Bilateral Bundle-Branch Block

Clinical and Electrocardiographic Aspects

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SUMMARY

The electrocardiograms and salient clinical features of seven patients with electrocardiographic evidence of various types of bilateral bundle-branch block (BBBB) have been presented. When the complexes change in form and the P-R interval varies, the mechanisms are usually best interpreted by using the concepts of first degree, second degree, and third degree bundle-branch block with or without block in the higher conduction system. The etiology of BBBB in many cases is idiopathic fibrosis of both bundles. The diagnosis of BBBB by ECG is important because of its relation to the subsequent development of complete heart block or Adams-Stokes syndrome, or both. In this series five of seven patients with BBBB had Adams-Stokes syndrome and three of seven had complete heart block.

ADDITIONAL INDEXING WORDS:
Adams-Stokes syndrome

With the development of effective treatment for heart block, the diagnosis of bilateral bundle-branch block (BBBB) has assumed increased importance. In some cases the electrocardiographic (ECG) abnormalities of BBBB precede the serious clinical consequences of complete heart block, notably Adams-Stokes attacks. This communication is concerned with the clinical aspects of seven patients and the interpretation of their ECGs which illustrate various mechanisms of bilateral bundle-branch block. Previous reports were either of a theoretical nature or presented only isolated illustrations, usually of a single type of BBBB.

Current pathological studies confirm earlier work in that complete heart block usually shows organic damage, principally fibrotic lesions, of both bundles of His. Mahaim, Yater and associates, Lenegre, and Lev found microscopic pathology of both bundles consistently in their studies. Yater, in a review of cases of complete atioventricular block, found that both bundle branches were usually involved. As a rule, one branch was much more involved than the other. Although in four of the six cases, there were no lesions of the heart adjacent to the bundles, he attributed the etiology to arteriosclerotic heart disease. He also stressed that a bundle branch need not be destroyed completely at a given level to give bundle-branch block (BBB). The QRS configuration and duration depended on the relative degree of damage.

Recent reports of the pathology in complete heart block serve to document further the presence of BBBB lesions. Lenegre reported that arteriosclerotic heart disease was felt to be the direct cause of BBBB in only one fourth of his 62 cases. In the majority of his patients the direct cause was not known. Slightly over half had evidence of myocardial infarction. Seven of the 62 were found to have aortic valve disease, usually calcific aortic stenosis. Four of 62 had subtotal destruction of the main bundle, 32 had a lesser degree of

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involvement, and 26 had normal common bundles. Forty patients were found to have total destruction of the right bundle and 19 others, subtotal destruction. The left bundle was totally destroyed in nine and subtotally, in 24. Many combinations of right and left bundle involvement were observed, but complete destruction of both bundle branches occurred in only six of 62 patients. In general, the fibrotic lesions were localized and nonspecific and were found more often in the right bundle.

In addition to pathological studies such as the above, clinical investigations have shown a relatively high number of patients with no associated heart disease. The highest incidence of no associated heart disease to account for complete heart block was found in the study of Zoob and Smith. Thirty of 51 patients were felt to have "primary" complete heart block. Curd and associates 6 gave an incidence of 14% for the number of patients without clinically associated etiology. The study of Friedberg and associates, from whom four of the seven patients of this study were drawn, found 28% without specific related disease.

In 1912, Mathewson described several cases of bundle-branch block, in one of which alternating complexes indicated transient conduction of both branches. Since then many cases have been described which have been interpreted as BBBB. 10

Lenegre observed an electrocardiographic progression of bundle-branch block (BBB), usually right, followed by right BBB (RBBB) in the precordial leads with marked left axis deviation in limb leads (atypical RBBB) leading to complete heart block frequently associated with Adams-Stokes attacks.

Scherf and Shoook, in 1926, described experiments in which one bundle of the dog heart was cut and the other pressed lightly. With this technique, complete heart block and the several types of less severe forms of heart block were produced. The combinations included lengthened P-R intervals and various forms of second degree block.

