Right Bundle-Branch Block Associated with Left Superior or Inferior Intraventricular Block  
Associated with Acute Myocardial Infarction  

By Patrick J. Scanlon, M.D., Ray Pryor, M.D., and S. Gilbert Blount, Jr., M.D.

SUMMARY
Over an 11-year period, 28 instances of acute myocardial infarction and bilateral bundle-branch block were encountered. Twenty-two of these patients had right bundle-branch block and left axis deviation, and six had right bundle-branch block and block of the inferior radiation of the left bundle. The overall incidence of complete heart block in these 28 cases was 21%. In-hospital mortality for the whole group was 36%, whereas it was 33% for those patients who developed complete heart block. From these results it is concluded that for the patient with bilateral bundle-branch block and acute myocardial infarction the prophylactic insertion of a temporary transvenous pacemaker is not only warranted, but is probably indicated.

Additional Indexing Words:  
Bilateral bundle-branch block  
Myocardial infarction  
Complete heart block  
Intraventricular block  
Right bundle-branch block

So-called bilateral bundle-branch block in acute myocardial infarction has been described in a small number of cases and is said to carry a very poor prognosis. The cases of bilateral bundle-branch block so far reported have consisted primarily of right bundle-branch block associated with left axis deviation.

We report a larger group of patients with bilateral bundle-branch block and acute myocardial infarction. We include patients with impaired conduction through the inferior (posterior) intraventricular radiation of the left bundle branch as well as those with block of the superior (anterior) radiation (left axis deviation), with or without peri-infarction block.

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wave in lead aVL was taller than that in leads I and aVR. Superior peri-infarction block was diagnosed in the face of pathologic Q waves in leads I and aVL, with left axis deviation.

Criteria for the diagnosis of block of the inferior intraventricular radiation of the left bundle (IIVB) included a clockwise frontal plane vector with a mean axis of approximately 90°, small 0.02-sec Q waves in leads III and aVF, a middle-aged or older patient (>40 years) with a medium to heavy body build and no clinical evidence of any condition which might lead to right ventricular hypertrophy or enlargement. Inferior peri-infarction block was diagnosed when a clockwise loop was seen with pathologic Q waves in leads II, III, and aVF with a mean axis to the right of 60°.

Cases Studied

Our patients include all those seen with bilateral bundle-branch block associated with acute myocardial infarction during the years 1958 through 1969. They were taken from a total of more than 200 patients seen during that time with partial bilateral bundle-branch block (not necessarily related to infarction).

Some of these patients had evidence for the presence of bilateral bundle-branch block prior to the onset of their acute myocardial infarction; others had this pattern when first seen with acute myocardial infarction without knowledge of their previous electrocardiographic patterns.

Many of these patients were seen prior to the availability of a coronary care unit. Therefore, continuous monitoring was not performed in the entire group. Because of this occasional patient with bilateral bundle-branch block may have been missed.

The prophylactic use of a transvenous pacemaker was not employed in any patient, though some of those who developed complete heart block were paced.

Late follow-up is available in 14 of the 18 patients who survived for at least 6 weeks from the time of infarction. This ranged from 1 mo to 9 ½ years; four patients died during the first year of follow-up, and five were seen for at least 1 year following infarction.

Results

We found 28 cases which had bilateral bundle-branch block in association with acute myocardial infarctions. The majority of patients had right bundle-branch block and left axis deviation; two had peri-infarction block, and 20 had block of the superior intraventricular radiation of the left bundle branch (SIVB).

There were six patients with right bundle-branch block and block of the inferior intraventricular radiation, five of whom had inferior peri-infarction block. Only one patient had left bundle-branch block as part of this syndrome. The left bundle-branch block was temporary and replaced by right bundle-branch block and left axis deviation.

Right Bundle-Branch Block and Left Axis Deviation (Table 1)

In the group with right bundle-branch block and left axis deviation, there were 15 males and six females, with an age range of 54 to 88 years (mean, 71 years). C. W. suffered two acute myocardial infarctions which were associated with bilateral bundle-branch block, and so is included twice in our statistics as cases 12 and 13.

Electrocardiograms were available preceding their acute infarction for only six patients in this group. Three of these showed bilateral bundle-branch block already present, one showed left axis deviation, and two were normal. Only three patients had a history of previous myocardial infarction, one of whom was case 12 which was included twice. His first infarction was anterior in location, his second inferior. The location of the previous infarction is unknown in the other two patients with old infarcts.

