INCOMPLETE BUNDLE BRANCH BLOCK

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Received April 1, 1944

Lewis (1925) stated that delay in the passage of the impulse down a bundle branch could alter materially the form of the electrocardiogram. Wilson (1940) defined *incomplete* bundle branch block as due to a conduction defect that merely retards the passage of the impulse through one of the main bundle branches, and stated that it gives rise to electrocardiograms which are "transitional, both as regards the length of the QRS interval and the form of the ventricular deflections between those that represent complete bundle branch block and those of normal outline."

Partial bundle branch block is used to describe curves that contain both bundle branch block and normal complexes. There may be a rhythmic sequence, as in 2 : 1 bundle branch block, or occasional normal QRS deflections may occur in a record predominantly composed of bundle branch block complexes.

In common with the New York Heart Association's nomenclature, Wilson (1944) regards 0.12 sec. as the minimum QRS duration ordinarily compatible with the diagnosis of *complete* bundle branch block. In this country, however, a QRS that exceeds 0.10 sec. has generally been considered as indicating complete bundle branch block providing other criteria are present (Lewis, 1925; and Hunter, Papp, and Parkinson, 1940). This small divergence may be due to a different technique in measurement. In the cases to be described the QRS has been measured straight across at the widest point in any lead. In no case has it measured more than 0.10 sec., when incomplete bundle branch block has been judged to be present.

Case I. A man, aged 58, was admitted to hospital in April 1940 with a history of paroxysms of tachycardia lasting up to 24 hours during the last six months. The B.P. was 220/120. The heart was enlarged to the left. A diastolic murmur of aortic incompetence was audible. The W.R. was negative. He had no paroxysms while in hospital.

After discharge he was seen at intervals as an out-patient, when he said he was well and free from attacks. It was later learnt from his wife that he had had many paroxysms but did not wish to re-enter the hospital.

On December 27, 1940, he reported recent nocturnal dyspnoea and oedema of ankles. The pulse was 110 with premature contractions. The B.P. was 230/150. The heart was enlarged to left and right. There was a small effusion at the base of the left lung. The liver was enlarged and tender. The blood urea was 98 mg. per 100 c.c. He was admitted and was given 0.5 mg. digoxin intravenously with 0.5 mg. orally, followed by 0.25 mg. six-hourly. On December 30 the pulse was 160. Following carotid sinus pressure it fell abruptly to 80. At the same time the ward sister reported that she was doubtful how much digitalis he was retaining since he spat out the tablets whenever he could. On the following morning he was worse. The pulse was again 160: the oedema had increased; he was orthopnoic. It was decided to give a further dose of 0.5 mg. digoxin intravenously under electrocardiographic control. Twenty minutes after the injection he was clearly suffering from digitalis poisoning with constant retching and diarrhoea. He was given atropine and morphine but he died the same night.

At *autopsy* (Dr. S. Wray) the heart was greatly enlarged due to hypertrophy of the left ventricle. There was gross atheroma involving particularly the anterior descending branch of the left coronary, which was almost occluded. The mitral and aortic valves showed atheromatous changes on the cusps with some fibrotic contraction of the left posterior aortic cusp. There was gross atheroma of the aorta. The kidneys were granular.

A section of the myocardium supplied by the anterior descending branch shows hypertrophy of the muscle bundles with loss of staining power and cellular outline. The findings suggest that the

hypertrophied muscle has been receiving an inadequate supply of blood due to the arterial atheromatous changes.

Electrocardiograms taken prior to his final admission show that the T waves in leads I and II varied from being upright to a coved inversion. (Fig. 1). This may have been due to variations in

FIG. 1.—Case 1. (A) 22/7/40. T I and T II upright. (B) 11/11/40. Bowed inversion of T I and T II.

the blood supply down the anterior descending branch or possibly may be related to the paroxysms of tachycardia. Fig. 2 taken on the day of his last admission shows a ventricular rate of 116. The rhythm is normal except for auricular premature systoles, some having aberrant ventricular responses. These aberrant responses are not confined to the auricular premature systoles but follow normal P waves at the end of lead I. The fourth complex in lead I, also following a normal P wave, has a

FIG. 2.—Case 1. 27/12/40. Auricular premature contractions with aberrant ventricular responses. Aberration present also after normal P waves at the end of lead I. The fourth complex in lead I, following a normal P wave, has a deep S wave.

deep S wave. Fig. 3 shows a nodal tachycardia with a rate of 140. Fibrillary waves can be seen, especially in lead III. On account of difficulties with the light the film was thin and another was taken immediately after (Fig. 4). This shows the same nodal tachycardia with fibrillary waves but the duration of the QRS of the alternate complexes in lead I has increased to 0.09 sec. Fig. 5 was taken 15 minutes after the intravenous injection of 0.5 mg. digoxin. The ventricular rate is now 194. Alternation of the complexes is present in leads I and III, giving the appearance of a bi-directional ventricular tachycardia, but the QRS is nowhere more than 0.09 sec. The alternate complexes in lead I are similar to the isolated complex seen in Fig. 2.

