The Changing Electrocardiogram in Wilson Block*

By Cornelio Papp, M.D. and K. Shirley Smith, F.R.C.P.

The significance of the Wilson variety of right bundle-branch block was investigated on the basis of serial electrocardiographic changes seen in almost half of the series. Wilson block may originate from both anteroseptal and posterior infarction; in the former it may form a solitary relic without prognostic significance; in the latter it denotes extensive septal involvement. It may modify the injury pattern of anterior infarction and may abolish the signs of posterior infarction; cardiac infarction may approximate it to the classic form of right bundle-branch block. Except in two patients, less than 4 per cent of the series, it was related to pathologic causes; it should therefore be regarded as a sign of organic heart disease.

Right bundle-branch block has of late years lost much of the sinister reputation which it used to have, and which still attaches to left bundle-branch block. Numerous authors have emphasised the harmlessness of the variety of right bundle-branch disorder first defined by Wilson, Johnston, Hill, Macleod and Barker in 1934. Bundle-branch block, whether right or left, is often regarded as a stable electrocardiographic pattern and we have been surprised to find that in almost half of our patients showing the Wilson type of right bundle-branch block, the record has at one time or another changed. It seemed likely that a study of such serial changes might help to solve some of the problems connected with this disorder of conduction, especially its relation to coronary disease. In this paper, we present the results of a review of 53 patients showing the Wilson pattern of right bundle-branch block. In particular, we have sought to establish the diagnostic and prognostic meaning of changes in the electrocardiogram in this condition, and to emphasise the fact that only rarely is it without morbid significance.

Long before it was proved to be due to right bundle-branch block, Oppenheimer, Rothschild and Mann emphasised the good prognosis in a special form of intraventricular block where the widening and notching of the ventricular complex was "entirely confined to the terminal portion of the second limb of the R wave." Their impression, gained in 10 patients, was reaffirmed by Van Desten and Dolganos and has been since endorsed in a number of papers.

In Wood, Jeffers and Wolferth series of 64 patients showing this pattern, only 29 had definite signs of heart disease. In the largest series ever published, that of Dry, Willius and Reeser, only 22 per cent of 492 cases had angina or congestive heart failure when the lesion was discovered. Although most of these patients were in the arteriosclerotic age group, 62 lived 10 years or more. There was a high mortality of 20 per cent during the first year because of the association with cardiac infarction, but after this, the life expectancy curve tended to parallel that of the normal population. These authors advocated a separate group for the "wide S" variety of bundle-branch block because of its relatively benign significance when "unattended by coexistent findings indicative of organic heart disease." Similar conclusions were reached by Perera, Levine and Erlanger and by Schulze, who also denied the prognostic significance hitherto attached by Franke to the width of the S-wave. Out of a series of 281 patients observed by Shreenivas, Messer, John-

From the Cardiac Department of Charing Cross Hospital and of the London Chest Hospital, and the private files of the authors.

* The wide S wave pattern of bundle branch block is here called Wilson block. This term is used to acknowledge the debt we all owe to Frank N. Wilson who with his associates first studied this electrocardiographic pattern which he was able to reproduce in dogs by section of the right branch of the bundle—and to substitute for an eight word description a two word designation. The term Wilson block in this context has been in common use in Europe for many years.
son and White, \(16\) 27 per cent lived longer than five years; but this proportion rose to 39 per cent in 186 patients who could be traced. The survival time after the first year was, however, only slightly better as compared with left bundle-branch block, (5.7 and 4.9 years respectively), possibly because of the greater mortality of the right bundle-branch block patients in the younger age group in which this was mostly associated with rheumatic heart disease.\(^{11}\) If the series in which right bundle-branch block was associated with coronary heart disease were analyzed separately, as was done by Vazifdar and Levine\(^{12}\) in 1952, the prognosis in right bundle-branch block was distinctly better than in left bundle-branch block. They found also that among 31 patients with bundle-branch block who were alive and well with no other sign of heart disease 5 to 21 years after the electrocardiographic diagnosis of bundle-branch block, 27 had right and only 4 left bundle-branch block. Most authors agree that bundle-branch block, whether left or right, has little prognostic significance, if not associated with clinical heart disease\(^ {13, 14, 15}\) and that the causative heart disease and particularly cardiac enlargement are the determining factors in prognosis. It is, however, established that the Wilson type of right bundle-branch block is the one which more often occurs as an isolated electrocardiographic abnormality. Series of normal population are more convincing in this respect than the pathologic series published from cardiac clinics.