The site of the myocardial infarction was not consistently of the anterior wall, as might be supposed in view of the blood supply of the blocked conduction pathways. The infarction was inferior or true posterior or both in six patients. Another two patients had evidence of both anterior and inferior wall infarction, and in three patients the site of infarction was uncertain. It could possibly be assumed that bilateral bundle-branch block was previously present in those patients with acute inferior infarction, as was known to be true in cases 13 and 16.

The most consistent electrocardiographic abnormality at the time of hospital admission was left axis deviation, which was present in 18 of 19 cases in which complete heart block was not already present. Right bundle-branch block alone was the presenting conduction
abnormality in only one patient. Left axis deviation and right bundle-branch block were present together at the time of admission in 11 patients, whereas right bundle-branch block developed at a later time in the remaining patients. Right bundle-branch block tended to be more intermittent and recurrent than left axis deviation. Left axis deviation tended to be more persistent than right bundle-branch block.

Complete heart block was present in three patients at the time of admission; in none of these patients was it permanent, lasting 3 weeks in two patients (cases 3 and 8) and only 1 day in one (case 11). In three other patients complete block developed at a later time. C. G. (case 5) developed complete heart block on the second hospital day; this persisted for 7 days; then conduction returned to a pattern of bilateral bundle-branch block. Case 19 (C.H.) developed complete heart block several hours after admission and died within an hour of the development of block in spite of an unsuccessful attempt at pacing. The sixth case of complete heart block (case 6, C.T.), was not documented though presumed on the basis of three Stokes-Adams syncopal attacks. The overall incidence of complete heart block in the face of right bundle-branch block and left axis deviation associated with acute myocardial infarction was 27%.

Only two patients underwent successful insertion of a transvenous pacemaker because of complete heart block. The pacemaker was used until complete heart block spontaneously disappeared in each patient. Each of these patients died subsequently, one at 6 weeks and one at 10 weeks following acute infarction. The immediate cause of death was uncertain in each case, and in neither was autopsy performed. The other four patients with complete heart block did not undergo pacemaker insertion. Three of these survived the acute infarction, though two died 5 and 8 mo later. One of these, case 13, had autopsy confirmation of his infarction. The sixth patient, mentioned previously, died in asystole shortly after the development of complete heart block. Autopsy was not performed.

Of the 22 patients with right bundle-branch block and left axis deviation, eight died within 6 weeks of the time of infarction, a mortality rate of 36%. Four of these patients had postmortem examination. In all of these there was diffuse myocardial fibrosis and coronary artery disease, though no acute infarction was detectable in two patients who died within 24 hours of hospital admission. Detailed studies of the conduction system were not performed in any of this group. The site of infarction did not appear to affect mortality significantly, as four of 12 (33%) with anterior infarction, and four of nine (44%) with inferior infarction died within 6 weeks of the acute episode.

Follow-up information was available on 14 of those 18 patients who survived beyond 6 weeks of the time of infarction. Duration of follow-up was less than a year in nine patients, four of whom died during that period. One patient survived with bilateral bundle-branch block for 2 years, then died with a second infarction. The longest known survivor developed Stokes-Adams attacks 9 years following his infarction; during the 9-year period he had persistent bilateral bundle-branch block. His syncopal episodes were relieved following the insertion of a permanent transvenous pacemaker.

**Right Bundle-Branch Block and Inferior Intraventricular Block**

A much smaller group of patients had right bundle-branch block and block of the inferior intraventricular radiation of the left bundle (table 2). All but one of these six patients had inferior peri-infarction block. There were five males and one female, with an age range of 47 to 79 years (mean, 61 years). Three patients in this group had electrocardiographic evidence of old inferior myocardial infarction and right bundle-branch block before the acute infarction which prompted their inclusion in this study. The site of acute infarction was anterior in two of these three and inferior in the other. The site of infarction in the remaining patients was anterior in one and inferior in two.