Rosenbaum and Lepeschkin 10 and Lepeschkin have discussed the ECG diagnosis of BBBB in a systematic fashion applying the concepts of first degree, second degree, and third degree block of the bundles of His. Prolongation of the P-R interval may therefore be due to block in either the nodal area and common bundles or to block in one or the other bundles of His. When an ECG pattern with prolonged P-R interval is associated, at other times, with a bundle-branch block pattern and a shorter P-R interval, the delay in atrioventricular conduction in the initial tracing was presumed to have been in the bundle branches. Second degree block in the bundle branches is represented by a variety of patterns such as alternating right and left bundle-branch block and various forms of second degree heart block.

Third degree block in a bundle branch may be manifested by a simple BBB pattern if the other bundle is normal or has only first degree block. Any combination of first degree, second degree, or third degree heart block can occur, but the diagnosis of the exact mechanism may be apparent only when the pattern changes to reveal the conduction abnormality in the opposite branch.

The following examples will illustrate several forms of BBBB. Each discussion of an ECG will be preceded by a clinical description.

**Report of Cases and Electrocardiograms**

**Case 1**

A 63-year-old white female was admitted to The Mount Sinai Hospital on February 20, 1965, with the chief complaint of fainting. Past history included rheumatoid arthritis with diffuse vasculitis, recurrent supraventricular tachycardia, and an episode of upper gastrointestinal bleeding. The fainting episodes had begun in May 1964 and had continued, but at times only dizziness was experienced. Subsequent events strongly suggested that these episodes were Adams-Stokes in nature. Electrocardiograms prior to this admission indicated first degree atrioventricular block (0.24 sec), normal QRS duration, and left axis deviation (October 30, 1964) and left bundle-branch block (LBBB) with P-R interval of 0.24 sec (January 20, 1965). The electrocardiograms during hospitalization varied considerably with periods of tachycardia, probably supraventricular, occasional second degree
Bilateral bundle-branch block with the most probable mechanism being first degree block of each bundle. (All figures are discussed in detail in the text.) In this and the following figures: P represents P waves; A, the impulse in the sinus node and atrium; AV, the impulse in the atrioventricular area including the common bundle; V, conduction below the common bundle with the interrupted line being normal conduction through the separate bundles (by convention 0.20 sec). The right and left bundles are represented by the oblique solid or interrupted lines in the V area. Their relative lengths indicate delay in one or the other bundle branch. The right bundle branch is on the reader's left and the left branch on the reader's right. Letters below each beat indicate normal conduction (N), left bundle-branch block (L), right bundle-branch block (R), nonconducted P wave (O), and unknown conduction (-). Horizontal lines with "2" indicate possible delay in the A-V node or common bundle. In the V area a short perpendicular line ending on oblique line indicates complete block.

Electrocardiogram (Fig. 1)

In the first tracing (February 24, 1965) 1 to 1 conduction is present at a rate of 75 per minute. The P-R interval measures 0.24 sec and the pattern is that of LBBB. The second ECG taken 6 days later has a pattern of RBBB again with 1 to 1 conduction but with a P-R interval of 0.28 sec. Both electrocardiograms are from lead
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V1. That the atrioventricular (A-V) conduction should vary coincidentally with the change in bundle-branch block is unlikely. With LBBB, the right bundle probably conducts with a delay of 0.24 sec but could have a mechanism of second degree or third degree block as illustrated. Here it is presumed that the delay down the right bundle prolongs the P-R interval to an abnormal degree. When the right bundle develops either greater first degree, second degree, or third degree block, the left bundle conducts with a P-R interval of 0.28 sec. While the pattern was LBBB, the left bundle was probably capable of conduction with this delay, but this was not suggested until the second tracing showing RBBB was recorded. Because the rates in both tracings are 75 per minute, this change cannot be attributed to a rate dependent bundle-branch block. The possibility that the atrioventricular node or the common bundle contributed to some of the abnormal delay cannot be excluded (see "AV" area of figure 1, first complex of each strip). In summary, incomplete bilateral bundle-branch block is illustrated. When LBBB is present, the left bundle is most probably capable of conduction with a total P-R interval equal to its P-R interval during RBBB. Therefore, these two ECGs are interpreted as bilateral bundle-branch block, asynchronous, first degree.