Among 100 electrocardiograms with bundle-branch block in an insurable group collected by Rodstein, Gubner, Mills, Lovell and Ungerleider,\(^ {16}\) 77 had right and 23 left bundle-branch block, while among 83 uninsurable patients with cardiac conditions the proportion was 65 and 35 per cent respectively. This difference in the series as a whole is not relevant. But there was a much greater incidence of right bundle-branch block in the younger age group, almost all insurable; out of 30 persons below the age of forty, 27 had right bundle-branch block, while left bundle-branch block increased with advancing age. Difference in mortality between right and left bundle-branch block was not considerable, but the mortality of the insurable group was similar to that of the population as a whole. Unfortunately, no account was taken of the type of right bundle-branch block. Among 29 members of the United States Navy with bundle-branch block electrocardiograms and no associated heart disease, 18 has a wide S pattern right bundle-branch block, and this was the case in four out of six in whom the abnormality was known to have existed for 8 to 19 years. Langley, Red and Utz,\(^ {17}\) who published the series forming part of 100 cases of bundle-branch block among 6900 members of the navy, emphasized the favorable prognosis of the Wilson type of right bundle-branch block. Fisch\(^ {18}\) reported 11 cases of the same type in patients without any evidence of heart disease, whose electrocardiograms were taken mainly as routine studies. Seven out of these 11 were aged 19 to 35 years. Wolfram\(^ {19}\) collected 52 patients with bundle-branch block and without significant heart disease; of these 35 had right, 17 left bundle-branch block. Out of 35 with right sided block 26 had no evidence of organic heart disease.

In a series of 6132 cases comprising 4387 cardiac patients and 1745 normal subjects, Taimont, Carouso, Mège and Lenègre\(^ {20}\) found the Wilson-type block in 0.74 per cent of the normal subjects, in 2.25 per cent of those with left-sided heart disease, in 2.14 per cent of those with right sided heart disease and in 0.9 per cent of those with both ventricles affected. Its incidence in coronary and arterosclerotic heart disease was 4 per cent. Lepeschkin\(^ {20}\) estimated that from the clinical point of view about 40 per cent of the patients with “wide S” type of right bundle-branch block were apparently normal, and 50 per cent had no cardiac enlargement. In those with cardiac lesions, coronary or hypertensive heart disease was found in 60 to 70 per cent, valvular heart disease in 8 to 10 per cent, congenital heart disease in 1 to 5 per cent and acute or chronic cor pulmonale in the remainder. In incomplete right bundle-branch block the incidence is different, according to Michaelides, Costeas, Vitsakakis and Lekos\(^ {21}\); valvular and congenital heart disease accounted for about 40 per cent, arteriosclerotic and hypertensive heart disease for 18 per cent, chronic cor pulmonale for 16 per cent, while
there was no heart disease in 23 per cent of their series or in 50 per cent of the series of Masini, Testoni and Farulla 22.

The absence of clinical heart disease in many patients with right bundle-branch block has been explained by the anatomic peculiarities of the right branch of the bundle. Compared with the left one, which fans out broadly soon after its emergence from the fibrous septum, the right branch has a long isolated course until it branches out to the free wall of the right ventricle. The superficial situation beneath the septal endocardium and its unique blood supply by the septal branches of the left coronary artery make it particularly vulnerable in right ventricular dilatation, localized septal myocarditis, chronic coronary artery disease and acute septal infarction. To explain the presence of right bundle-branch block in patients under the age of 40, Vazifdar and Levine 32 suggested that it might be the residue of some harmless intercurrent virus infection involving the myocardium, while Goldberger 23 even denied its pathologic significance and regarded it merely as a physiologic variation of conduction. The rare instances in which transient right bundle-branch block appeared under sympathetic overstimulation 24 or following startle reaction 25 give some support for this contention.

Faced with an electrocardiogram showing a Wilson type of right bundle-branch block, the physician may make one of the following interpretations: (1) that it represents only a functional variation of the conducting system; (2) that it has limited diagnostic significance, being the relic of an extinct process, myocardial or coronary, involving the right branch; (3) that it has diagnostic significance denoting an acute condition injuring the right branch, such as a circumscripted or extensive septal lesion; that it is due to right ventricular hypertrophy.

Evidently, the decision rests with the other findings, clinical, radiologic and electrocardiographic, showing the presence and extent of the cardiac affection. If the patient is under 40 and the heart otherwise normal, the electrocardiographic signs will carry little diagnostic or prognostic value. In patients over 50, in the same circumstances, some doubts may arise, particularly in connection with life insurance risks.

If, on the other hand, a cardiac lesion coexists, the difficulties of interpretation will be considerable, particularly in patients with anginal pain where right bundle-branch block may be as much a sign of recent infarction as of a healed lesion.

In the hope of solving at least some of these problems we have investigated the diagnostic and prognostic significance of the changing electrocardiogram in the Wilson variety of right bundle-branch block.

Material

Fifty-three consecutive patients with records of Wilson (wide S) type of right bundle-branch block, in whom more than one record was available, were collected. The selection was based on the usual criteria of a QRS complex at least 0.11 second wide in which a slender R of normal voltage preceded a wide slurred S wave in lead I or II, and where a secondary R wave with a delay of at least 0.07 second was recorded in right ventricular leads. Our cases, therefore, belong to group II and III of Bayley's 26 classification; some difficulty was experienced in the selection of cases with cardiac infarction or right ventricular hypertrophy where R1 may become lower than S1 is deep. If there was doubt, the patient was included in the series, provided V1 or V2 confirmed the existence of right bundle-branch block. The cases fell into two main groups: group A comprising 33 patients with known episodes of cardiac infarction; group B comprising 20 patients in whom no infarction had taken place.

