Delay and block of cardiac impulse caused by enhanced phase-4 depolarization in the His-Purkinje system

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The underlying mechanism of bradycardia-dependent bundle-branch and paroxysmal atrioventricular block appears to be enhancement of phase-4 depolarization in a branch or in a natural or acquired monofascicular pathway.

Clinical records of these forms of impaired conduction occurring in the bundle-branches, with either longer or shorter cardiac cycle lengths, are presented and analysed. These also include the combination of Mobitz type I atrioventricular block with variable degrees of bundle-branch block, as a representative example of narrow ventricular escape beats firing in the zone where prominent diastolic depolarization is present.

Spontaneous depolarization during phase-4 is the electrophysiological mechanism which enables the automatic cells to be effective or latent pacemakers (West, 1955a, b). Occasionally, under certain pathological conditions, the ventricular specialized conduction system may exhibit prominent diastolic depolarization (Weidmann, 1955; Singer, Lazzara, and Hoffman, 1967; Wennemark and Ruesta, 1971), the mechanism underlying bradycardia-dependent bundle-branch block (Massumi, 1968; Elizari *et al.*, 1968; Sarachek, 1970; Schamroth and Lewis, 1971; El-Sherif, 1972; Rosenbaum *et al.*, 1973b; Barold and Schamroth, 1973). The development of atrioventricular block due to enhanced phase-4 depolarization (Singer *et al.*, 1967) occurs either in the His bundle, or in a branch or division of the conducting system when the remaining routes are blocked.

Association of enhanced automaticity and impaired conductivity owing to the development of generalized diastolic depolarization in the higher parts of the main intraventricular conduction fascicles, may be clinically exemplified by narrow ventricular ectopic beats located in the zone showing conduction delays.

This paper presents electrocardiograms of patients

with bradycardia-dependent block, which can be explained in terms of phase-4 depolarization, and discussions on the concepts related to this electrophysiological mechanism based upon the corresponding records.

Subjects and methods

The group consisted of 11 patients, of whom 6 had bradycardia-dependent bundle-branch block (Table 1), and 5 had bradycardia-dependent paroxysmal atrioventricular block (Table 2). The main clinical and electrocardiographic features are summarized in these Tables. All electrocardiograms were recorded with a Sanborn Model 296 direct writing electrocardiograph at a paper speed of 25 mm/second. Vectorcardiograms in the three-plane projections were recorded in the supine position by the Frank system, with a Hewlett-Packard 1520-A machine, using the fourth intercostal space.

Bipolar recordings of the bundle of His were obtained as previously described (Scherlag *et al.*, 1969), with a 4F bipolar pacing catheter. Each bipolar lead was connected to the AC inputs of a multichannel oscilloscopic photographic recorder, with filter frequency set at 40 and 200 cycles/sec. Records were taken at 100 mm/sec.

All analysed beats were of sinoatrial origin. RR intervals were measured to determine the ranges of normal and aberrant intraventricular conduction in the bradycardia-dependent bundle-branch block. The same measurements were performed with the RP intervals in bradycardia-dependent paroxysmal atrioventricular block. In several cases, slowing of the heart rate was achieved by carotid sinus pressure.

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TABLE 1 Bradycardia-dependent bundle-branch block

Case No.	Age (yr)	Sex	Diagnosis	Intraventricular conduction aberrancy	Conduction ranges in bundle		
					Phase-3 block	Normal conduction	Phase 4-block
1	47	M	Coronary heart disease	Left bundle-branch block	0.84-1.40	1.22-2.42	2.02-5.80
2	45	M	Aortic disease	Left bundle-branch block	—	0.75-1.18	1.16-1.30
3	61	F	Coronary heart disease	Left bundle-branch block	0.62-0.80	0.70-1.42	1.34-3.56
4	64	M	Coronary heart disease	Right bundle-branch block	0.54-1.20	1.18-2.04	1.84-4.08
5	58	M	Coronary heart disease	Left bundle-branch block	0.52-0.84	0.74-1.28	1.20-8.02
6	53	M	Coronary heart disease	Right bundle-branch block	0.58-0.64	—	0.84-1.24

The conduction ranges in bundle are expressed throughout as RR intervals in seconds.

