Electrocardiographic Observations in Bradycardia and Tachycardia-Dependent Atrioventricular Block

Relationship to Supernormal Phase of Intraventricular Conduction*

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This report describes the clinical course of a patient with bradycardia and tachycardia-dependent atrioventricular block. Bradycardia dependent A-V block (phase 4 block) was transient and precipitated by spontaneous slowing of the sinus rate, atrial and ventricular extrasystoles. The degree of slowing (critical RP* interval) required to induce A-V block increased progressively over a three-day period. Bradycardia-dependent A-V block was terminated mostly by critically timed spontaneous or paced ventricular escape beats, but normally conducted atrial impulses also appeared to restore A-V conduction on several occasions. The tachycardia-dependent component was manifested by an unusual fatigue phenomenon in the His-Purkinje system seen only at an atrial pacing rate of 150 per minute. These observations document the presence of both bradycardia and tachycardia-dependent A-V block in the presence of a normal H-V time and also illustrate the dynamic nature of both phase 4 block and the period of “supernormal” intraventricular conduction.

Several recent reports have emphasized the existence and significance of bradycardia-dependent atrioventricular (A-V) and intraventricular conduction disorders1-5 (phase 4 block) and documented their occasional coexistence with tachycardia-dependent conduction blocks.6-9

This report describes the clinical course of a patient with “paroxysmal” A-V block, both bradycardia and tachycardia-dependent. The sequential electrocardiographic and electrophysiologic observations give some insight into the mechanism of bradycardia-dependent A-V block and its relationship to the period of “supernormal” intraventricular conduction.

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Case Report

The patient is a 75-year-old woman who was hospitalized for investigation of syncope. She had been treated for mild congestive heart failure for several years. An electrocardiogram (ECG) five years before admission revealed sinus rhythm and an old inferior wall myocardial infarction. The duration of the QRS was 0.08 sec, and the PR interval was 0.18 sec.

The ECG on admission showed high degree A-V block (Fig 1). The QRS complex of the subsidiary pacemaker exhibited complete right bundle branch block with right axis deviation, suggesting an origin from the anterolateral part of the left ventricle. Intermittent conduction of supraventricular beats with a left bundle branch block configuration occurred after idioventricular beats only when the RP interval was between 500 and 640 msec (ie, during the “supernormal” period of conduction). The conduction pattern remained unchanged after 0.5 mg of atropine was administered intravenously.

About 90 minutes later, when 1:1 A-V conduction (with complete left bundle branch block) had returned, continuous monitoring revealed several prolonged episodes of ventricular asystole precipitated by spontaneous ventricular extrasystoles. A temporary demand pacemaker was inserted, and continuous monitoring subsequently confirmed that A-V block could be precipitated only by bradycardia. Bradycardia-dependent A-V block lasted for only 48 hours. On the third day, electrophysiologic studies including His bundle recordings demonstrated tachycardia-dependent A-V block in the His-Purkinje system.

Serial enzyme determinations (serum glutamic oxaloacetic transaminase, creatine phosphokinase, lactic dehydrogenase with fractionation) did not suggest an acute myocardial infarction. On admission, the digoxin level was found to be 1.1 ng/ml, with the potassium 3.1 mEq/liter. Digoxin was withheld for several days and potassium chloride solution was administered orally.

A permanent demand pacemaker was implanted, and the patient has remained asymptomatic. Over the next few months, A-V conduction was evaluated several times by inhibiting the demand pacemaker with chest wall stimula-
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Initiation of A-V Block (Fig 2,3,4)

A-V block was consistently precipitated by (1) spontaneous slowing of the sinus rate (Fig 2A, B); (2) early nonconducted and late conducted atrial extrasystoles (Fig 2C, D); (3) junctional extrasystoles (or ventricular extrasystoles from the right bundle branch) as in Figure 2E; (4) ventricular extrasystoles (a) spontaneously occurring multifocal ventricular extrasystoles as in Figure 2F and 3A, (b) electrically induced ventricular paced beats. Figure 3B-E illustrates sequential prolongation of the RP* interval as determined by the introduction of paced ventricular beats at various times in the cardiac cycle. On the third day, RP* intervals as long as 1,720 msec failed to produce A-V block (Fig 3E).