Group A included six patients with increasingly severe angina in whom it could be assumed on clinical grounds that infarction had occurred, though their records showed only right bundle-branch block. No previous normal records were available for comparison in four patients. Out of these 33, the electrocardiogram changed in 19. The changes were from normal QRS to persistent right bundle-branch block, from infarction pattern to transient or persistent right bundle-branch block or the reverse, and the various combinations of infarction patterns added to right bundle-branch block and

| TABLE 1.—Age and Sex in 53 Patients with Wilson Type of Right Bundle-Branch Block |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| ?i              | 11-19 | 20-40 | 50-60 | 60-69 | 70-79 | Total |
| Males . . . . . . | 1   | 6    | 10   | 13   | 15   | 45    |
| Females . . . . . | —   | 2    | 2    | —    | 4    | 8     |
| Proportion of men to women = 6:1 |
their regression. Out of 20 patients in group B serial electrocardiographic changes were found in four. They included transient right bundle-branch block and the addition of A-V block to right bundle-branch block. More than half of the patients in the whole series were over 60 years of age. There were six times as many men as women (table 1).

RESULTS

Group (A). Thirty-three Patients with Known Cardiac Infarction

Table 2 summarizes the findings in this series. Slight cardiac infarction, where cardiac pain is the only feature, and severe cardiac infarction where shock and failure dominate the picture, produce right bundle-branch block in equal proportion, and this is even more the case if the infarction is anterior. When right bundle-branch block is associated with posterior infarction the lesion is almost always severe. The six patients with increasingly severe angina, in whom there were no additional electrocardiographic changes, were listed as having slight infarctions. The survival period was calculated to the end of the second month after infarction; no follow-up study is included.

In table 3 are listed the electrocardiographic serial changes in 19 patients of group A, those with infarction. Right bundle-branch block was an early manifestation of cardiac infarction in 15; in four of these it was intermittent with normal complexes or with left bundle-branch block (cases 3, 9, 14 and 5). It did not abolish the signs of anterior infarction and these regressed in most instances, leaving right bundle-branch block with flat or inverted T waves in some of the left ventricular leads. It was transient in three patients where normal QRS (cases 6, 14 and 16) with infarction pattern appeared temporarily or became the stable pattern. In case 13 it developed gradually through the pattern of partial right bundle-branch block. In Case 16 it regressed gradually, leaving a pattern of partial right bundle-branch block within 6 to 14 months of anterior infarction. In case 8 with septal infarction, right bundle-branch block appeared the day before death. In two (cases 7 and 18) with clinical infarction, the right bundle-branch block pattern did not change; in both of these, normal electrocardiograms were available for comparison. In case 18, the right bundle-branch block took many years to develop through the intermediate stage of partial right bundle-branch block. It was found one year before infarction, and remained unchanged for months after it. Two and a half years later the right bundle-branch block disappeared, leaving a record of left ventricular strain (fig. 1). The three patients in whom normal electrocardiograms were available before right bundle-branch block developed all showed S waves in lead I or II, whether the heart was horizontal or vertical. No right ventricular leads were available to decide whether this was already the expression of partial right bundle-branch block.\(^7\) The addition of A-V block to right bundle-branch block was seen once in antero-septal and twice in posterior infarction, all severe. The following examples illustrate our findings.

Severe Septal Infarction and Right Bundle-Branch Block. The first record shown in figure 2 was taken a few hours after infarction accompanied by loss of consciousness and profound shock which lasted until death three days later; it shows subendocardial ischemia in the lateral areas of the left ventricle (leads aV\(_l\) and V\(_6\)) with reciprocal changes in aV\(_R\) (acute septal ischemia) (fig. 2A, case 8). The second record taken two days later and one day before death, shows right bundle-branch block with antero-septal infarction, best seen in V\(_2\), V\(_3\) and V\(_4\), where notched and wide Q waves of 0.06 mm. precede the secondary R wave (fig. 2, B). The RS-T pattern in V\(_2\) is the one repeatedly observed in other cases of right bundle-branch block with infarction; it consists of an RS-T period strictly level and isoelectric which passes abruptly into an inverted V-shaped ter-

