Bradycardia-Dependent Bundle-Branch Block
A Critique and Proposed Criteria

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SUMMARY
Four cases of bundle-branch block (BBB) occurring with slow heart rates in patients with heart disease are presented. Recognition of bradycardia-dependent BBB is often difficult, and its differentiation from ventricular escape mechanism and from bilateral BBB requires careful analysis. A set of five criteria based on our own observations is presented with a review of the literature. The responsible mechanism for this seemingly paradoxical situation is not known. However, spontaneous depolarization of one or the other of the bundle branches in late stages of diastole appears to be the most plausible explanation.

Additional Indexing Words:
Ventricular escape mechanism Electrogram

INTERMITTENT BLOCK of the right or left branch of the bundle of His is a common observation when the heart rate is increased physiologically, pharmacologically, or during ectopic rhythms and reflects incomplete physiochemical recovery of the conduction pathways. The appearance of left or right bundle-branch block (BBB) upon slowing of the heart rate is much rarer. Altogether, less than 10 cases were said to have existed up to 1965, but the diagnosis appears acceptable in only four of them (table 1). The rarity of this particular type of conduction disturbance and its physiological connotations justify the following report of four cases that satisfy the criteria herein proposed.

Report of Cases
Case 1 (fig. 1)
Strips A and B were recorded when a mild arrhythmia was noted in this 79-year-old woman with aortic stenosis. The two segments in strip C were obtained 1 and 3 min after intravenous injection of 1 mg of atropine sulfate. In A and B, beats 3 through 6 and beats 8 and 10 display typical right bundle-branch block (RBBB) with RSR' configuration. They are preceded by sinus P waves at P-R intervals of 0.18 to 0.19 sec and thus are indistinguishable from normally conducted sinus beats. However, they differ from beats 1, 2, 7, 9, and 11 through 14 in that the beats with RBBB configuration are preceded by longer cycles, exceeding 1.38 sec, while the normally conducted beats are preceded by cycles 1.38 or less. RBBB occurs again when the heart rate is accelerated by atropine to 68/min or faster (cycle lengths, 0.92 sec or shorter) with the P-R interval remaining 0.18 to 0.19 sec (fig. 1C).

Comment
This is a relatively simple case of bradycardia-dependent RBBB. The beats are clearly conducted at normal P-R intervals. It is of particular interest that the right branch of the bundle of His also fails to conduct at the relatively rapid rate of 68/min after injection of atropine.

Case 2 (fig. 2)
The two strips of lead V1 are records from a 35-year-old man with viral myocarditis. Supraventricular tachycardia, probably atrial, at the rate of 190/min that was present at the beginning of strip A terminated spontaneously at the arrow. The next six beats, four of which are shown, are all preceded by P waves at P-R intervals of approximately 0.16 sec at a relatively slow heart rate of 83 to 90/min. The QRS configuration at the slow rate is that of typical RBBB,
which is considered to represent the bradycardia-dependent type of conduction disturbance. Strip B, showing conducted P waves, was recorded the next day and displays normal intraventricular conduction at this rate of 122 beats/min.

Comment

The only explanation other than bradycardia-dependent BBB is a ventricular escape mechanism in which the idioventricular beats displaying RBBB are fortuitously preceded by P waves. This occurrence in six consecutive beats, however, is highly improbable, and also the sinus arrhythmia present (R-R intervals of 0.83, 0.65, 0.63, and 0.71 sec) and the QRS configuration, which is that of typical RBBB, speak against a ventricular escape rhythm. In the latter situation, the

Abbreviations: MI = myocardial infarction; ASHD = arteriosclerotic heart disease; PMD = primary myocardial disease; HCVD = hypertensive cardiovascular disease.
rhythm is generally quite regular, and the QRS configuration is that of atypical BBB.