FIG. 3.—Case 1. 30/12/40. Nodal tachycardia at a rate of 140. Fibrillatory waves deforming the S-T period are visible in leads II and III.

FIG. 4.—Case 1. 30/12/40. Nodal tachycardia at a rate of 132, fibrillatory waves being seen in lead II. The alternate complexes in lead I have the QRS widened to 0.09 sec.

Comment. Aberrant ventricular responses appeared first following both auricular premature beats and normal auricular beats. One of these had a deep S wave, and was interpreted as intermittent incomplete right branch block. During the early stages of the nodal tachycardia the duration of the QRS of alternate complexes increased to 0.09 sec. without any significant alteration in their shape. Finally the direction of the alternate complexes changed to that of the isolated complex of incomplete right branch block. This gave the appearance of a bi-directional ventricular tachycardia with a QRS duration of only 0.09 sec., but was due to a partial (2 : 1) incomplete right branch block.

Case II. A man, aged 60, was seen first in 1938 on account of dyspnoea on exertion. The pulse was 84; B.P., 144/84. He weighed 14 st. 6 lb. The heart was enlarged to left and right, and he had mitral incompetence. Signs of moisture were present at the bases of the lungs. He was advised to reduce his weight and his activities.

FIG. 5.—Case 1.—31/12/40. Nodal tachycardia at a rate of 194, with bidirectional ventricular responses (QRS, 0.09 sec.). Alternate complexes in lead I are similar to the isolated complex of Fig. 2. Partial (2 : 1) incomplete right branch block.

FIG. 6.—Case 2. 28/5/38. Small Q and notching of R in lead II (QRS, 0.09 sec.). Lead III, QRS "W" shaped.

In 1940 the size of the heart had increased further and digitalis was commenced in the form of Guy's pill.

In 1942 the auricles began to fibrillate and from that time he required frequent courses of neptal with ammonium chloride to avert congestive failure.

In June 1943 he suffered a cerebral embolism with temporary aphasia and an extensor plantar response on the right side. In the autumn of that year he died suddenly.

The first electrocardiogram taken in 1938 had a Q and a notched R in lead II, the QRS being 0.09 sec. In lead III the QRS is also 0.09 sec. and the deflections are "W" shaped (Fig. 6). The comment was made at the time that bundle branch block might soon supervene. This proved incorrect and the curve remained unchanged, except for the addition of many ventricular premature contractions arising singly and in couples. In 1942, after the onset of fibrillation, the ventricular complexes were virtually unaltered, but in lead III the first, third, and last complexes are quite normal (Fig. 7). Complexes of this type were never seen at any other time. In 1943 a slight widening of the QRS in lead I was observed, although the duration in that lead was still only 0.08 sec. Otherwise there was no change, but "V" leads taken at the same time show a QRS of 0.12 sec. in V.2 with a

FIG. 7.—Case 2. 1/3/42. Auricular fibrillation. Basic complexes unchanged. First, third, and last complexes in lead III normal.

FIG. 8.—Case 2. 22/5/43. Leads I, II, and III virtually unchanged. V leads: left bundle branch block. (QRS, 0.12 sec.) Unipolar limb leads show heart in normal position.

FIG. 9.—Case 2. 13/6/43. Paroxysmal left bundle branch block in lead I (QRS, 0.14 sec.).

shape characteristic of left branch block (Fig. 8). Two months later—after the cerebral embolus—a paroxysm of left branch block was recorded with a QRS of 0.14 sec. (Fig. 9).

Comment. The normal complexes seen in 1942 (Fig. 7) indicate that his usual curves were due to delayed conduction down a bundle branch. In the following year the “V” leads had the shape of a left branch block with a QRS of 0.12 sec. (the equivalent of 0.11 sec. in limb leads), and a paroxysm of left branch block was recorded two months later.