<table>
<thead>
<tr>
<th>Severity</th>
<th>No. of cases</th>
<th>Site of infarction</th>
<th>Sev. ang.</th>
<th>Alive</th>
<th>Dead</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slight</td>
<td>16</td>
<td>Ant. - lat. Post. Ant. post. Sep.</td>
<td>6</td>
<td>15</td>
<td>1</td>
</tr>
<tr>
<td>Moderate</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Severe</td>
<td>15</td>
<td>6</td>
<td>6</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>33</td>
<td>14</td>
<td>2</td>
<td>6</td>
<td>24</td>
</tr>
</tbody>
</table>
TABLE 3.—Serial Electrocardiographic Changes in Right Bundle Branch-Block with Infarction (19 Patients)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>Severity</th>
<th>Site</th>
<th>First ECG.</th>
<th>Interm. ECG.</th>
<th>Final ECG.</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 8.</td>
<td>73</td>
<td>F</td>
<td>severe</td>
<td>sept.</td>
<td>subendo. isch.</td>
<td>—</td>
<td>R.B.B.B.; ant. lat.</td>
<td>died. P.M.</td>
</tr>
<tr>
<td>Fig. 2.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>regression infarct pattern</td>
<td>R.B.B.B.</td>
<td></td>
</tr>
<tr>
<td>Case 1.</td>
<td>69</td>
<td>M</td>
<td>moderate</td>
<td>ant.</td>
<td>ant. sept.</td>
<td>R.B.B.B.</td>
<td>R.B.B.B.</td>
<td>died 2½ yrs. later in C.H.F. P.M.</td>
</tr>
<tr>
<td>Case 2.</td>
<td>63</td>
<td>M</td>
<td>moderate</td>
<td>ant.</td>
<td>ant. sept.</td>
<td>R.B.B.B.</td>
<td>—</td>
<td>regressing infarct pattern L.B.B.B.</td>
</tr>
<tr>
<td>Case 5.</td>
<td>67</td>
<td>M</td>
<td>severe</td>
<td>ant.</td>
<td>ant. sept.</td>
<td>R.B.B.B.</td>
<td>—</td>
<td>died 1 month later in attack. recov.</td>
</tr>
<tr>
<td>Case 10.</td>
<td>70</td>
<td>M</td>
<td>slight</td>
<td>ant.</td>
<td>ant. sept.</td>
<td>R.B.B.B.</td>
<td>—</td>
<td>same recov.</td>
</tr>
<tr>
<td>Fig. 4.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>regression infarct pattern</td>
<td>R.B.B.B.</td>
<td>recov.</td>
</tr>
<tr>
<td>Case 13.</td>
<td>77</td>
<td>F</td>
<td>slight</td>
<td>ant.</td>
<td>ant. QRS normal lat.</td>
<td>—</td>
<td>partial R.B.B.B.</td>
<td>well</td>
</tr>
<tr>
<td>Fig. 5.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>R.B.B.B.</td>
<td>normal QRS; ant. sept. later; R.B.B.B.</td>
<td>recov.</td>
</tr>
<tr>
<td>Case 11.</td>
<td>77</td>
<td>M</td>
<td>slight</td>
<td>ant.</td>
<td>altern. R.B.B.B. and normal QRS.</td>
<td>—</td>
<td>normal QRS; flat T1</td>
<td>recov.</td>
</tr>
<tr>
<td>Case 14.</td>
<td>42</td>
<td>M</td>
<td>severe</td>
<td>ant.</td>
<td>intern. R.B.B.B. and P-R + T, inv. normal</td>
<td>—</td>
<td>same normal 4 yrs. later</td>
<td>well</td>
</tr>
<tr>
<td>Case 17.</td>
<td>54</td>
<td>M</td>
<td>slight</td>
<td>?</td>
<td>normal 1949.</td>
<td>R.B.B.B. 1951</td>
<td>R.B.B.B. 1951; same after inf.</td>
<td>well</td>
</tr>
<tr>
<td>Fig. 10.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>regressing infarct pattern</td>
<td>R.B.B.B.</td>
<td>recov.</td>
</tr>
<tr>
<td>Case 7.</td>
<td>77</td>
<td>M</td>
<td>severe</td>
<td>post.</td>
<td>R.B.B.B.</td>
<td>R.B.B.B.; P-R 0.4 sec.</td>
<td>R.B.B.B. only (3 wks. later)</td>
<td>recov.</td>
</tr>
<tr>
<td>Fig. 9.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>—</td>
<td>R.B.B.B. only (3 wks. later)</td>
<td>recov.</td>
</tr>
<tr>
<td>Case 6.</td>
<td>54</td>
<td>M</td>
<td>severe</td>
<td>post.</td>
<td>QRS normal. P-R normal.</td>
<td>—</td>
<td>post.; QRS normal; P-R 0.2</td>
<td>died 2 wks. later</td>
</tr>
<tr>
<td>Case 8.</td>
<td>54</td>
<td>M</td>
<td>severe</td>
<td>post.</td>
<td>R.B.B.B. ant. sept.</td>
<td>—</td>
<td>R.B.B.B. only (3 wks. later)</td>
<td>recov.</td>
</tr>
<tr>
<td>Fig. 7.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>—</td>
<td>R.B.B.B.</td>
<td>recov.</td>
</tr>
</tbody>
</table>

C.H.F. = Congestive heart failure.

Thus, the usual signs of acute myocardial injury seem absent, for the RS-T period is not elevated or humped. The right bundle-branch block is here not of the Wilson type, for R₁ has a small voltage; but this is due to the acute injury to the left ventricle and the absence of opposing left ventricular potentials. (c.f. the minute R in V₇). There is complete heart block. At necropsy (fig. 3) there was a recent infarct in the interventricular septum and the adjacent portion of the anterior and posterior wall of the left ventricle. The anterior part of the septum was thinner than the posterior part and the ventricle was here beginning to dilate. The right atrium and ventricle were dilated. The remainder of the myo-
cardium showed diffuse patchy fibrosis. Severe calcified atheroma in both coronary arteries and their main branches considerably reduced the vascular lumen, but no occluding thrombus could be found.