Case 2

This patient was a 55-year-old white male with a past history of hypertension, diabetes mellitus, and arteriosclerotic heart disease manifested by myocardial infarction and angina. The previous electrocardiograms showed initially left axis deviation, P-R interval of 0.12 sec, and RBBB with rsR' in V1 (May 1954). This ECG may be interpreted as "atypical right BBB." Two years later, in May 1956, the axis had changed to marked right axis deviation with a taller R' in V1. At this time an anterolateral myocardial infarction was diagnosed by both clinical course and ECG. During this admission, without digitalis administration, the patient progressed from first degree heart block to second degree (principally 2:1), and then to transient complete heart block. When he left the hospital the basic pattern was unchanged but the P-R interval had increased to 0.20 sec. At the time of the tracing illustrated (fig. 2), the patient was admitted again for myocardial infarction. During this admission transient complete heart block was again noted. In August 1957, complete heart block was established with Adams-Stokes syndrome. This mechanism continued until his death in refractory heart failure on November 3, 1958.

Electrocardiogram (Fig. 2)

Lead III is illustrated with 2 to 1 heart block and two types of ventricular complexes. The first two are RBBB in pattern with a P-R interval of 0.18 to 0.19 sec; the remaining complexes are LBBB in pattern with P-R interval of 0.30 to 0.31 sec. The P-R intervals during any particular QRS pattern are essentially equal. Various reasons may be offered to explain why the P-R interval is longer with the LBBB than with the RBBB pattern. This form of A-V block may be due to a 2 to 1 block in the A-V node or common bundle, but it may also be explained by

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synchronous 2 to 1 block in both bundle branches. In the latter case, the right bundle delay is assumed to be fixed at 0.30 sec with 2 to 1 right bundle block and the left bundle conducts with a normal P-R of 0.18 sec and 2 to 1 block. This tracing is felt most probably to represent synchronous but unequal second degree (2:1) bundle-branch block. The normal P-R interval when the impulse is conducted through the left bundle indicates that the prolonged P-R interval when the impulse is conducted through the right bundle is due to impaired conduction in the right bundle and not in the A-V node. When a single beat, such as the first beat, is seen alone, there is indication that conduction is normal through the A-V node, main bundle, and the left bundle branch. The block in the right branch may be first degree, second degree, or third degree, or a combination. The following P waves and the second conducted beat indicate that there is 2 to 1 block in both bundle branches. The prolonged P-R interval before the third QRS complex with a LBBB pattern indicates not only a 2 to 1 block in the right branch but also a first degree block when the impulse is conducted through that branch, as shown by the P-R interval of 0.30 sec.

Case 3
This 71-year-old white female with a history of hypertension and recent angina pectoris was admitted on November 12, 1959. In the spring of 1959 she had suffered an Adams-Stokes episode with residual weakness of the right leg. At that period the patient was receiving digitalis, and 2 to 1 block was noted. On the day of the current admission the patient was hypertensive with a pulse rate of 36 due to 2 to 1 heart block. Mild congestive heart failure was also present. Treatment, including use of diuretics, isoproterenol (Isuprel), and atropine, yielded a good temporary response, but the patient was unexpectedly found dead on November 23, 1959.

Electrocardiogram (Fig. 3)
Lead V₁ represents 2 to 1 conduction with RBBB pattern in the conducted beats and V₆.
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Figure 4

Bilateral bundle-branch block with changing mechanisms in both branches.

taken 5 days later, shows LBBB with 2 to 1 conduction. There is a difference in P-R intervals with a P-R interval of 0.23 sec in the presence of RBBB and a P-R interval of 0.18 sec in the presence of LBBB. Two to one block at the A-V node or common bundle would explain the 2 to 1 abnormality in A-V conduction, but a change in the A-V node conduction time coincident with each bundle-block pattern is unlikely. A synchronous, unequal, 2 to 1 BBBB would most simply explain the tracing. Thus, when LBBB is present and the impulse is conducted down the right bundle, the normal P-R interval of 0.18 sec indicates normal conduction in the right bundle as well as the A-V node and main bundle. The prolonged P-R interval during RBBB when the impulse passes down the left bundle indicates that the delayed conduction is in the left bundle since conduction was shown to be normal in the A-V node and common bundle. Since the records were take a few days apart, it is possible though unlikely that the conduction through the A-V node and bundle was normal on November 17 but prolonged on November 12.