Termination of A-V Block

Supraventricular conduction returned in several ways: (1) Supraventricular beats, all with a PR interval of 0.24 sec, were seen on three occasions (Fig 4H). The constancy of the PR interval strongly suggests conduction of a sinus impulse with first degree A-V block, but escape beats from His bundle or right bundle branch cannot be excluded. These beats have a PR interval equal to those of conducted beats during periods of relative bradycardia (Fig 5D-F). (2) Ventricular escape beats with critical timing; three morphologic forms were observed: (a) a ventricular escape rhythm, (Fig 5A-D). This was seen only transiently and its failure to emerge subsequently was probably due to overdrive suppression from continuous supraventricular conduction;11 (b) single ventricular escape beats (Fig 2B); (c) ventricular-paced beats (Fig 4A-G). The effect of paced ventricular beats was studied by delivering single pacing stimuli with various timing. A-V block was consistently perpetuated by an RP interval longer than the RP* interval, but A-V conduction was always restored whenever the RP interval was shorter than the RP* interval.

Tachycardia-Dependent A-V Block and Special Electrophysiologic Studies

On the third day, His bundle recordings were performed by standard techniques,12 using a no. 5 bipolar catheter with electrodes 1 cm apart. A tetrapolar electrode catheter was also inserted from the arm and positioned in the high right atrium. The AH (110 msec) and HV (45 msec) intervals were normal during sinus rhythm at 63 per minute13,14 (Fig 6A). The HV interval remained constant at 45 msec after long pauses caused by paced-ventricular beats as well as single and paired atrial stimulation together with carotid sinus pressure.

The refractory periods of the A-V conduction system were evaluated during sinus rhythm by the extrastimulus technique.15 A single atrial premature depolarization (A2) was introduced after every eighth sinus beat (A1) at progressively shorter intervals (A1-A2 interval) until the atrium was refractory. The cardiac cycle was scanned twice by delivering rectangular pulses of 2-msec duration (at twice the diastolic threshold) at increments of 5 msec from a battery operated programmed stimulator (Medtronic SP 1340A Medronic Inc, Minneapolis). The effective refractory period (ERP) of the atrium (longest interval between the last sinus beat and the delivered stimulus S at which S failed to capture the atrium) was 230 msec during sinus rhythm and longer than the ERP of the A-V node. During sinus rhythm (PP = 840 msec), the interval between two consecutively con-
ducted His bundle depolarizations (H1-H2 interval) could not be shortened further than 400 msec.

The atrium was then paced at various rates by abrupt acceleration of the rate. Sinus rhythm was allowed to return for 30 sec whenever the pacing rate was changed. At an atrial pacing rate of 120/min (spike-to-spike interval, S-S = 500 msec) the ERP of the atrium was 235 msec and longer than the ERP of the A-V node, while ERP of the His-Purkinje system was now less than 380 msec (shortest observed H1-H2 interval) as tested by the extrastimulus technique after one minute of regular atrial pacing (Fig 6B).

When the SS interval was 415 msec, there was 1:1 A-V

![Figure 2. Initiation of bradycardia-dependent A-V block. Lead 2 in A and B, lead 1 in C-F. A, B, Initiation of A-V block by spontaneous slowing of sinus rate; C, A-V block precipitated by non-conducted early atrial extrasystole seen as deformity on ST segment of sixth beat; D, A-V block precipitated by late atrial extrasystole which does not appear to reset sinus node (interval between sixth and eighth P waves remains essentially double basic PP interval; normal response because extrasystole occurs late in PP cycle); E, A-V block precipitated by functional extrasystoles (or ventricular extrasystoles from right bundle branch); F, A-V block following ventricular extrasystole (second beat). Conduction of early atrial extrasystole observed in first few hours after admission (tenth beat in which RR interval is 400 msec) might be interpreted as suggesting absence of tachycardia-dependent A-V block at that time.7](image)