Slight Anteroseptal Infarction and Right Bundle-Branch Block. The records of case 10, a patient with hypertensive heart disease, aortic incompetence and gradually worsening angina, portray the combination of right bundle-branch block, anteroseptal infarction and left ventricular hypertrophy (fig. 4). The Q waves in V1 and V2 may not be due to septal necrosis. The initial R is absent because the heart is enlarged and V1 faces the right auricle (P in V1 is inverted). The depth of Q is determined by left ventricular hypertrophy; in fact a minute R appears in V2. The subepicardial infarction is here shown by the RS-T and T deformities in V1, V3, V4 and their successive regression. The first record again shows, in V1 and V2, the abrupt terminal T inversion from

---

**Fig. 1.** Case 18. A man, age 61. Partial right bundle-branch block at the age of 39 (A). Complete Wilson-type block at the age of 47 when hypertension was found (B). Record unchanged during three years while clinical cardiac infarction occurred (C). Disappearance of Wilson-type block and pattern of left ventricular strain (D).

**Fig. 2.** Case 8. A woman, age 73. Subendocardial infarction (A), followed by Wilson-type block and anteroseptal infarction two days later, one day before death (B).
a horizontal RS-T level, which in successive records changes into the gradual R-T slope which imperceptibly fuses with an inverted T and so becomes the pattern of bundle-branch block and not that of an infarction.

Case 16 had a solitary bout of pain lasting half an hour without clinical or laboratory signs of infarction; this was followed during subsequent weeks by occasional attacks of angina. The electrocardiogram showed right bundle-branch block only (fig. 5A). One month later the record showed normal QRS with T inversion in V₁, V₂, V₃ and V₄, the pattern of anteroseptal subepicardial infarction (fig. 5B); successive records at one and three months showed a reversion to right bundle-branch block pattern, but T in V₄ became flat (fig. 5C). The record five months later showed restoration almost to normal except for V₁, where a secondary R wave, 0.08 second after the beginning of QRS, proved, that incomplete right bundle-branch block still existed (fig. 5D). One year after infarction the Wilson block reappeared with upright T waves in V₂ and V₃ (fig. 5E).*

*This patient had a recent anterior transmural infarction in the area of the previous subepicardial infarct, from which she recovered.
Fig. 5. Case 16. A woman, age 56. Wilson-type block following slight cardiac infarction (A) and (C), alternating with anteroseptal pattern (B). Regression of infarction pattern, partial right bundle-branch block remaining in V1 (D). Wilson-type block with recovery of T waves in V2 and V3, one year after infarction (E).

Fig. 6. Case 13. A woman, age 77. Anterolateral infarction (A) with electrocardiographic recovery (B). Wilson-type block nine months after infarction (C).
The records of case 13 show this development in reverse. Here, too, cardiac infarction six weeks previously was followed by sporadic attacks of angina. The first record of figure 6 showed the pattern of extensive anterolateral, subepicardial injury from V2 through V6, with corresponding appearances in aVF (fig. 6A). The next record revealed the regression of these signs, but with simultaneous development of incomplete right bundle-branch block in V2 (fig. 6B). The third record, made nine months after the first showed complete right bundle-branch block which has remained the stable pattern since (fig. 6C).

While in figures 4 and 6 some ischemic relic remained months after infarction (flat or inverted T waves in left ventricular leads), the record of case 19 showed restoration to pure Wilson block pattern three weeks after slight anterior infarction (fig. 7A and B).

Severe Posterior Infarction and Right Bundle-Branch Block. Case 6 was admitted to a hospital with cardiac pain which had been persisting for more than two days. The first record in figure 8 showed posterior infarction (A); the second record (B) showed only right bundle-branch block with latent heart block; the deep Q3 and the inversion of T3 were in keeping with the Wilson block; the only sign significant of infarction was the RS-T depression in V5. The Wilson type block did not reappear in subsequent records (C). The patient died 16 days after admission.

Some of the arrhythmias which may be associated with right bundle-branch block in severe posterior infarction are illustrated in fig-
Fig. 9. Case 7. A man, age 70. Wilson-type block with complete A-V block in extensive posterior infarction (A). Wilson variety of block with 2:1 flutter (B). Sinus rhythm and pattern of posteroanterior infarction (Vr-V6) denoting septal involvement (C).

Fig. 10. Case 15. A man, age 60, with slight posterior infarction in association with pre-existing Wilson form of block. S wave in lead I with horizontal heart suggests incomplete right bundle-branch block in 1949, no right ventricular leads recorded (A). Wilson variety of block the day of clinical infarction; tall T in V3 suggests posterior infarction (B). R-T elevation in leads II, III and aVF confirms posterior infarction (C). Regression of acute changes, flat T in V6 (D).

The electrocardiogram on the day of admission in this critically ill patient, who eventually recovered, showed complete A-V block with acute posterior infarction and right bundle-branch block (fig. 9A). Sinus rhythm was restored the next day, but three weeks later he developed auricular flutter (fig. 9B). The record five weeks after admission again showed sinus rhythm; the infarct had then extended to the anterior wall by extensive septal involvement (posteroanterior infarction) (fig. 9C).