Case III. A woman, aged 81, was admitted to hospital in November 1941 on account of abdominal pain which had lasted a week. She had been breathless for some months and more recently had been orthopnoëic. The pulse was 120 and irregular from auricular fibrillation. The B.P. was 100/80. The heart was enlarged to the left: there were no murmurs. The liver was enlarged and tender. The bases of the lungs were congested. The blood urea was 113 mg. per 100 c.c. The white cells numbered 28,000.

She was given 1 grain of digitalis folia, four times daily, and three days later the rhythm returned to normal. Subsequently she developed a cerebral embolism with a left-sided facial palsy, from which she did not recover.

The cardiogram taken on 7/11/41 shows auricular fibrillation with a ventricular rate of 175. The ventricular complexes have the shape of a left bundle branch block but the QRS is only 0.10 sec.

FIG. 10.—Case 3. 7/11/41. Auricular fibrillation with ventricular rate of 175. Incomplete left branch block. (QRS, 0.10 sec.)

FIG. 11.—Case 3. 10/11/41. Left axis deviation. Diphasic T in lead I.

(Fig. 10). Three days later only left axis deviation is present, the QRS duration having returned to normal with the resumption of normal rhythm (Fig. 11).

Comment. The first cardiogram conforms in all respects to a left bundle branch block except that the QRS is insufficiently widened. It was interpreted at the time as indicating advanced left axis deviation or possibly incomplete left bundle branch block. The first alternative was negated by the second record.

Case IV. A man, aged 69, was admitted to hospital in 1938 on account of dyspnoëa, cardiac asthma, and œdema. The B.P. was 200/110. The auricles were fibrillating. The heart was greatly enlarged to left and right. The blood urea was normal. The W.R. was negative.

He was given digitalis and injections of salyrgan with ammonium chloride, but the congestive failure was only partially relieved and he died three months later.

Several cardiograms were taken showing auricular fibrillation and inversion of T in leads I and II. In one record (Fig. 12) intermittent bundle branch block complexes occurred in leads I and II: their shape is of Type A right branch block with a QRS of 0.10 sec. A fortnight later intermittent branch block complexes were again seen in leads I and II: these had the shape of the wide SI pattern and the QRS was 0.12 sec. (Fig. 13).

Comment. The intermittent right branch block is unstable since the shape of the complexes changes. The QRS duration of 0.10 sec. in those of the first record is insufficient for complete bundle branch block.

Case V. A man, aged 74, was admitted to the surgical side of the hospital on 8/7/42 with retention of urine. He had spent the greater part of the last two years in bed on account of shortness of breath

FIG. 12.—Case 4. 10/8/38. Auricular fibrillation with diphasic T waves in leads I and II. First complex in leads I and III and second in lead II have the shape of a "Type A" right bundle branch block with QRS of 0.10 sec.

FIG. 13.—Case 4. 24/8/38. Third complex in lead I and second in lead II wide SI pattern of right bundle branch block. (QRS, 0.12 sec.)

and œdema of the legs. He had been given digitalis, but the dosage is not known. The pulse was 100 and irregular from auricular fibrillation. The B.P. was 130/90. The heart was greatly enlarged to right and left. There was œdema of the legs. The blood urea was 57 mg. per 100 c.c. The leucocytes numbered 18,000. A supra-pubic intubation (Mr. Pearce) was performed on 27/7/42. On August 1, since the rate had increased, 1 grain of digitalis folia, t.d.s. was started. This was reduced a fortnight later to 1 grain daily, and he was discharged free from œdema in September.

The first cardiogram taken on 21/7/42 (Fig. 14) shows auricular fibrillation with a partial left bundle

FIG. 14.—Case 5. 21/7/42. Auricular fibrillation with ventricular rate of 100. Partial left bundle branch block. (QRS, 0.12 sec.) Complexes with normal QRS duration being present in all leads. B.B.B.I. complexes vary in shape.

branch block (QRS, 0.12 sec.). There is considerable variation in the shape of the ventricular complexes, and the first complex in lead I and the third in lead II together with some in lead III have QRS deflections of normal duration. In Fig. 15 (18/8/42) the QRS deflections have all a normal duration with the exception of the first two in lead I in which the duration is 0.10 sec. Auricular fibrillation persists with left axis deviation. By 10/9/42 normal rhythm has returned and the ventricular complexes have assumed a form intermediate between the two preceding curves with a QRS duration of 0.12 sec. (Fig. 16).