TABLE 2 Bradycardia-dependent atrioventricular block

Case No.	Age	Sex	Intraventricular aberrancy	Diagnosis	Intervals (ms)			AV conduction ranges		
					AH	HV	PR	Early AV block zone	AV cond.	Tardy AV block zone
1	29	M	Right bundle-branch block + left posterior hemiblock	Cardiomyopathy	—	—	0.24	0.16-0.44	0.36-1.30	1.22-1.96
2	78	M	Right bundle-branch block + left anterior hemiblock	Idiopathic chronic AV block	110	80	0.19	0.20-0.50	0.54-1.08	1.11-2.88
3	64	F	Right bundle-branch block + left posterior hemiblock	Coronary heart disease	100	80	0.18	0.24-0.34	0.32-0.64	0.56-1.14
4	80	M	Right bundle-branch block + left anterior hemiblock	Idiopathic chronic AV block	—	—	0.22	0.23-0.34	0.32-0.58	0.56-1.04
5	65	M	Left bundle-branch block	Coronary heart disease	—	—	0.22	0.18-0.33	0.30-0.76	0.72-1.09

Atrioventricular conduction ranges are expressed throughout as RP intervals in seconds.

Case reports

Intermittent rate-dependent bundle-branch block

Case 1 Electrocardiographic and vectorcardiographic study (Fig. 1A), performed during first admission to hospital of a 49-year-old patient with a two-year history of angina pectoris, indicated spontaneous intermittent left bundle-branch block and antero-septal ischaemia. A long tracing (Fig. 2), performed a week later during which vagal manoeuvres were carried out (Comeau, Hamilton, and White, 1938; Dressler, 1959), showed left bundle-branch block at the shortest and longest diastolic intervals; at an intermediate range the intraventricular conduction was normal. Three conduction ranges were observed: the first, with RR intervals between 0.84 and 1.40 sec and left bundle-branch block aberrancy; the second, between 1.22 and 2.42 sec with normal intraventricular conduction; and the third, in

which paradoxically the left bundle-branch block reappeared between 2.02 and 5.80 sec. In the graph (Fig. 1B) obtained from the above-mentioned study, representing 954 beats of sinoatrial origin, the three conduction areas previously described are delimited: a) phase-3 left bundle-branch block (beats arriving at the left bundle-branch during its absolute or relative refractory period (Katz and Pick, 1956)); b) normal intraventricular conduction; and c) phase-4 left bundle-branch block (impulses reaching fibres of the left bundle-branch with reduced levels of membrane potential caused by spontaneous diastolic depolarization).

Sinus impulses with left bundle-branch block aberrancy, which overlap with the beats corresponding to both ends of the normal conduction zone, should be noted (Rosenbaum *et al.*, 1973a; Garcia and Rosenbaum, 1972).

Two months later when the patient was readmitted

because of increased symptoms, left bundle-branch block was registered which was not modified by changes in cardiac frequency. This electrocardiographic pattern remained unchanged in follow-up examinations performed during the next year and a half.

This case typifies a bradycardia-dependent left bundle-branch block which has the following outstanding features: 1) intraventricular aberrancy occurring

simultaneously with pronounced prolongation of the cardiac cycle length; and 2) transitory stage of the phenomenon of bradycardia-dependent left bundle-branch block.