![Figure 3. Initiation of bradycardia-dependent A-V block by ventricular extrasystoles and paced ventricular beats. Lead 2 in A, and lead 1 in B-E. A, A-V block precipitated by ventricular extrasystole. In this electrocardiogram and all others irregular firing of artificial pacemaker was deliberately induced. B-D, A-V block precipitated by critically timed paced ventricular beats. In B, recorded at 24 hours, RP* interval was 1,020 msec. In C and D RP* interval increased to 1,180 msec toward end of second day when relatively late ventricular paced beats failed to engender sufficiently long postsystolic pauses to precipitate A-V block. At that time, carotid sinus pressure consistently failed to precipitate A-V block despite slowing of spontaneous sinus rate of 30 beats per minute. Retrograde P waves follow paced beats. E, Third day prior to His bundle recording: Combination of carotid sinus pressure and early paced ventricular beats failed to produce A-V block despite RP intervals as long as 1,720 msec. Retrograde P waves follow paced beats.](image)
conduction, with AH and HV intervals of 130 and 45 msec, respectively (Fig 7, top). At a pacing rate of 150/min (SS 400 msec), the first 14 paced atrial beats were all conducted with absolutely constant AH and HV intervals of 195 and 45 msec, respectively. However, the HV interval of the 15th beat suddenly increased to 65 msec (after the fourth spike in Fig 7, middle) and the 16th paced atrial beat (after the fifth spike) was blocked below the site of recording of the His bundle potential. This was followed by 2:1 block below the site of recording of the His bundle potential. During this pacing period, the rate, AH and HH interval (400 msec) remained absolutely constant. In contrast, atrial pacing at a

![Figure 4. Termination of bradycardia-dependent A-V block (lead 1 in A, C-G, and lead 2 in B, H). A-G. Delivery of paced ventricular beats at various times in cardiac cycle. A-V conduction never resumed when paced ventricular beat occurred in second half of sinus (PP) cycle. Paced beats in first half of PP cycle often but did not invariably restore A-V conduction. Note that paced beats in middle of B and F fall in first half of PP cycle, yet do not restore A-V conduction because succeeding sinus P wave occurs too late; presumably because sinus node was reset by retrograde atrial activation. A-V block always occurred when RP interval was longer than RP* at that particular time. A-V conduction was restored when RP was shorter than RP** interval. This relationship held true in repeated and lengthy observations with very long electrocardiographic recordings to minimize fortuitous circumstances. H, Resumption of A-V conduction by supraventricular beat with PR interval of 0.24 msec.](image)

![Figure 5. Strips A and D, lead 2 recorded on first day, show escape ventricular rhythm observed shortly after insertion of temporary pacemaker. Compare with Figure 1. RP* interval was 720 msec after supraventricular beat. It was, however, slightly longer after a ventricular escape beat. (In strip A, left, last F is conducted after RP interval of 720 msec, and RP interval before ventricular fusion beat (F) in strip D measures 780 msec). Influence of QRS morphology on duration of RP*, also seen in Figure 1, appears to reflect time of arrival of impulse to area undergoing phase 4 block. Strips E-F: Lead 2 recorded on first day showing sinus arrhythmia and rate-related changes in PR interval. RP* was 720 msec at that time. Note 40 msec prolongation of PR interval when RP increases from 500 to 680 msec. PR interval shortened when PP interval decreased after relatively long preceding PP interval. This excludes concealed reentry into A-V node.](image)
Discussion

The site of bradycardia-dependent A-V block was almost certainly below the A-V node and unrelated to vagal influence. The slight prolongation of the Pr interval seen during spontaneous slowing of the sinus rate (Fig 5E-F) probably occurred at the expense of the HV interval as a manifestation of bradycardia-dependent intraventricular block,21 rather than at the expense of the AH interval secondary to erratic changes in vagal tone.