**Case 3 (fig. 3)**

The four strips of lead V₁ were selected from a continuous record taken during a period of several minutes in a 68-year-old man with alcoholic cardiomyopathy. The P-R intervals in 0.01 sec appear in small type below each P-R segment. Beats 1, 2, and 3 display normal intraventricular (IV) conduction at a heart rate of 73 to 74/min. Beats 4, 5, 6, and 7, which are
also conducted from the atrium, show LBBB at the heart rate of 75 to 76/min. The occurrence of LBBB with minimal acceleration of heart rate is not uncommon and is in keeping with the concept of critical heart rate. The shift between LBBB and normal configuration in beats 12 through 17 is also related to the length of the preceding cycles. Except for beat 14, which is a nodal escape, the beats in this strip are conducted from the atria at P-R intervals of 0.19 to 0.20 sec. Beats 8 through 11 are those of typical RBBB at the slow heart rate of 39 to 40/min, resulting from 2:1 A-V block. Beats 8 to 11 and 20 to 22 are examples of bradycardia-dependent RBBB. That these beats are conducted from the atrium is indicated by the P waves preceding them at P-R intervals of 0.18 to 0.19 sec and by the typical RS' configuration of the ventricular complexes.

Comment
This case shows not only the tachycardia-dependent LBBB at a critical rate between 74 and 75/min but also exhibits the bradycardia-dependent RBBB in beats conducted at the slow rate of 39 to 40/min. Complete block of the right bundle branch and 2:1 block of the left could explain beats 8 to 11 and 20 to 22. However, the ability of the right bundle to conduct normally in beats 1 to 7 and 12 to 17 in the same tracing makes the latter explanation unlikely.

Case 4 (fig. 4)
This continuous strip of V1 shows spontaneous change from normal IV conduction to RBBB when the rate slows from 67/min to 65/min in a patient with arteriosclerotic heart disease. All the beats are preceded by identical P waves at constant P-R intervals leaving no doubt concerning the conducted nature of the beats.

Comment
In this simple case, bradycardia-dependent BBB is the only plausible explanation.

Discussion
The report credited for being the first to have called attention to the entity of bradycardia-dependent BBB is that of Vessel in which a case of atrial fibrillation with slow ventricular rate and BBB was described (Vessel's case 2). Study of the published tracings, however, clearly shows that the beats having BBB occur in perfectly regular, slow runs at 39 to 40/min and are characteristically the result of an idioventricular escape rhythm, while the beats conducted from the fibrillating atria are irregular and possess normal, narrow QRS. In fact, multiple fusion beats and intermediate forms visible throughout the tracing strongly favor a dual origin for the two types of beats. Criteria 1 and 2 proposed in this communication (see below) and the accompanying figure 5 will elaborate further on this point. Case 8 of Comeau and associates published 3 years earlier has also been credited to be an example of bradycardia-dependent BBB. Here, too, careful analysis of the tracings and the accompanying text reveals that LBBB occurred only twice in single beats when the heart rate was slowed to 40 and 30 beats/min, respectively, through carotid sinus stimulation. Even though the one beat illustrated by the authors is preceded by a reasonable P-R interval, a ventricular escape mechanism cannot be ruled out, as pointed out in Criterion 3 (see below).

The first unquestionable case of BBB occurring with slowing of the rate was that of Dressler published 20 years later. Here, the beats exhibiting LBBB were conducted from the atria at P-R intervals of 0.20 sec, and the heart rate at which LBBB appeared was approximately 44/min. In the case of Wallace
and Lazlo, intermittent BBB was observed in a 46-year-old man with old myocardial infarction. The beats showing LBBB generally occurred after slowing of the heart rate from 80-100 to 45-60/min. However, since identical IV conduction disturbance also occurred with acceleration of the heart rate from 80-100 to 117-140/min, the authors expressed doubt as to the existence of a causal relationship between the BBB and the slowing of heart rate. It must be noted, however, that occurrence of BBB upon minor acceleration of heart rate is a recognized phenomenon in ischemic heart disease and need not detract from consideration of bradycardia-dependent BBB in the same patient. Case 1 of the present report exemplifies this phenomenon.