Case 2 A 45-year-old patient with aortic valve disease, cardiac failure, and syncopal attacks: basic tracing (Fig. 3) shows a sinus rhythm with first degree atrioventricular

FIG. 1 Panel A: *Electrocardiogram and vectorcardiogram with intermittent left bundle-branch block. In each lead the first beat is without, and the second with, left bundle-branch block. Inverted T waves in leads V1 to V4 are observed in the normal conducted beats.* Panel B: *Graphic representation of 954 conducted beats with normal intraventricular conduction, and left bundle-branch block aberrancy according to their corresponding RR intervals. Time intervals are depicted in hundredths of seconds. NC, normal conduction; phase-3 LBBB, phase-3 left bundle-branch block; phase-4 LBBB, phase-4 left bundle-branch block; RR, RR intervals.*

FIG. 2 A to E, discontinuous record of lead I. Tracings of conducted sinus beats presenting normal and aberrant QRS complexes with left bundle-branch block pattern according to previous cycle length. The distribution of the mentioned beats are schematically represented at the bottom of this figure. See text for further explanation. NC, normal conduction; PH-3 LBBB, phase-3 left bundle-branch block; PH-4 LBBB, phase-4 left bundle-branch block; RR, RR intervals.

block (PR 0.28 sec) left ventricular hypertrophy, and incomplete left bundle-branch block. An electrocardiogram performed two days later (Fig. 4) shows, in strips A, B, and C, conducted beats preceded by identical P waves with constant PR intervals and different degrees of left bundle-branch block starting from an RR interval of 1.16 sec. The variability of conduction disturbance is directly related to the previous diastolic pause; greater degrees of left bundle-branch block aberrancy are preceded by longer RR intervals.

In strip D an increase in the sinus frequency establishes a 2:1 atrioventricular block and the conducted beats also present variable degrees of left bundle-branch block.

A new record obtained three days later (Fig. 5A) showed different degrees of bradycardia-dependent left bundle-branch block and the appearance of narrow ventricular escape beats with pure incomplete right bundle-branch block pattern starting from an RR interval of

1.30 sec. In addition, atrioventricular dissociation with a similar ectopic rhythm firing at a rate of about 50/min was recorded during the same study (Fig. 5C). Two months later, while the patient was receiving digitalis (Fig. 6, strip A), prolongation of first degree atrioventricular block up to 0.32 sec was noticed. In strip B, an increment of the sinus frequency causes a second degree atrioventricular block with a 3:2 and 4:3 Luciani-Wenckebach ratio. The fluctuations in diastolic pauses originated by the dropped P waves determine the appearance of variable degrees of left bundle-branch block in the beats, which reinitiate the cycle maintaining a constant PR interval of 0.32 sec. In strip C a further increase in the sinus frequency establishes a 2:1 atrioventricular block with similar characteristics to the one observed in Fig. 4, strip D. During this study a vectorcardiogram was performed simultaneously, registering beats of sinoatrial origin with progressive left bundle-branch block aberrancy.

FIG. 3 Electrovectorcardiogram showing sinus rhythm, left ventricular hypertrophy, and incomplete left bundle-branch block with first degree atrioventricular block.

FIG. 4 Electrocardiogram (discontinuous strips of standard lead I) showing in A, B, C, and D, variable degrees of left bundle-branch block aberrancy related to previous cycle length. See text for further explanation.

FIG. 5 Panel A: Simultaneous recording of leads I and V1. Variable degrees of bradycardia-dependent left bundle-branch block developed during induced slowing of the heart rate (carotid sinus pressure). Appearance of narrow ventricular escape beats exhibiting pure incomplete right bundle-branch block aberrancy, preceded by diastolic intervals exceeding 1.28 sec. Panel B: Twelve lead electrocardiogram of narrow ventricular escape beats with pure incomplete right bundle-branch block pattern. Panel C: Atrioventricular dissociation. Narrow ventricular ectopic rhythm with pure incomplete right bundle-branch block configuration discharging at a rate of about 50 beats/min. (RR intervals are indicated in hundredths of seconds.)

This case presents the following outstanding features: 1) variable degrees of aberrant intraventricular conduction, taking place conjointly with slight increases in cardiac cycle length; 2) coexistence of Luciani-Wenckebach phenomenon with variable degrees of bradycardia-dependent left bundle-branch block; 3) differing degrees of phase-4 left bundle-branch block, associated with narrow ventricular ectopic beats exhibiting pure incomplete right bundle-branch block configuration; 4) inability to obtain phase 3 left bundle-branch block (tachycardia-dependent left bundle-branch block) because of the development of 2:1 atrioventricular block when heart rate is increased.