The following points strongly favor infranodal block during bradycardia-dependent A-V block: (1) There was no clinical and electrophysiologic evidence of A-V nodal dysfunction. (2) The precipitation of A-V block by pauses following atrial and ventricular extrasystoles speaks strongly against A-V nodal block; few reports of such cases have been published,4,5,7,18 particularly in connection with ventricular extrasystoles.4,8 (3) Bradycardia-dependent conduction block (phase 4 block) favors a lesion outside the main body of the A-V node.19,20

In our patient, degenerative disease of the conducting system, digitalis, hypokalemia or ischemia either singly or in combination could have caused phase 4 block.20 Most reported cases of bradycardia-dependent A-V block appear related to phase 4 block in the left bundle branch.4,21-23 In our patient, the morphology of the ventricular escape rhythm (Fig 1,5) and escape beats (Fig 2) terminating bradycardia-dependent A-V block suggests phase 4 block in the right bundle branch, but a lesion within the His bundle (in association with fixed left-bundle-branch-block) cannot be ruled out.

The association of bradycardia-dependent A-V block with tachycardia-dependent infranodal block suggests a lesion at the same site responsible for both phenomena. Damato et al21 recently demonstrated during abrupt atrial pacing at rates of 150/min or less that functional block could occur in a normal His-Purkinje system when its ERP was longer than the ERP of the A-V node. This type of functional block occurs at the onset of pacing and follows the second paced beat because of the mathematical relation between ventricular cycle length and refractoriness.21 In our patient, however, A-V block in the His-Purkinje system precipitated by atrial pacing at 150/min is definitely pathologic for the following reasons: (1) The ERP of the His-Purkinje system was less than 380 msec at an atrial pacing rate of 120/min. Thus, at an atrial rate of 150/min when the HH interval was 400 msec, the ERP of normal His-Purkinje system should have been shorter than 380 msec22-24 and block should not have occurred. (b) The sudden development of A-V block after a delay of 15 beats suggests an abnormal “fatigue” phenomenon within the His-Purkinje system,25 because normal His-Purkinje tissue should progressively shorten its refractory period after a sudden increase in rate.26,27 This phenomenon could perhaps be considered as a remarkably atypical 16:15 type 1 block in the His-Purkinje system in which the only visible increment of conduction was represented by the sudden prolongation of the 15th HV interval before block occurred. These observations also highlight one of the limitations of the extrastimulus technique in the clinical evaluation of His-Purkinje conduction.

The association of tachycardia and bradycardia-dependent bundle branch block has now been well documented.1,8 Rosenbaum et al7 recently emphasized a similar association in “paroxysmal” (bradycardia-dependent) A-V block. In two patients without regular A-V conduction, Rosenbaum et al7 defined the presence of tachycardia-depen-

Figure 6. His bundle recordings. A, Recording during sinus rhythm (AH = 110 msec, HV = 45 msec). B, Atrial pacing at rate of 120 per minute. S1 denotes pacemaker spike of basic atrial drive (A1H1 = 105 msec). After one minute of regular pacing, premature atrial stimulus S2 was applied 255 msec after S1, resulting in A1-A2 interval of 325 msec; A2 is conducted to ventricle with A2H2 interval of 145 msec. H1-H2 interval of 380 msec indicates that effective refractory period of His-Purkinje system measures less than 380 msec at heart rate of 120 per minute. HBE = His bundle electrogram; HRA = high atrial electrogram. S = pacing stimulus delivered in high right atrium. A = depolarization recorded at site of His bundle recording. Fifty msec time lines generated electronically.
dent A-V block (which they called phase 3 block) on the basis of the RP interval of blocked beats. Their conclusions may not be valid because concealed retrograde conduction of ventricular escape beats to the A-V node might have caused functional A-V nodal block of subsequent P waves at an RP interval as long as 520 msec, particularly because the A-V node is often abnormal in diffuse disease of the conducting system. In this respect, retrograde conduction of ventricular beats to the A-V node has been well documented in complete A-V block due to trifascicular block. Similarly, in the cases of Rosenbaum et al with regular A-V conduction, the blocked beats at an RP interval of 200-280 msec or even up to 420 msec might also represent functional A-V nodal block rather than a true tachycardia-dependent infranodal component of “paroxysmal A-V block.” In the absence of His bundle recordings, the conclusions of Rosenbaum et al regarding the existence of a separate tachycardia-dependent infranodal component in their cases of “paroxysmal A-V block” must therefore remain speculative. Rosenbaum et al suggested ventricular escape beats restore A-V conduction by retrograde concealed conduction into the area undergoing phase 4 unidirectional block. The observed retrograde P waves (Fig 1) provide support for this mechanism. The RP* interval measured from the onset of the QRS complex would therefore depend on the time required for the impulse to reach the site of phase 4 block. This would explain the slight variation of the RP* interval, with changes in QRS morphology at any given moment (Fig 1,5). We measured the RP* interval by ignoring the presence of early retrograde or sinus P waves (ie, before the supernormal period), but this does not invalidate our conclusions. An early sinus P wave could have been blocked either in the A-V node or at the site of the lesion in the His-Purkinje system (concealed conduction). If, indeed, block had occurred at both of these sites (according to the timing of the early sinus P wave), the large number of RP* intervals measured after ventricular beats (at any one sitting) would have varied considerably. The absolute constancy of the RP* after ventricular beats (and its very close similarity to the RP* after a supraventricular beat when evaluated at
the same sitting) suggests that early sinus P waves were always blocked at the same site, i.e., the A-V node (due to concealed retrograde conduction of the ventricular impulses) so that sinus P waves did not interfere with the infranodal site of phase 4 block. The retrograde P waves obviously do not interfere with the validity of the RP* because such P waves are secondary to concealed conduction into the area undergoing phase 4 block. Resumption of A-V conduction following a supraventricular beat must have a different and as yet unknown mechanism.