Comment. The QRS deflections in the first cardiogram varied considerably in shape apart from the normal complexes. When the rate was slowed with digitalis the duration of the QRS became normal but transitional complexes (QRS, 0.10 sec.) occurred. Finally bundle branch block returned, having a form intermediate between the general shape of the first record and the left axis deviation of the second.

Case VI. A woman, aged 74, was admitted to hospital on 11/1/44 under the care of Dr. Glen Reah, who kindly afforded facilities for observation. The auricles were fibrillating. The B.P. was

FIG. 15.—Case 5. 18/8/42. Auricular fibrillation. Left axis deviation with inversion of T in lead I and depression of S-T in lead II. The QRS of the first two complexes in lead I is 0.10 sec.

FIG. 16.—Case 5. 10/9/42. Normal rhythm. Left bundle branch block. (QRS, 0.12 sec.) Shape of complexes differs from those in Fig. 14.

FIG. 17.—Case 6. 13/1/44. Auricular fibrillation with ventricular rate of 160. Left bundle branch block (QRS, 0.11 sec.)

100/60. The heart was enlarged to the left, and there was mitral incompetence. Crepitations were present at the bases of both lungs, and the liver was enlarged and tender. Since she had received no digitalis for the previous fortnight, it was decided to obtain a rapid effect by means of digitalis lanata. Accordingly 8 c.c. of cedilanid was given intravenously and slowing occurred within thirty minutes. Four days later normal rhythm returned, and two days later she suffered an embolism into her right leg. From this she made a satisfactory recovery without embolectomy.

The first electrocardiogram (Fig. 17) shows auricular fibrillation with a ventricular rate of 140. Left bundle branch block is present with a QRS duration of 0.11 sec. Fig. 18 taken 35 minutes after

FIG. 18.—Case 6. 13/1/44. Lead I, 35 minutes after cedilanid, 8 c.c. intravenously. QRS now normal except first complex.

cedilanid shows a ventricular rate of 104, and the QRS deflections have returned to normal, except the first in the lead. Fig. 19 (17/1/44) shows the return of normal rhythm. All the ventricular complexes, with the exception of the last two in lead III (which resemble the normal complexes shown in Fig. 20),

FIG. 19.—Case 6. 17/1/44. Lead I. Nodal rhythm at rate of 100: no evidence of auricular activity: incomplete right (wide SI pattern) B.B.BI. (QRS, 0.10 sec.) Lead II. Nodal rhythm at 96: P waves deforming the S-T interval of alternate complexes. Incomplete right B.B.BI. Lead III. P waves move forward from S-T till S-A node takes control at fifth complex when QRS returns to normal.

FIG. 20.—Case 6. 20/1/44. Normal rhythm. Normal QRS. Digitalis depression of S-T.

are of the wide SI type of right branch block but the QRS does not exceed 0.10 sec. In lead I there is no evidence of auricular activity, the rhythm arising from a centre in the A-V node with a rate of 100. In lead II inverted P waves can be seen deforming the S-T interval of alternate complexes. In lead III the rate has fallen to 90 and P waves can be made out in each complex. The first deforms the S-T interval, the second is almost buried in the QRS: then P moves forward until the auricle gains control of the rhythm in the fifth complex. At that point the ventricular complexes change and become normal. Later the curve reverted to partial left branch block (QRS, 0.12 sec.), normal complexes being present in leads II and III (Fig. 21). Then the duration of the QRS increased to 0.14 sec.,

FIG. 21.—Case 6. 22/2/44. Partial left bundle branch block. (QRS, 0.12 sec.) Normal QRS follows premature systole in lead II. Normal complex also seen in lead III.

FIG. 22.—Case 6. 24/2/44. Left bundle branch block (QRS, 0.14 sec.). Transitional complex (QRS, 0.10 sec.) follows compensatory pause in lead I.

and Fig. 22 shows a transitional complex with a QRS of 0.10 sec. after the compensatory pause of a premature ventricular systole. Later again the ventricular complexes became normal.

Comment. This case shows unusual instability of the bundle branch block. The following changes were noted. Left branch block (QRS, 0.11 sec.): normal deflections after intravenous digitalis; incomplete right branch block (QRS, 0.10 sec.) shortly before the resumption of normal rhythm: normal QRS deflections; partial left branch block (QRS, 0.12 sec.): left branch block (QRS, 0.14 sec.) with a transitional complex (QRS, 0.10 sec.): normal QRS deflections.