Slight Posterior Infarction and Right Bundle-Branch Block. There was only one solitary bout of moderate pain in this hypertensive and obese patient (case 15) who is considered under the heading and whose tracings are shown in figure 10. He had slight pyrexia for one day and no further attacks of chest pain followed. An electrocardiogram recorded three years previously showed an S1 with horizontal heart; this suggests partial right bundle-branch block though no right ventricular leads were then recorded to confirm this (fig. 10A). The electrocardiogram a few hours after the attack showed a Wilson block, but nothing suggesting recent in-
and they were listed as having chronic cor pulmonale. Out of the remaining five, lone auricular fibrillation was present in two, and paroxysmal flutter in one, while two had no heart disease to account for the bundle-branch block.

The serial electrocardiographic changes in this group are summarized in table 5. In three out of four at one time or another, during their protracted illnesses, A-V block was recorded and two of them were in congestive heart failure. In case 35, who had fainting attacks suggesting the Stokes-Adams syndrome, no A-V block could be recorded, though it is known that normal A-V conduction may exist be-

Table 4.—Right Bundle-Branch Block Without Cardiac Infarction in 23 Patients

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of cases</th>
<th>Card. enl.</th>
<th>Cong. heart fail.</th>
<th>No. of cases</th>
<th>A-V block.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.S.H.D. &amp; H.H.D.</td>
<td>9</td>
<td>7 5 2 2</td>
<td></td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>M.S.</td>
<td>2</td>
<td>2 2 1 1</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>A.S.</td>
<td>1</td>
<td>1 1 1 1</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>A.S.D.</td>
<td>1</td>
<td>1 1 1 1</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Chron. Cor pulm.</td>
<td>2</td>
<td>2 2 2 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lone aur. fibr. &amp; flutter</td>
<td>3</td>
<td>3 3 3 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No H.D.</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

A.S.H.D. = arteriosclerotic heart disease; H.H.D. = hypertensive heart disease; M.S. = mitral stenosis; A.S. = aortic stenosis; H.D. = heart disease; A.S.D. = atrial septal defect.

Table 5.—Electrocardiographic Changes in Right Bundle-Branch Block without Infarction in Four Patients

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Card. enl.</th>
<th>Electrocardiographic changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 35.</td>
<td>73</td>
<td>M</td>
<td>A.S.</td>
<td>L.V.++</td>
<td>R.B.B.B.; normal P-R</td>
</tr>
<tr>
<td>Fig. 11.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Normal QRS L.V. strain</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>C.H.F.</td>
<td>R.B.B.B. &amp; C. A-V B.</td>
</tr>
<tr>
<td>Case 40.</td>
<td>43</td>
<td>F</td>
<td>M.S.</td>
<td>sl. no</td>
<td>2:1 A-V.H.B. normal QRS; v. rate 40</td>
</tr>
<tr>
<td>Fig. 12.</td>
<td></td>
<td></td>
<td></td>
<td>C.H.F.</td>
<td>R. &amp; L.B.B.B.; P-R 0.3 sec</td>
</tr>
<tr>
<td>Case 52.</td>
<td>77</td>
<td>F</td>
<td>A.S.H.D.</td>
<td>Gross C.H.F.</td>
<td>R.B.B.B.; C. A-V B.</td>
</tr>
</tbody>
</table>

A.S. = aortic stenosis; H.H.D. = Hypertensive heart disease; M.S. = Mitral stenosis; A.S.H.D. = arteriosclerotic heart disease; L.V. = left ventricle; C.H.F. = Congestive heart failure; A-V H.B. = Atrioventricular heart block; C. A-V B. = Complete atrioventricular block.

...fraction (fig. 10B). On the third day of illness, a 1 mm. RS-T elevation was seen in leads II, III and aVp (fig. 10C). The suspicion of posterior infarction was confirmed by the appearance of an increasing height of T waves in V1, V2 and V3 in later records when the posterior signs were regressing (fig. 10D). In the last electrocardiogram T1 and TavV1 became flat.

Group B. Right Bundle-Branch Block without Infarction

The relevant findings in these 20 patients are contained in table 4. Arteriosclerotic and hypertensive heart disease was present in nine, and in these cardiac enlargement of various degree was an almost constant feature, with congestive heart failure in five. Three had valvular, and one congenital, heart disease; in two, right bundle-branch block was associated with chronic bronchitis and advanced emphysema, between attacks when Stokes-Adams seizures are associated with bundle-branch lesions. The cardiogram of right bundle-branch block was frequently replaced by normal QRS patterns (fig. 11), the P-R interval remaining normal. In case 40, who had mitral stenosis with little cardiac enlargement and no failure, the transient right bundle-branch block was governed by the ventricular rate (fig. 12). The critical rate at which right bundle-branch block ap-

![Fig. 11. Case 35. A man, age 73 with calcareous aortic stenosis. Transient Wilson block (A), alternating with QRS of normal duration and left ventricular strain pattern (B) at the same rate.](image-url)
Fig. 12. Case 40. A woman, age 44, with mitral stenosis. A-V block (2:1) with normal QR8 at a ventricular rate of 38 (A). Wilson form of block with normal A-V conduction at a rate of 68 (B).