Rate-dependent atrioventricular block

Case 1 A 27-year-old man with Chagasic cardiomyopathy had sinus arrhythmia and advanced atrioventricular block, requiring a permanent demand type pacemaker. During his stay in hospital paroxysmal episodes of atrioventricular block were observed (Fig. 7), which alternated with complete atrioventricular block and 1:1 atrioventricular conduction. The electrocardiogram (Fig. 8A) clearly reflects disturbances of the intraventricular conduction characterized by a PR interval of 0.24 sec and right bundle-branch block, with block in the

posterior inferior division of the left bundle-branch (left posterior hemiblock). In a long tracing recorded during a series of syncopal attacks (Fig. 7A), it may be observed that the first conducted sinus impulse (1st) is followed by an episode of sinus depression which initiated a period of ventricular asystole, interrupted by a notched P wave (2nd) representing a blocked atrial beat with an RP interval of 1.34 sec. The following P wave (3rd) is also blocked, thus resulting in a diastolic pause which is terminated by a ventricular escape beat. The next sinus impulse (4th) which follows the escape beat is conducted to the ventricles with an RP interval of 0.50 sec, the succeeding two atrial beats (5th to 6th) also being conducted. Then a new episode of sinus depression produces a similar sequence which is repeated in strips A and B. Periods of ventricular asystole varying in duration from 2.55 to 8.20 sec, provoked either by spontaneous sinus depression (strips A and B), or premature artificial stimulus (strip C), can be observed all through this figure.

To establish the range of propagation and blockage of atrial impulses in the atrioventricular conduction system, 1224 beats were analysed (Fig. 8B). The immediate post-escape conduction was tabulated independently and PP and PR intervals were also measured. Atrial post-escape impulses (Fig. 8B above dotted line)

FIG. 6 Three strips of lead I selected from continuous record. A) Sinoatrial beats with first degree atrioventricular block. B) Variable degrees of bradycardia-dependent left bundle-branch block in the first sequence beat of Luciani-Wenckebach period. C) 2:1 atrioventricular block with different degrees of left bundle-branch block aberrancy according to previous diastolic length. Bottom: Vectorcardiogram: horizontal plane. From right to left, different degrees of left bundle-branch block.

FIG. 7 Panels A and B: Short and longer runs of paroxysmal atrioventricular block all initiated by spontaneous sinus depression. Panel C: Same event induced by artificial premature atrial impulse (arrow). See text for further explanation.

present an early atrioventricular blocked zone; another one of conducted beats; and a third late atrioventricular blocked zone. The rest of the studied beats (Fig. 8B below dotted line) show an atrioventricular conduction zone followed by another one of blocked beats. In this group, early atrioventricular blocked zone is not observed, since spontaneous ventricular asystole was always initiated by depression of the sinus pacemaker.

B

FIG. 8 Panel A: Electrovectorcardiogram of right bundle-branch block with left posterior hemiblock and first degree atrioventricular block. Panel B: Graphic representation of RP intervals corresponding to 1224 conducted and non-conducted supraventricular impulses. Post-escape conducted and blocked beats are above the dotted line. Time intervals are depicted in hundredths of seconds. See text for detailed discussion. Abbreviations, A-V block, atrioventricular block; A-V cond, atrioventricular conduction; RP, RP intervals.

Analysing all the beats in the graph corresponding to Fig. 8B, the existence of a conduction zone and two blocked areas can be seen. Blocked atrial impulses overlapping the beats corresponding to both ends of the atrioventricular conduction zone may be observed. Sinus impulses (below dotted line) which occur after an RP interval longer than 1.20 sec or shorter than 0.40 sec, are blocked, and those (above dotted line) occurring with an RP interval between 0.40 sec and 1.30 sec after a ventricular escape beat, are conducted. Therefore, only atrial impulses which follow a ventricular escape beat or another supraventricular conducted beat within a limited period are transmitted to the ventricles.