Case 4
This patient was an 89-year-old white male with a history of localized carcinoma of the prostate, renal insufficiency, arteriosclerotic heart disease with congestive heart failure, and a remote cerebrovascular accident. At the time of the tracing taken on August 16, 1957, Adams-Stokes episodes were diagnosed. Digitalis was discontinued and no other therapy was given for the
episodes. The patient was readmitted on September 22, 1957, after another Adams-Stokes episode associated with documented ventricular fibrillation. During this hospitalization, several more Adams-Stokes attacks were observed. The second ECG was taken during this second hospitalization. The next tracing was taken in the course of a third hospitalization during which the patient died. The cause of death was bronchopneumonia and no fainting episodes or periods of complete heart block occurred.

Electrocardiogram (Fig. 4)

In the first strip a 2 to 1 LBBB conduction pattern is observed with the P-R interval of 0.23 sec for the conducted beats. The increased P-R interval and 2 to 1 block may be due to delay and block in the A-V node or common bundle as indicated in the figure, but subsequent records suggest otherwise. The second tracing has a right bundle-branch block pattern with a shorter P-R interval than the first (0.20 sec versus 0.23 sec) and 1 to 1 conduction. This suggests that when the left bundle branch was blocked (first strip) the P-R interval was prolonged because of delayed conduction down the right bundle, whereas when the right bundle branch was blocked the P-R interval was shorter (0.20 sec) because there was less delay in conduction down the left bundle. The subsequent incidence of intermittent left and right bundle-branch block suggests that the 2 to 1 A-V block is more likely to be due to varying degrees of bilateral bundle-branch block than to a transient block through the A-V node. Probably the right bundle during

Figure 5

Bilateral bundle-branch block with a rate related response of the R bundle.

the second strip is capable of conduction with a delay similar to that in the first tracing. This is confirmed in the third tracing in which the right bundle does conduct with a similar delay. The function of the left bundle has changed once again because the initial three and last complexes are of normal duration with prolonged P-R intervals. Here, a synchronous equal bundle-branch block began the tracing, but improvements in the conduction through the right bundle allow 1 to 1 conduction with a LBBB pattern without change in the P-R interval. Once again, changes in conduction through A-V node and common bundle may play a role, but the arrhythmias can be explained most simply by block in each branch alone.

Case 5

This 79-year-old white male had no previous history of cardiovascular disease prior to admission on June 13, 1957. In the week before admission, the patient had suffered from three Adams-Stokes attacks. The tracing in figure 5 was taken during this admission. Physical examination revealed a pulse rate of 37 per minute and mild congestive heart failure. The pulse rate was 50 per minute on discharge. Electrocardiograms during this admission showed arrhythmia, LBBB with P-R interval of 0.18 sec, occasionally with P-R interval of 0.16 sec, and periods of complete heart block. His hospital course was uneventful in spite of the episodic complete heart block and no further Adams-Stokes attacks occurred.
Electrocardiogram (Fig. 5)

This ECG reveals intermittent second degree block which is never greater than 2 to 1. Usually a LBBB pattern occurs when the P-R interval appears shorter than usual. But in this electrocardiogram, in which the P-R interval is most accurately measured in lead II, the P-R interval preceding the narrow complexes is 0.18 sec and that of the LBBB complexes is 0.20 sec. Since, in left bundle-branch block, conduction is through the right bundle, the longer P-R interval during LBBB indicates delayed conduction in the right bundle, that is, right as well as left bundle-branch block.

The shortest P-R interval is seen in lead V₁ preceding an RBBB complex (fifth QRS complex in the fourth strip). There is no dropped beat preceding this complex, that is, conduction is 1 to 1. This P-R interval is not only shorter than that preceding LBBB complexes but also is somewhat shorter than that preceding normally conducted, narrow, QRS complexes. The P-R interval preceding the RBBB beat is due to better conduction in the A-V node and common bundle and may be related to the 1 to 1 conduction prior to this beat with slower A-V conduction during 2 to 1 block when the QRS is narrow.