In a more recent report on this subject Vessel and Lowen reviewed the literature on the subject and added two new cases. Again, their case 1, because of atrial fibrillation, was not acceptable on the basis of Criterion 2 (see below). Case 2 was one of incomplete heart block in which slowing of the ventricular rate due to 2° A-V block was associated with the appearance of LBBB. However, careful study of the published strips shows that the P-R intervals preceding LBBB beats are shorter than those situated before the narrow QRS complexes, thus making the diagnosis of bilateral BBB inescapable. Most recently, Bauer and associates presented two cases of bradycardia-dependent BBB which probably satisfy the criteria proposed in this presentation. The leads were recorded from the monitor oscilloscope and, in the one tracing published, it is not clear whether the left or right bundle branch failed.

The addition of our four cases to the existing four in the literature serves to indicate that the phenomenon does indeed exist and that further search may unearth many new cases. The responsible mechanism cannot be gleaned from the information contained in the published and present cases. However, it may be speculated that one or the other bundle branch regains its functional refractoriness at the end of long diastolic periods.
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after having been responsive in earlier phases of diastole. Clearly, the responsiveness in earlier stages of diastole cannot be ascribed to the supernormal phase of conduction, for this explanation would be tenable only if the boundary separating the normal from BBB beats were situated near the end of the T wave of the preceding beats. In fact, because of slow heart rates observed in this study, the boundary was situated at a considerable distance beyond this point. Another possible explanation is that of a direct vagal effect on the branch of the bundle, causing a functional block. This was a favored explanation for QRS changes observed after carotid sinus stimulation as early as 1911 and again evoked in 1938 by Comeau and associates. In our experience, however, the IV conduction disturbances we have observed following stimulation of the carotid sinus could invariably be explained on the basis of slowing and the emergence of a ventricular escape rhythm.

The experiments of Moe and associates on dogs have shown that the right bundle branch may be inherently “weaker” than the left and that “at low driving rates the right bundle may often be the cardiac tissue with the longest effective refractory period.” Moe and his colleagues demonstrated further that these differences were inherent in the His bundle and its branches and that they were independent of the vagus, the sympathetics and the A-V node. These observations provide ample support for the thesis that the two bundle branches may not behave similarly in response to various influences, but fail to explain the emergence of BBB after a period of normal conduction. The most tenable explanation for the observed BBB at slow heart rates, in our view, is the inherent ability of one or the other bundle branch to depolarize spontaneously during diastole (phase 4 depolarization) and thus become refractory to the next impulse arriving from the atria or the A-V node. This phenomenon which characterizes all pacemaker tissue of the heart is shared by both bundle branches, as shown clearly by Singer and co-workers. According to these authors, “Spontaneous diastolic depolarization of automatic cells could result in diminished excitability and conduction disturbances.” They stated further that: “Impulse propagation into the His-Purkinje system when membrane potential is reduced by phase 4 depolarization may, therefore, be a cause of atrioventricular and intraventricular conduction disturbances, particularly those occurring at slow heart rates.”

The role of digitalis in this type of BBB has not received attention. It is of some interest that all of our patients were receiving this drug, and two of them were thought to be overdigitalized.

Sources of Error In and Criteria For Diagnosis of Bradycardia-Dependent BBB

Figures 5 and 6 will assist in describing certain common errors and in presentation of the proposed criteria. The three strips of lead V₆ in figure 5 were recorded from three separate individuals, but the beats are numbered sequentially. The numbers below the bottom strip show the R-R cycles between beats 28 to 37. In the top row, beats 1, 2, 5, 6, 8, 9, 10, 14, and 15 are normally conducted with P-R intervals of 0.16 sec and QRS duration of 0.10 sec. Beats 3 and 11 are clearly premature ventricular in nature, followed by ventricular escape beats 4, 12, and 13, respectively. The