This case exemplifies paroxysmal atrioventricular block; its more remarkable features being: 1) episodes of paroxysmal atrioventricular block originated by changes in atrial frequency; 2) a ventricular escape beat followed by sinus impulse with an adequate time relation to re-initiate the atrioventricular conduction; 3) transitoriness of paroxysmal atrioventricular block periods, alternating with 1:1 atrioventricular conduction and complete atrioventricular block.

Case 2 This 78-year-old patient had Adams-Stokes episodes before his paroxysmal atrioventricular block was recognized. These disappeared after the implantation of a pacemaker. Electrocardiogram and vectorcardiogram on admission (Fig. 9) showed right bundle-branch block with left anterior hemiblock, and a PR interval of 0.19 sec; it should also be stressed that a prolonged HV time of 80 msec was recorded.

Fig. 10, corresponding to episodes of paroxysmal atrioventricular block, indicates how the acceleration or decrease of atrial frequency gives rise to periods of ventricular asystole. Atrioventricular conduction is re-established when a P wave, occurring after the ventricular escape beat, and maintaining a critical RP interval with the latter, is conducted to the ventricles.

To determine the range of conduction or blockage in the atrioventricular conduction system, 628 atrial beats

were studied, delimiting: a) a blocking range between 0.20 and 0.50 sec; b) an atrioventricular conduction range from 0.54 to 1.08 sec, and again c) a blocking range between 1.11 and 2.88 sec.

This case presents the following features: 1) paroxysmal atrioventricular block caused by variations of the atrial frequency; 2) termination of the atrioventricular block episode conditioned by the appearance of a ventricular escape beat and its timing with the first

FIG. 9 Electrocardiogram and vectorcardiogram of right bundle-branch block with left anterior hemiblock and a PR interval of 0.19 sec. Bottom tracing: His bundle electrogram (HBE) recorded simultaneously with lead II.

FIG. 10 Short runs of paroxysmal AV block initiated (V1) by sinus depression and (V2) by a blocked atrial premature beat.

conducted P wave; 3) intraventricular trifascicular block, with a normal PR interval and a prolonged HV time (Narula *et al.*, 1971), indicating delayed conduction probably in the main left bundle or in its posterior division; 4) episodes of paroxysmal atrioventricular block interposed with 1:1 atrioventricular conduction and complete atrioventricular block.

Discussion

Impulse propagation in incompletely repolarized fibres of the His-Purkinje system caused by prolongation of the refractory period, or in fibres which develop phase-4 depolarization, may be affected so as to result in significant delay or block.

Under certain experimental and clinical pathological conditions: hypoxia and ischaemia (Trautwein and Dudel, 1956); increased stretch (Dudel and Trautwein, 1954; Singer *et al.*, 1967); and contusion of the intraventricular branching system (Kretz and Da Ruos, 1972), the cardiac cycle length variability constitutes an outstanding factor in the development of different forms of impaired conduction, making evident that both phase-3 and phase-4 block may be detected, differentiated, and their evolution determined by clinical (Masumi, 1968) and experimental electrocardiography. Therefore, pronounced decrease in cardiac frequency may determine paradoxical blockages of branches or divisions, owing to the development of phase-4 depolarization (Case 1, Fig. 2). Similarly, delay or interruption in the above-mentioned system may also be evidenced by slight prolongation of the cardiac cycle length (Case 2, Fig. 4) which has been attributed by Singer and Ten Eick (1971) to the probable combination of enhanced phase-4 depolarization and diminished membrane responsiveness.