Case 6

This patient, a 67-year-old white male, underwent laparotomy in 1952 for a possible dissecting aneurysm of the abdominal aorta. Over the next 10 years, the patient was admitted for several problems including hypertension, renal insufficiency, arteriosclerotic heart disease with two myocardial infarctions, and angina pectoris. A review of the electrocardiograms since 1952 showed progression from a normal complex with P-R interval of 0.17 sec to RBBB with P-R interval of 0.20 sec and left axis deviation (another
example of “atypical” RBBB), to 2 to 1 conduction with RBBB, then to 1 to 1 conduction with LBBB with P-R interval of 0.30 sec. At the time of the illustrated tracing (fig. 6), the patient had pulmonary edema and anemia. Following this admission the patient was observed in complete heart block associated with Adams-Stokes episodes. Later his ECG reverted to an RBBB pattern, 1 to 1 conduction, with a P-R interval of 0.20 sec.

Electrocardiogram (Fig. 6)

Two types of wide ventricular complexes are noted in V₁. The first, fourth, sixth, and eighth are RBBB in configuration; the others are LBBB. The P-R interval preceding the RBBB complexes is 0.14 sec, the P-R intervals preceding the LBBB complexes are 0.34 sec except before the third ventricular complex where it is 0.36 sec. In addition, a second degree block (Wenckebach’s phenomenon) is present with the fourth, seventh, and tenth P waves not being conducted. Sinus arrhythmia causes the P-P interval to vary by approximately 0.20 sec.

Several mechanisms could explain this sequence of events. The second degree block may be localized to the A-V node or common bundle or to the bundle branches (synchronous second degree block of each bundle branch): (1) If the block is in the upper conducting system (schema A, fig. 6), a long delay would permit the left branch to conduct normally with a P-R interval of 0.14 sec, but repolarization is then delayed longer than in the right branch allowing the right branch to conduct after a P-R interval of 0.34 sec. Wenckebach’s phenomenon in the A-V node or common bundle is suggested by the slightly longer P-R interval of the second LBBB beat followed by a nonconducted P wave. This basic pattern is repeated with the longer delays being followed by short P-R intervals and RBBB, then LBBB, and then a nonconducted P wave. (2) The pattern can be explained without invoking A-V block (schema B, fig. 6) if the right bundle has a second degree block with 4 to 3 and 3 to 2 conduction and prolonged P-R interval. The left bundle has a second degree block also, but the ratio is not clear nor is the conduction time suggested except after a long pause when normal conduction occurs. (3) If both bundles

Figure 7

Bilateral bundle-branch block with 3 to 2 block.
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conducted with Wenckebach's periods which were synchronized (schema C, fig. 6), the conduction times would have to be unequal with the increase being faster in the left than in the right. (4) Another possibility (schema D, fig. 6) is that each branch has a high degree of second degree block and a nonconducted P wave when the blocked intervals are synchronous. In this case further block of either bundle would result in a high degree of heart block. Second degree block at the A-V node or common bundle may be associated with any one of the combinations discussed.

Case 7

A 63-year-old white male was admitted on February 19, 1965, because of headache, paresis of the left lower leg, and fever. The history was a complicated one, including diabetes mellitus, recent staphylococcal pneumonia, bilateral otitis media, and a probable brain-stem lesion. There was no history of arteriosclerotic heart disease, hypertension, Adams-Stokes syndrome, or complete heart block. The ECG changes were recorded during a period of oscillating fever but were not associated with Adams-Stokes episodes. No digitalis was administered.