*Circulation, Volume XLII, December 1970*
### Table 1

<table>
<thead>
<tr>
<th>Case no. &amp; initials</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Previous ECG</th>
<th>Old MI</th>
<th>Site of present infarction</th>
<th>Admission ECG</th>
<th>3^e block</th>
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<td>1</td>
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<td>F</td>
<td>–</td>
<td>–</td>
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<td>+</td>
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<tr>
<td>2</td>
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<td>70</td>
<td>F</td>
<td>LAD</td>
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<td>3</td>
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<td>82</td>
<td>M</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>+</td>
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<td>4</td>
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<td>M</td>
<td>–</td>
<td>–</td>
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<td>+</td>
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<tr>
<td>5</td>
<td>C.G.</td>
<td>68</td>
<td>F</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>+</td>
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<tr>
<td>6</td>
<td>C.T.</td>
<td>54</td>
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<td>WNL (3 yr)</td>
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<td>H.H.</td>
<td>67</td>
<td>M</td>
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<td>–</td>
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<td>+</td>
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<tr>
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<tr>
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<tr>
<td>10</td>
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<td>77</td>
<td>M</td>
<td>–</td>
<td>Prob.</td>
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<tr>
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<td>M</td>
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<td>–</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
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<td>M</td>
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<td>–</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>13</td>
<td>C.W.*</td>
<td>63</td>
<td>M</td>
<td>RBBB, LAD, old ant. MI</td>
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<td>+</td>
<td>+</td>
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<tr>
<td>14</td>
<td>B.V.</td>
<td>60</td>
<td>M</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>+</td>
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<tr>
<td>15</td>
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<td>M</td>
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<td>16</td>
<td>E.N.</td>
<td>88</td>
<td>F</td>
<td>RBBB, LAD</td>
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<tr>
<td>17</td>
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<td>73</td>
<td>M</td>
<td>–</td>
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<td>M</td>
<td>RBBB, LAD</td>
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<td>+</td>
<td>+</td>
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<tr>
<td>19</td>
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<td>M</td>
<td>3 wk old inferior MI</td>
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<tr>
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<td>B.T.</td>
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<td>F</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>21</td>
<td>H.G.</td>
<td>65</td>
<td>M</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>+</td>
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<tr>
<td>22</td>
<td>J.R.</td>
<td>70</td>
<td>M</td>
<td>–</td>
<td>–</td>
<td>+</td>
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</tbody>
</table>

*Patient C. W. had two infarctions and was counted twice.

Abbreviations: MI = myocardial infarction; LAD = left axis deviation; WNL = within normal limits; temp. = temporary; SA = Stokes-Adams attacks; CAD = coronary artery disease.

Those patients with inferior peri-infarction block all had this pattern at the time of hospital admission for acute infarction. The patient with IIVB without peri-infarction block developed this pattern at a later time. Right bundle-branch block was present on admission in all but one case.

Complete heart block did not develop in any patient in this small group. No patient underwent insertion of a pacemaker. Two of our six patients (33%) died within 6 weeks of infarction. Postmortem study in one of these (case 28), who had had right bundle-branch block on admission and then developed IIVB subsequently, revealed massive infarction of the intraventricular septum and of the adjacent left and right ventricular myocardium.

A follow-up examination and electrocardiogram were available in all of the four survivors. One of these died suddenly 3 mo after infarction, secondary to a ruptured thoracic aortic aneurysm. The other three were followed for 9 mo, 30 mo, and 6 years, respectively. None of these were known to have another infarction or to develop complete heart block.
For the whole group there was a 21% incidence of complete heart block. The mortality rate in these six patients with complete heart block was 33% during the hospital period and 83% for the 12-mo period following infarction. The overall mortality for all patients with bilateral bundle-branch block and acute infarction was 36% during the first 6 weeks.

**Discussion**

It is now accepted that the combination of right bundle-branch block and left axis deviation is the most common form of bilateral bundle-branch block, and it is recognized that this combination is a fairly frequent forerunner of complete heart block (CHB). Lasser and associates found that complete heart block developed in 10% of their cases of this form of right bundle-branch block. In our own group of 160 patients the incidence of CHB was 13.7%.

Specialized conduction system studies have shown that the anatomic derangement responsible for the development of left axis deviation is a lesion of the anterior (or superior)
Table 2
Right Bundle-Branch Block with Inferior Peri-infarction Block or with Inferior Intraventricular Block

<table>
<thead>
<tr>
<th>Case no. &amp; initials</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Previous ECG</th>
<th>Old MI</th>
<th>Site of present infarction</th>
<th>Admission ECG</th>
<th>Conduction disturbance</th>
<th>Death</th>
<th>Necropsy</th>
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<td>Anterolateral</td>
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<td></td>
<td></td>
<td></td>
<td>Inferior</td>
<td></td>
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</tr>
</tbody>
</table>

**RBBB and IPIB**

23 J.F. 66 M RBBB, Old Inf. PIB Inf. + Old + + –
24 M.L. 47 F Inf. with PIB Yes + Old + + – Day 3 No
25 F.W. 79 M RBBB with Inf. MI Inf. + + – 2½ yr
26 C.S. 56 M – – – + + + – 3 mo No
27 M.G. 57 M – – – + + + –
28 T.B. 61 M – – – + + + – Day 18 Massive MI; septum, & adjacent LV & RV

**RBBB and IIVB**

*No conduction disturbances of 2°, 3°, and LBBB.

Abbreviations: Inf. PIB = inferior peri-infarction block; Inf. MI = inferior myocardial infarction.

...
broad as the inferior radiation. Therefore, a smaller area of involvement would be necessary to block conduction in the former than in the latter.