Discussion

The electrocardiographic signs of cardiac infarction associated with right bundle-branch block have been thoroughly investigated in recent years by Somerville and Wood,99 and Dressler, Roesler and Schwager.11 While left bundle-branch block obliterates the signs of infarction in more than half of the cases, in right bundle-branch block these signs could be identified in 93 per cent of the patients. The published cases, however, almost all belonged to the severe category where Q waves and RS-T elevation deformed the pre-existing right branch block pattern. How far right bundle-branch block may represent cardiac infarction when patterns representing such a lesion are not present in the cardiogram has remained unknown. The pathologic findings in most cases are also unhelpful, for they conform to the earlier conclusions of Master, Dack and Jaffe,22 that there is no correlation between the vessels occluded or the location of the infarct and the type of conduction defect. Right bundle-branch block may even be associated with infarcts which do not reach the anatomic site of the right-bundle branch.23 In the earlier histologic reports,24, 25 the right bundle-branch block was only part of an extensive septal lesion which involved the left branch and the main bundle as well. It was only proved recently that an isolated interruption of the right branch of the bundle may be the only septal abnormality in Wilson block.26 But still there is a contrast between pathologic studies which demonstrate a severe lesion in its final phases and the clinical observations which show the initial abnormality remaining unchanged for decades without the patient deteriorating.

The appearance of the Wilson type of right bundle-branch block as a transient phenomenon in "slight" anteroseptal infarction,28 as in case 16, its gradual development following such a lesion as in case 13, and its sudden appearance without any additional signs of infarction in patients with anginal pain, as in case 17, shed light upon at least some of the cases. The right branch of the bundle is mainly supplied by the anterior perforating branches of the left coronary artery which is the artery more often affected. Since slight anteroseptal infarctions are not due to local arterial occlusions but to narrowing of the main coronary branch,27 it is understandable how ischemia in this locality may involve this structure. The additional electrocardiographic signs of infarction, if present, are those of slight cardiac infarction though somewhat modified. Q waves in V1 or V2 may be present or absent, but T waves will always be inverted in most anterior chest leads; the RS-T elevation may not appear since the addition of the current of injury to the initial RS-T depression of the bundle-branch block pattern may result in an RS-T period strictly at a level and followed by a V-shaped terminal T inversion. As electrocardiographic recovery in these forms of infarction may be complete (case 19, fig. 7) or almost so (case 16, fig. 5), the right bundle-branch block may remain without the added signs of infarction, or with flat T waves in left lateral leads only, as in three instances in our series. Rodriguez and Sodi-Pallares28 have shown that in dogs almost the whole of the thickness of the septum is activated by the left bundle branch. In right bundle-branch block


the delay in the activation of the right septal surface takes place in a relatively small area of 1.5 to 2 mm. thickness under the right septal endocardium in the lower third of the septum. In anteroseptal infarction, even if slight, this area may be easily damaged temporarily by edema, or permanently by scar formation, giving rise to right bundle-branch block, the presence of which does not make the prognosis any worse.

Right bundle-branch block was associated with slight posterior infarction only once in our series (case 16), and here it may have previously existed (fig. 10A). Its association with posterior infarction in five other cases, four of them with A-V block, alternating right and left bundle-branch block and posteroanterior infarction, proved that the extensive septal involvement reached the anterior third of the right septal area where the right bundle-branch is situated. Thus, the appearance of Wilson block in posterior infarction is a sign of increased severity. The signs of posterior infarction may be abolished by the coexistence of right bundle-branch block if the electrical axis is horizontal and a deep Q3 is present; the addition of A-V block to right bundle-branch block in the presence of clinical signs of infarction (fig. 8B) is then a conclusive sign that extensive posterior infarction has taken place.

More than half of the patients in the group without cardiac infarction had enlarged hearts. Cardiac enlargement was slight in four and gross in six, and of these, five were in congestive heart failure. The electrocardiographic changes in these consisted in the appearance of right bundle-branch block in addition to the pre-existing Wilson block, and were thus similar to the changes found in severe anterior or posterior septal infarction. The only difference was that these changes developed slowly and persisted for years; they were due to progressive septal fibrosis, which is part of the generalized myocardial sclerosis in these patients.

Transient right bundle-branch block (figs. 11 and 12) was seen in two patients with valvular heart disease. In the one with calcareous aortic stenosis there was gross cardiac enlargement and arteriosclerotic heart disease as well; in the other, a woman of 43 with mitral stenosis, cardiac enlargement was slight and there was no failure. Though the appearance of right bundle-branch block was here governed by the heart rate, in view of the existing rheumatic lesion the sympathetic influence must have been of secondary importance.

Sandberg, Wener, Master and Scherlis, advocate right and left carotid pressure, exercise tests, inhalation of amyl nitrite and of 100 per cent oxygen for 20 minutes, various respiratory maneuvers and changes of posture to convert intermittent or transient bundle-branch block into normal intraventricular conduction or vice-versa. In their cases of right bundle-branch block their results were at least doubtful. Since transient and intermittent right bundle-branch block may be caused by just as serious heart disease as established right bundle-branch block, it is difficult to see the practical importance of carrying out these tests.