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fortuitous appearance of P waves before beats 4 and 13 may mislead one in considering conduction from the atrium and the existence of bradycardia-dependent BBB. However, the identical intraventricular conduction disturbance in beat 12 which is not preceded by a P wave favors an escape mechanism. Moreover, the configuration of these beats, which is atypical for RBBB in V₁, is another point in favor of a ventricular origin. The very short P-R interval preceding beat 7 is still another point not consonant with conduction from the atrium along the normal pathways (see Criterion 1 below). The middle strip demonstrates a basically normal sinus rhythm with P-R intervals of 0.16 sec and a QRS duration of 0.07 sec. Beats 18 and 21, on the other hand, resemble RBBB, and since they are preceded by P waves at P-R intervals of 0.18 and 0.16 sec, respectively, may be considered examples of bradycardia-dependent BBB. Study of a very long strip of V₁ in this case finally uncovered beat 27 with identical IV conduction disturbance but without the preceding P wave, thus suggesting a ventricular origin for beats 18, 21, and 27. The failure of the long pause of 1.15 sec preceding beat 24 to cause bradycardia-dependent BBB in that beat further supports the thesis that the RBBB appearance in beats 18 and 21 was not BBB of the bradycardia-dependent variety. The bottom strip is a record of atrial fibrillation in which beats 30, 32, 36, and 37 display normal IV conduction. Beats 28, 29, 31, 34, and 35 show the appearance of RBBB and are preceded by long pauses, thus typical of ventricular escape beats. It would be impossible to rule out a nodal escape mechanism with bradycardia-dependent BBB. However, the presence of the fusion beat 33 favors the coexistence of a supraventricular beat with a ventricular focus, deplorizing the ventricles simultaneously.

Figure 6 depicts the tracing of a 56-year-old man with hypertension and LBBB. Beats 1, 4, 5, 6, and 10 represent his basic normal sinus rhythm with a P-R interval of 0.19 sec and QRS duration of 0.16 sec. Beats 3 and 9, displaying normal IV conduction with a QRS interval of 0.09 sec, are premature atrial beats with their ectopic P waves visible in the T waves of the preceding beats. It may be argued that beats 3 and 9 are the basic control beats with normal QRS complexes and that beats 1, 4, 5, 6, and 10 represent examples of bradycardia-dependent BBB. However, the beats with narrow QRS complexes are situated just beyond the end of the T waves of the preceding beats and, therefore, are very likely to be supernormally conducted.

From a review of the pertinent literature, the present study, and a study of hundreds of tracings of the types depicted in figure 5, a set of five criteria for diagnosis of bradycardia-dependent BBB has been developed, as follows:

1. The beat displaying wide QRS must be conducted from the atria to the ventricles along the normal pathways. This criterion demands that a P wave precede the QRS at a reasonable P-R interval of between 0.12 and 0.60 sec. Eliminated, on this basis, are the Wolff-Parkinson-White beats which are characterized by short P-R intervals and delta waves, and those beats which occur after extremely short or long P-R intervals and are therefore not likely to be conducted (fig. 5). Aberrantly conducted nodal escape beats with retrograde conduction to the atria cannot be considered at this time because of our inability to differentiate them from ventricular beats with retrograde ventricular-atrial conduction.

2. Atrial fibrillation or flutter must not be present, for in such cases the possibility of ventricular escape as the responsible mechanism for BBB after long cycles can never be eliminated (fig. 5). Moreover, bilateral BBB with shorter absolute refractory period but slower recovery in one branch than the other cannot be excluded when conduction alternates between RBBB and LBBB, as described by Lepeščkin.14

3. The phenomenon must appear in more than one beat because the appearance of BBB in a single beat can always be attributed to a ventricular escape, even though the beat
may be preceded by a P wave at a reasonable P-R interval (fig. 5). However, occurrence of such beats in sequence renders the probability of ventricular escapes being preceded by independent P waves at constant P-R intervals extremely remote.

4. Incomplete bilateral BBB with IV conduction varying from normal to LBBB or RBBB must be excluded. This criterion demands that the characteristic changes of the P-R intervals accompanying QRS alterations as outlined lucidly by Burchell in 1949 and more recently by Rosenbaum and Lepeschkin and Schloff and associates must not obtain. The use of a simple diagram proposed by the author and Sarin will assist in the analysis of tracings of suspected cases of bilateral BBB.

5. The concept of supernormal conduction must not apply to the normally conducted beats (fig. 6).

References


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