Since the ascending slope of spontaneous diastolic depolarization is gradual, the appearance of conducted sinoatrial beats with varying degrees of bundle-branch block aberrancy developing with increases in cycle length, may be attributed to atrial impulses reaching the affected fascicle at different levels of diastolic depolarization. Such electrocardiographic derangements in the left bundle-branch (Fig. 4) were clinically recorded. The reproducibility of such phenomenon in the canine intraventricular bundle-branch system was also achieved in our laboratory (Kretz, Da Ruos, and Leguizamón Palumbo, 1974). This finding confirms the hypothesis that different degrees of decremental conduction obtained with a slow heart rate are produced by the following basic mechanism: fluctuations in level of transmembrane potential caused by variations of spontaneous

diastolic depolarization in the contusion affected cells of the corresponding fascicle. In almost all the cases presented in this paper (Table 1) clear evidence of prolonged recovery associated with spontaneous diastolic depolarization was recorded (Fig. 11), indicating that such electrophysiological mechanisms coexist, both at the level of the higher parts of the main intraventricular conduction fascicles and in their peripheral ramifications (Elizari, Lazzari, and Rosenbaum, 1973) as well.

The development of bradycardia-dependent block in the branching portion of the intraventricular conduction system, caused by prolongation of the cardiac cycle length caused by a Mobitz type I or II atrioventricular block, has been pointed out (von Hoesslin, 1923; Scherf and Scharf, 1948; Vesell and Lowen, 1963; Sherf and James, 1969; Sepúlveda, Rosselot, and Ahumada, 1969; Schamroth and Lewis, 1971; Friedberg, 1971; Mazzoleni and Fletcher, 1971; Gallagher *et al.*, 1973). Clinical evidence of this association is shown in Fig. 6, strip B, where Luciani-Wenckebach periods, with varying degrees of left bundle-branch block aberrancy in the first sequence beat, are observed (Friedberg, 1972). Likewise, the coexistence of phase-4 block with Mobitz type I form of decremental conduction has been reproduced in both divisions of the canine left bundle-branch (Kretz and Da Ruos, 1972).

Considering that recent electrophysiological studies (Bailey *et al.*, 1972; Myerburg, Nilsson, and Gelband, 1972) carried out on bundle-branch and Purkinje muscle preparations have indicated that spontaneous diastolic depolarization occurs within the bundle-branches, it can be expected that enhanced phase-4 depolarization confined to a group of automatic cells within the main left bundle-branch, might create an ectopic pacemaker (Hoffman, 1966; Drake, Skom, and Singer, 1971). Narrow ventricular ectopic beats arising from such anatomical origin were recorded in one of our patients (Case 2, Fig. 5). This interpretation is based on Rosenbaum's classification of ventricular extrasystoles (Rosenbaum, 1969). Our case according to QRS pattern and classical criteria (Sodi-Pallares, Bisteni, and Medrano, 1964) corresponds to pure incomplete right bundle-branch block configuration (Fig. 5B), thus suggesting a site of origin within the main left bundle-branch (Rosenbaum *et al.*, 1970b; Puech *et al.*, 1971).

Since narrow ventricular ectopic beats do arise at the ventricular level from the higher parts of the main intraventricular conducting fascicles, it may be assumed that when diastolic depolarization develops in such areas, an association of impaired conductivity is most likely; therefore transmission of

FIG. II (Case 6). Transient rate-dependent right bundle-branch block during the course of an acute myocardial infarction. Panel A. Twelve-lead electrocardiogram showing diaphragmatic infarction and Mobitz type I atrioventricular block. Panel B obtained the same day reveals ventricular tachycardia. After direct current cardioversion, a transient right bundle-branch block was recorded (panel C). In panel D (leads I and V1) recorded 2 minutes later, each long ventricular diastolic pause caused by an induced ventricular complex (s) or by a dropped beat is followed by an atrial complex with a similar right bundle-branch block configuration (phase-4 block). Note progressively decreasing degrees of right bundle-branch aberrancy of beats closing cycles of 0.64 sec or shorter in duration (phase-3 block). Since ischaemic injured Purkinje cells may develop generalized diastolic depolarization concomitantly with prolonged phase-3, consistent block of the impulse in the right bundle-branch can be explained by assuming the presence of two different mechanisms that alternate with each other. Normal intraventricular conduction was restored 4 minutes later (strip E).

