proof of this hypothesis in man would require simultaneously recorded electrograms along the bundle branches and the His bundle.

Conclusions. The technique of ventricular stimulation to assess His-Purkinje function provides a look at His-Purkinje properties different from that provided by antegrade techniques. Using retrograde stimula-

tion techniques, it is apparent that the His-Purkinje system does not function as an "all-or-nothing" cable but consistently demonstrates slowing of conduction. While this is similar to the response of the A-V node under comparable stress, the cycle length responsiveness of the His-Purkinje system is markedly different from that of the A-V node. At faster drive cycle lengths, at which the effective refractory period and conduction time of the A-V node prolongs, retrograde His-Purkinje conduction and refractoriness shortens (12). While exact comparisons cannot be made between antegrade and retrograde studies, retrograde studies might provide useful information concerning the functional status of the distal His-Purkinje system. Such data might provide important predictive information in patients with bifascicular block and long H-V intervals in whom one is trying to assess the relative risks of heart block. Further studies are needed to confirm the utility of this technique in that endeavor. This technique may also be used to evaluate the relationship of distal His-Purkinje and ventricular conduction delays to the propensity for the development of ventricular tachyarrhythmias, an area currently under investigation in our laboratory.

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