The subject of supernormal conduction has recently generated a great deal of interest. In clinically, there is obviously nothing "super" about supernormal conduction because it refers to a paradoxical (in the sense that it is early) improvement of conduction in a tissue with depressed conduction. In their classic paper, Moe et al. presented many alternative mechanisms to explain apparent supernormality of A-V conduction. However, they did not deny the existence of supernormal conduction in the His-Purkinje system and, in fact, stated, "Relatively supernormal conduction, as a function of the rate of depolarization (dv/dt), may appear in Purkinje tissue of isolated preparations when widespread diastolic depolarization (pacemaker activity) takes place. The rate of rise of the action potential upstroke under these conditions will become progressively slower as the potential difference across the cell membrane decreases late in diastole." Yet, in the presence of bradycardia-dependent intraventricular block many investigators have been reluctant to invoke "supernormality" to explain improvement in conduction at relatively faster rates. Various descriptions and mechanisms have been cited: zone of opportunity (carefully distinguished from the supernormal phase), accordion effect (between phase 3 and phase 4), Wedensky facilitation and even pseudosuper normality, the latter being especially confusing because it also refers to the gap phenomenon of intraventricular conduction.

In severely damaged tissue, with phase 4 block the period of conduction would be confined to a narrow zone in early diastole (Fig 1,5) and would therefore correspond with the classic description of the brief supernormal phase of intraventricular conduction at the end of the T wave. Elizari et al. recently demonstrated in the dog that in phase 4 block, the zone of conduction gradually extends further into diastole as progressive improvement occurs in the damaged area (Fig 2,3). This concept was clearly expounded by Sarashke who pointed out that proximity to the T wave could not be used to define the period of "supernormal" conduction. Synchronous phase 4 depolarization with a gradual decline of the slope of depolarization would provide a plausible explanation for the progressive lengthening of the critical RP* interval in our case. However, the recent work of El-Sherif et al. (in dogs with acute ischemia) suggests that bradycardia-dependent block may be related to hypopolarization, shifts in the threshold potential and perhaps membrane responsiveness rather than enhanced phase 4 depolarization.

Whatever the mechanism, the sequence of events in our patient favors a more dynamic concept of the so-called "supernormal" phase (in reality simply the zone of conduction or according to Moe et al. the zone of relative supernormality) as it pertains to phase 4 block. The question arises whether supernormal conduction should be retained as a term in clinical cardiac electrophysiology, and, if so, whether it should refer to prolonged as well as brief (paradoxical) improvement of conduction. The need for better nomenclature, definitions and understanding of these phenomena in various clinical circumstances is clearly apparent.

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