In nine patients the heart was found to be of normal size. Two were hypertensive and over 60 years of age; two had severe emphysema and chronic bronchitis. The diagnosis of right ventricular enlargement in its early stages is notoriously difficult, and we thought that right bundle-branch block might be the sign of this. They were listed as chronic cor pulmonale. Of the remaining five, two had lone auricular fibrillation, and one had paroxysmal flutter. There were only two patients in our series in whom Wilson block persisted as the only cardiac abnormality: a man, aged 62, in whom this was discovered 19 years previously, and a youth of 18. This is an incidence of less than 4 per cent. Admittedly, most of our patients were of arteriosclerotic age, but our material was not selected and comprised a consecutive series of patients seen in hospital, consulting and general practice. We therefore believe that the Wilson variety of right bundle-branch block without organic heart disease is rare. The myocardial condition which causes it may be slight or severe, extinct, quiescent or active. Its significance must be assessed on the basis of the clinical, radiologic and electrocardiographic findings. Studies of the serial changes in the electrocardiogram in 43 per cent of our patients provided us with information on the severity
and extent of myocardial damage and proved an important factor in assessment.

**Summary**

Out of a series of 53 consecutive patients with the Wilson variety of right bundle-branch block, 23 (43 per cent) showed serial changes in the electrocardiogram. These were investigated to establish their diagnostic and prognostic significance.

Clinical episodes of cardiac infarction occurred in 33, and electrocardiographic serial changes were found in 19 of these. These included changes from normal QRS to persistent right bundle-branch block, from infarction pattern to transient or persistent right bundle-branch block, or the reverse, and the various combinations of infarction pattern added to right bundle-branch block and their regression.

The Wilson variety of right bundle-branch block may be the only sign of slight anteroseptal infarction. This was proved in four patients where the added infarction signs regressed within weeks or months, leaving either a pure right bundle-branch block pattern or such added equivocal signs as flat T waves in one or more left ventricular leads. Conversely, in two cases, right bundle-branch block developed gradually within 6 to 14 months out of an anteroseptal infarction pattern. The involvement of the right bundle-branch in slight anteroseptal infarction can be explained by its anatomic proximity and by its unique blood supply from the left coronary artery. The addition of Wilson block to slight anteroseptal infarction does not make the prognosis worse.

In slight posterior infarction, the right branch of the bundle is hardly ever involved; in severe posterior infarction, right bundle-branch block suggests extensive septal involvement, which is proved by its frequent combination with A-V block, left bundle-branch block and posteroanterior infarction. Here it is of serious prognostic significance.

Anterior infarction may modify the pattern of the Wilson variety of right bundle-branch block; the diminution of left ventricular potentials may transform it into the classic pattern of right bundle-branch block. Conversely, right bundle-branch block may also modify the signs of anteroseptal infarction by obliterating the RS-T elevation of acute injury. Acute posterior infarction may not show in Wilson block with a horizontal heart; the additional A-V block with clinical signs of infarction is here diagnostic.

There was no evidence of infarction in the remaining 20 patients. The serial changes in four of these consisted in the additional appearance of A-V block and temporary reappearance of a QRS of normal duration. These changes were observed in patients with arteriosclerotic heart disease and cardiac enlargement which accounted for almost half of this group. In those without cardiac enlargement, right bundle-branch block remained a stable pattern; it was here associated with minor heart disease, such as chronic cor pulmonale, hypertension, auricular fibrillation or paroxysmal flutter. One patient in whom the appearance of transient right bundle-branch block was governed by the ventricular rate had mitral stenosis as well. Only in two instances was there no sign of additional heart disease, and this was less than 4 per cent of the whole series. We believe, therefore, that the Wilson type of right bundle-branch block is a sign of organic heart disease, although the latter may be so slight as to escape detection.

**Summario in Interlingua**

Le signification del bloco de branca dextere typo Wilson eseva investigar super la base de cambiamentos serial del electrocardiogrammas de quasi 50 pro cento de un serie consecutive de 53 casos de iste disorde. Le bloco de Wilson pote haber su origine in infartos tanto anteroseptal como etiam posterior. In le infartos anteroseptal illo pote representar un relicto sin importancia prognostic. In infartos posterior illo indica un extense affectio septal. Illo pote modificar le configuration lesional del infarte anterior e supprimer le signos de infartos posterior. Infartos cardiac pote render lo simile al forma classic de bloco de branca dextere. Excepte in duo casos (i.e. in minus que 4 pro cento de serie), illo eseva invariabilemente connectite con causas pathologic. Ergo on debe considerar lo como un signo de morbo cardiac organie.
ACKNOWLEDGMENT

The authors are indebted to Miss Anne Smith for taking and mounting the electrocardiograms, and for her technical assistance.

REFERENCES


31. Dressler, W., Roessler, H., and Schwager, A.: The electrocardiographic signs of myo-


The Changing Electrocardiogram in Wilson Block
CORNELIO PAPP and K. SHIRLEY SMITH

Circulation. 1955;11:53-68
doi: 10.1161/01.CIR.11.1.53
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1955 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/11/1/53

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/