The second major consideration is the blood supply to the two radiations. The superior radiation is supplied by septal branches of the left anterior descending coronary artery. The right bundle branch is supplied by the same vessels. Therefore, interruption of blood supply via these vessels would be expected to cause damage in both conducting tissues. This probably explains the frequent association of RBBB and LAD as a form of bilateral bundle-branch block. The inferior radiation, however, is supplied by branches from either the right coronary artery or the circumflex branch of the left coronary artery and sometimes from both. Because of the more diffuse nature of its blood supply compared to that of the superior radiation, the inferior radiation would be ischemic less often. Also, because of their different sources of blood supply, the right bundle and the inferior radiation would be less often diseased concomitantly. When RBBB does occur with IIVB, more significant disease should be suspected, since presumably, more of the vascular supply to the heart is diseased.

This, however, was not the case in our study. Of those 22 cases of RBBB and LAD, only 11 had anterior myocardial infarction. Another eight had inferior or posterior wall infarction or both; two of these had had previous electrocardiograms, both showing right bundle-branch block and left axis deviation to be already present. If we assume that the other six also had had preceding right bundle-branch block, we might expect a higher mortality rate in this group, since in addition to anterior wall fibrosis (BBBB) there would be inferior wall infarction. This occurred with a 38% (three of eight cases) mortality in those with RBBB, LAD, and inferior infarction, and with a 27% (three of 11 cases) mortality in those with RBBB, LAD, and anterior wall infarction. However, this assumption may not be justified.

All but one of the patients with RBBB and IIVB had old or new inferior wall infarction. In the one exception the patient's massive septal infarction originally caused RBBB and later IIVB. Thus, we can see that a massive infarction secondary to disease of the anterior descending artery can result in block of the inferior radiation even though the latter is presumed to be supplied by one of the other coronary arteries.

The third reason for a difference of occurrence is that IIVB is less easily recognized than SIVB (LAD).

The usual incidence of complete heart block in acute myocardial infarction is about 5%. Our incidence of this complication with bilateral bundle-branch block was four times that rate (21%). Except for the small number of cases (six), it is difficult to explain the fact that none of our patients with RBBB and IIVB developed complete heart block. Theoretically, anterior infarction occurring in the face of RBBB and IIVB would make the patient more prone to have CHB than would inferior infarction, since the anterior infarction would be more likely to compromise the remaining conduction pathway (superior radiation).

Present in-hospital mortality for patients with acute myocardial infarction is of the order of 15 to 20%. Our mortality rate of 36% reveals that patients with bilateral bundle-branch block have a poorer prognosis than those without this conduction problem. Of interest is the fact that in those patients who developed complete heart block, mortality was essentially the same (33%) as for the whole group with bilateral bundle-branch block.

Godman and associates, in a recent report concerning the frequency and prognosis of bundle-branch block in acute myocardial infarction, included 21 patients with various forms of bilateral bundle-branch block. Eleven of these patients were similar to our group, in that they had RBBB and LAD. The other 10 patients had bilateral block of a type not included in our study. The incidence of complete heart block was 55% (six of 11 cases) for those with right bundle-branch block and...
left axis deviation, and 71% (15 of 21) for the whole group.

The difference in frequency of complete heart block between their study and ours might be related to the fact that they included patients who suddenly developed ventricular standstill as having complete heart block. This was not done in our series. However, this would not explain the difference in mortality between the two groups; we are unable to give a good explanation for this difference.

In their study Godman and associates used prophylactic pacing in 31 patients with bundle-branch block (not necessarily with BBBB), nine of whom developed complete heart block subsequently. Pacing was successful in all of these patients, though eight subsequently died. These authors reported a high incidence of pacemaker-induced arrhythmia and complications. They concluded that prophylactic pacemaker insertion was unwarranted in these patients.

Patients with bilateral bundle-branch block do seem to have more diffuse myocardial involvement than patients without it, and they may, therefore, die more readily in spite of complete heart block when it develops than do other patients with acute infarction. Our mortality rate of 33% for those who develop complete heart block, however, indicates that the majority will survive the acute phase of infarction. It seems, then, that prevention of asystole at the time of onset of complete heart block would give the majority of patients with this syndrome a greater chance for survival. Our incidence of pacemaker complication, primarily using the subclavian approach, has been very low. Thus, we feel that in patients with bilateral bundle-branch block in the face of acute myocardial infarction, prophylactic insertion of a temporary transvenous pacemaker is warranted and probably indicated.

References
Right Bundle-Branch Block Associated with Left Superior or Inferior Intraventricular Block: Associated with Acute Myocardial Infarction
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