Electrocardiogram (Figs. 7 and 8)

The first tracing, taken February 23, 1965, is interpreted as incomplete right bundle-branch block with second degree heart block. Irregular increments of the P-R interval do not allow one to make the diagnosis of classical Wenckebach's phenomenon although the ECG pattern resembles it.15 Two days later second degree block remains, but the recurrent pattern is an LBBB complex, then a narrow complex followed by a nonconducted P wave. Three to two A-V block (schema A, fig. 8) would explain the basic conduction abnormality with the right bundle recovering more quickly than the left after a long pause. When the P wave initiating the narrow beat is conducted, the abnormal P-R interval may be due to either equal right and left BBB or higher delay in the A-V node or common bundle. As in previous examples, the

Figure 8

Schemata A, B, and C referring to V2-25-65, figure 7.
mechanism may be explained without A-V or common bundle delay. Here the two bundles have a synchronous block every third beat (schema B, fig. 8). Conduction through the right bundle may have Wenckebach's phenomenon, the P-R interval of the QRS after the nonconducted P wave being 0.35 sec, and then lengthening to 0.38 sec to equal the delay of the left bundle. The left bundle may be capable of conducting with a P-R interval of 0.38 sec in the left bundle-branch block beat or may be completely incapable of conduction (3 to 1 block of the left bundle; schema C, fig. 8). In the last tracing the mechanism is RBBB with a normal P-R interval of 0.14 sec. This tracing confirms the diagnosis of BBBB but gives no clue to the possible mechanisms in the above records. The conduction of the right branch could be more delayed, being a form of second degree block or third degree block.

Discussion

This communication presents four patients from a group of 100 patients with Adams-Stokes episodes collected during a period of 15 years before techniques of pacemaker implantation became available. The remaining three were noted recently in the course of routine ECG interpretation. The true incidence of BBBB is probably much higher than the gross figures would indicate. Many of the early tracings of patients with Adams-Stokes syndrome were unavailable. If patients with suspected Adams-Stokes episodes were followed by frequent ECG recordings or by being monitored, many more instances of transient BBBB would be likely to come to light.

An awareness of the problem is necessary for recognition of BBBB in the ECG. When a BBB pattern is present with a prolonged P-R interval or longer P-R interval than previous tracings, BBBB can be suspected. Subsequent recording may then show changes in pattern that allow one to make a definite diagnosis of BBBB. The exact electrocardiographic mechanism has been difficult to prove especially with regards to the degree of second degree block of the bundles. In some cases of BBBB only functional changes in conduction may occur that do not indicate as dire a prognosis as Adams-Stokes episodes, complete heart block, and associated complications. As has been seen from this group of patients, the diagnosis of BBBB is of importance because it often precedes the development of complete heart block and Adams-Stokes syndrome.

The average age of this group of patients was over 70 years. Five of the seven suffered from Adams-Stokes episodes but, as pointed out above, four were drawn from a population of such patients. Three of seven had documented complete heart block and four suffered from a manifestation of arteriosclerotic heart disease. Hypertension was present in four patients and, as discussed above, the direct cause of the lesions in the bundles cannot be easily inferred from the clinical form of heart disease. All patients but one suffered from either Adams-Stokes syncope or complete heart block.

Several forms of block in both bundles of His are not illustrated. Delay in the onset of the intrinsicoid deflection of conducted beats in both the right and left precordial leads, indicating BBBB, has been described by Boyadjian and Van Dooren. In the records available to us, this form of BBBB was not found. Another example is the pattern of LBBB in the limb leads with RBBB in the precordial leads. None of this type was found in the present series. The pattern described by Lenegre, marked left axis deviation with RBBB in the precordial leads, was observed twice. When bundle-branch block patterns occur in complete heart block, one or the other bundle can be presumed to be involved. These patterns on occasion alternate, but in this situation an idioventricular focus may be present below the main bundles and, therefore, may not indicate bundle-branch block per se. When the QRS pattern in complete heart block is narrow, the bundles are functionally competent.

References


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The Heart as a Symbol

Man's heart can harden; it can become inhuman, yet never nonhuman. It always remains man's heart. We are all determined by the fact that we have been born human, and hence by the never-ending task of having to make choices. We must choose the means together with the aims. We must not rely on anyone's saving us, but be very aware of the fact that wrong choices make us incapable of saving ourselves.

Indeed, we must become aware in order to choose the good—but no awareness will help us if we have lost the capacity to be moved by the distress of another human being, by the friendly gaze of another person, by the song of a bird, by the greenness of grass. If man becomes indifferent to life there is no longer any hope that he can choose the good.—ERICH FROMM: The Heart of Man: Its Genius for Good and Evil. New York, Harper & Row, Publishers, 1964, p. 150.
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