DISCUSSION

In *complete* bundle branch block the speed of conduction down one branch of the bundle is so much slower than down the other that the impulse passes across the septum to activate the ventricle on the affected side. In *incomplete* bundle branch block the difference between the two sides is less. The affected ventricle may receive the impulse partly from across the septum and partly via its own branch, or else, when the difference is slight, wholly via its own branch though with some delay. Alternatively there may be delay in conduction down both branches caused by "a general depression of the conductivity of the Purkinje tissue" (Wilson *et al.*, 1944) and the delay may be approximately equal on both sides. Such cases might be expected to show a slight increase in the duration of the QRS with minimal changes in the shape of the complexes, and nothing to show a predominant lesion in either branch. These changes are sometimes seen in the aberrant ventricular responses that often follow premature auricular systoles, or are found in auricular tachycardia or flutter (Slater, 1930).

There are thus three groups: those with bilateral bundle branch delay, shown by a widened QRS without axis deviation (Case 1); those with delay in either right or left branch (Cases 1, 2, 3, 4, and 6); those showing a combination of the two (Cases 5 and 6).

In Case 1 slightly widened QRS deflections occurred first after auricular premature systoles, and later at every alternate beat. There was little alteration in the shape of these complexes and they are likely to be due to delay in conduction down both branches.

The last curve of Case 1 showed a bidirectional type of tachycardia with alternate complexes pointing downwards, and the same shape was seen earlier in an isolated complex following a normal P wave. Bidirectional tachycardia has been recorded in digitalis poisoning by Schewenson (1922) and by Luten (1925). Luten suggested that the rhythm originated from a point near the bifurcation of the main stem, and that there was impairment of conductivity down each branch alternately. However, the sequence of events in Case 1 all point to delay down the right branch only.

In Cases 3 and 4 and in the episode of right branch block in Case 6 there is no doubt as to the side affected. The curves conform in every particular to left and right branch block respectively except that the duration of the QRS is 0.10 sec. Had not normal complexes been present, either in the same record or a few days later, they might have been interpreted as showing advanced axis deviation and ascribed to hypertrophy of the muscle. This type of incomplete branch block may, therefore, be more common than is supposed. In Case 2 the standard leads, although abnormal, did not indicate any axis deviation, but the chest leads showed delay down the left branch.

Transitional complexes provide the most reliable evidence of incomplete bundle branch block, since, in the absence of complete A-V block with independent ventricular centres, they cannot be due to anything else. They are rare, being found in only two of the thirteen cases of paroxysmal bundle branch block studied by Comeau, Hamilton, and White (1938). But the branch block in Cases 5 and 6 was unstable, both showing partial branch block at times. Case 6 had no fewer than five changes in the duration of the QRS, which ranged from 0.08 to 0.14 sec. A similar example was recorded by Herrmann and Ashman (1930). In Case 5 the branch block complexes varied in shape in the first cardiogram, and differed again after the resumption of normal rhythm.

The duration of the QRS deflections in these transitional complexes was 0.10 sec. and, especially in Case 6, they did not show much axis deviation. The transitional complex in Case 6, too, occurred during a phase of left branch block (QRS, 0.14 sec.) and followed an episode of incomplete right branch block (QRS, 0.10 sec.). They are, therefore, probably due to the combined effect of a bilateral delay, accounting for some of the widening of the QRS, with additional delay down one branch leading to a moderate axis deviation, the proportion of each varying in different cases.

SUMMARY

Six cases of incomplete bundle branch block have been described. In none did the duration of the QRS exceed 0.10 sec. when incomplete bundle branch block was judged to be present. In all of the cases normal complexes have been present for comparison, either in the same record or within a short period.

The evidence suggests that the cases could be divided into three groups.

The first shows a slight increase in the QRS without axis deviation as exemplified by the aberrant ventricular response to an auricular premature systole. These are probably due to a bilateral delay down each main branch (Case 1).

The second shows delay down one branch, fulfilling the criteria for bundle branch block except that the QRS does not exceed 0.10 sec. (Cases 1, 2, 3, 4, and 6).

The third shows transitional complexes (Cases 5 and 6). In these cases it is likely that the transitional complexes were due to a combination of bilateral delay down each main branch with additional delay down one branch, since both cases had an unstable branch block which sometimes changed from right to left, and there was not much axis deviation although the QRS duration was 0.10 sec.

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