the atrial impulse through such enhanced phase-4 areas must be delayed or blocked (Watanabe, 1971). This assumption appears to be strongly supported by the findings previously reported (Fig. 5A), where sinus beats showing variable degrees of bradycardia-dependent left bundle-branch block coexisted with narrow ventricular ectopic beats with pure incomplete right bundle-branch block. Variations in the spontaneous firing rate of such

centres of impulse formation can be explained by changes in levels of diastolic membrane potential in conjunction with variations in the extent of phase-4 depolarization and variability of the threshold potential (Singer *et al.*, 1967) in the cells located within the boundaries of the main left bundle-branch.

The development of phase-4 depolarization in the monofascicular pathway (His bundle), or in a

branch or division confronted by blockage of the remaining fascicles, will give rise to bradycardia-dependent atrioventricular block (Singer *et al.*, 1967). Disturbance of the atrioventricular conduction assigned to this mechanism has been referred to in recent reports: in monofascicular atrioventricular block (Slama *et al.*, 1969), in bifascicular atrioventricular block (Coulmel *et al.*, 1971), and in trifascicular atrioventricular block, both clinically (Rosenbaum *et al.*, 1973c) and experimentally (Da Ruos *et al.*, 1971).

All these, because of their clinical features, have been included in the paroxysmal atrioventricular block group (Sachs and Traynor, 1933), and are outstanding because of the sudden appearance of atrioventricular block, caused by acceleration or decrease of atrial frequency. Atrioventricular conduction is resumed after the escape of a subsidiary pacemaker, the latter keeping an adequate timing with the first atrial impulse which is conducted to the ventricles. These peculiarities observed in all our clinical (Fig. 7 and 10) and experimental (Da Ruos *et al.*, 1971) cases lead us to assume that this ventricular escape beat would have the function of depolarizing by retrograde conduction the blocked phase-4 fascicle, thus permitting the reinitiation of anterograde atrioventricular conduction (Rosenbaum *et al.*, 1973c).

From the findings in Tables 1 and 2 it seems that the bradycardia-dependent bundle-branch and atrioventricular block have common characteristics: a) a premature blocked zone, b) another one of conduction, and c) a tardy block zone. The presence of late blocking evidenced in branches by a bradycardia-dependent block of the affected fascicle or a bradycardia-dependent atrioventricular block, when occurring in a normal or acquired monofascicular pathway, is significant. It may be inferred from the above that the existence of a common mechanism attributable to prominent phase-4 depolarization appears to be a plausible explanation for the late blocking zone in bradycardia-dependent forms of impaired conduction.

In our cases of bradycardia-dependent atrioventricular block, supernormality cannot be invoked, since a wide range of atrioventricular conduction has been observed (Table 2) (Lewis and Master, 1924). In spite of the fact that repetitive concealed conduction of atrial impulses may provoke prolonged periods of ventricular asystole (Langendorf and Pick, 1964; Chung, 1971), this report establishes the close relation between the prolongation of the cardiac cycle length and the development of enhanced phase-4 depolarization as the underlying mechanism of the bradycardia-dependent paroxysmal atrioventricular block. There-

fore, it is unlikely that, in our cases, ventricular asystole may be attributed to concealed conduction (Moore, Knoebel, and Spear, 1971).

As chronic trifascicular atrioventricular block is often preceded by blockage in two or more of the three fascicles of the bundle-branch system (Rosenbaum *et al.*, 1970a), the most probable site where bradycardia-dependent paroxysmal atrioventricular block will develop in patients with right bundle-branch block coexisting with left anterior or posterior hemiblock and a prolonged HV interval (Table 2) seems to be in the main left bundle or in one of its principal subdivisions. This has been experimentally proved in the canine heart (Da Ruos *et al.*, 1972). Bradycardia-dependent paroxysmal atrioventricular block as a forerunner of complete atrioventricular block was almost invariably observed in our clinical cases. Therefore, an understanding of this type of impaired conduction may be clinically important because of the therapeutic value of cardiac pacemakers.

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