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

Clinical Setting, Prognosis, and Relation to Complete Heart Block

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SUMMARY
In an 11-year period 209 cases of partial bilateral bundle-branch block were seen. These included patients with right bundle-branch block and either left superior or left inferior intraventricular block. The majority of patients had evidence of coronary artery disease or hypertension, though a significant number had no clinical evidence of heart disease. The majority of patients had follow-up ECG tracings, with an average follow-up for the whole group of about 2 years. The incidence of complete heart block was 14.4% (30 of 209). Complete heart block developed more than 10 years after the discovery of bilateral bundle-branch block in several patients. It is anticipated that with more complete and longer follow-up the incidence of complete heart block will be even higher.

Additional Indexing Words:
Bilateral bundle-branch block
Hypertension
Coronary artery disease

It is now well recognized that the combination of right bundle-branch block (RBBB) and left axis deviation (LAD) represents the most common form of "partial" bilateral bundle-branch block (BBBB) and that this combination is a fairly frequent forerunner of complete heart block (CHB). Another form of partial BBBB, the combination of RBBB and block of the inferior intraventricular radiation (fascicle) of the left bundle (IIVB), also known as left posterior hemiblock, is now being recognized as a real clinical entity and as another potential precursor of CHB.

In this paper we will review our experience with these two forms of BBBB.

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Received May 26, 1970; accepted for publication July 17, 1970.
Figure 1

Right bundle-branch block and left superior intraventricular block and subsequent complete heart block in a 66-year-old male with no apparent underlying heart disease.

Criteria for the diagnosis of block of the inferior intraventricular radiation (fascicle) of the left bundle (IIVB) included a clockwise frontal plane vector with a mean unblocked axis of approximately 90° and a terminal 0.04-sec, unblocked vector at 90° or slightly rightward, with small 0.02-sec Q waves in leads III and aVF. The patient was often middle-aged or older (> 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 (fig. 3).

Inferior peri-infarction block was considered to be a form of IIVB, though it will be considered independently in our statistics. It was diagnosed when a clockwise, unblocked frontal plane vector was seen with pathologic Q waves in leads II, III, and aVF, with a mean unblocked axis to the right of 60° (fig. 4).

From these definitions it is seen that patients with so-called incomplete RBBB were excluded, as were those patients with right ventricular hypertrophy manifested by an R' in the right precordial leads. Hopefully, all those patients with a counterclockwise loop and a superior and rightward terminal vector representative of right ventricular enlargement (sometimes called pseudo-left axis deviation) were also excluded. It is recognized that patients with IIVB are difficult to differentiate from normal persons and from patients with early right ventricular enlargement.

Cases Studied

All patients with bilateral bundle-branch block (BBBB) of either type described, who were seen initially at our hospital from 1958 through 1968 were included in the study.

All electrocardiograms interpreted as showing RBBB were extracted from the electrocardiographic files for 1958 to 1968, inclusive. The unblocked forces of each tracing for each of these patients were evaluated for LAD or IIVB. Because these latter diagnoses were unrecognized during the early years of the study, many tracings...
with abnormality of the unblocked forces had been originally interpreted as showing only RBBB.

We were able to extract a total of 209 instances of bilateral bundle-branch block: 147 of SIVB; 13 of superior peri-infarction block; 24 of IIVB; and 25 of inferior peri-infarction.

Results
Superior Intraventricular Block (SIVB) and RBBB

This was by far the most common type of BBBB discovered, there being 147 cases in all. There were 100 males and 47 females. The patients ranged in age from 4 mo to 92 years. Bilateral bundle-branch block occurred spontaneously in 129 patients, whereas it resulted from surgery in 18 of 22 patients with congenital heart disease with this conduction defect.

Etiology (Table 1)

The most common type of underlying heart disease in these patients was coronary artery disease. Fifty-three patients had clinical or electrocardiographic evidence of coronary artery disease or both. Twelve of these 53 had hypertension in addition to coronary disease; another 18 had hypertension without evidence of coronary artery disease. Eighteen of those with coronary artery disease or hypertension, or both had additional evidence of pulmonary disease (usually chronic obstruction), and another 12 had the latter alone. Diabetes was present in 18 patients, 11 of whom also had coronary artery disease. Obesity was common in all groups and was the only abnormality found in four patients with SIVB and RBBB. There were occasional patients with rheumatic heart disease (aortic stenosis in each instance), hyperkalemia, neuromuscular disorders, and myocardial disease.

Twenty-two patients had congenital heart disease. In only four of these 22 patients did BBBB develop unrelated to attempt at surgical correction of their defect. One of these was a 47-year-old woman with a secundum type of atrial septal defect who developed BBBB and then permanent CHB before her defect was closed. Another was a 28-year-old man with clinical evidence of a
ventricular septal defect without significant pulmonary hypertension. The third patient was a 15-year-old boy with congenital mitral insufficiency felt to be part of the endocardial cushion defect complex. The fourth case was that of a 10-year-old boy with a primum atrial septal defect.

Of the 18 patients with congenital heart disease with BBBB occurring subsequent to surgery, nine had LAD prior to surgery and developed RBBB with surgery (table 2). Four of these patients had ventricular septal defects, and five had endocardial cushion defects; two of the latter group had isolated primum type of atrial septal defect. The other nine patients developed both LAD and RBBB secondary to surgery. Three of these had closure of a ventricular septal defect, four had total repair of tetralogy of Fallot, one had closure of a primum atrial septal defect, and one had repair of an A-V communis type of endocardial cushion defect.

Of the whole group of 147 patients, 20 had no apparent underlying abnormality to explain the development of BBBB.

Follow-up

Only one follow-up tracing was available in 22 patients. Follow-up tracings were available in the remaining 125 patients, ranging from 2 days to 12 years, with an average period of 18.9 mo.

In most patients, both LAD and RBBB were discovered concomitantly. However, in 31 patients LAD was known to have appeared first, while in 11 RBBB preceded LAD. RBBB was more frequently intermittent than was LAD.

*Figure 3*

Right bundle-branch block and left inferior intraventricular block in a 60-year-old male with hypertension.
PARTIAL BILATERAL BUNDLE-BRANCH BLOCK

Figure 4

Right bundle-branch block and inferior infarction with peri-infarction block in a 62-year-old male with symptomatic coronary artery disease.

Complete Heart Block

Twenty of these 147 patients (13.6%) developed complete heart block. This was documented electrocardiographically for 18 patients, while two others had recurrent Stokes-Adams attacks. Eleven of these patients had coronary artery disease; one was just obese; one had myocarditis (thought to be related to mumps); three had congenital heart disease (CHB developed following surgery in two), and four patients were otherwise normal. In most cases CHB developed early in the follow-up period, though several patients had BBBB for more than 5 years before CHB occurred.

Deaths

Twenty-four of these 147 patients are known to have died, and 16 had postmortem examination. In each case necropsy confirmed the clinical diagnosis, though the conducting system was not thoroughly examined in any patient.

Superior Peri-infarction Block and RBBB (Table 3)

There were only 13 patients in this group, 10 males and three females. Their ages ranged from 50 to 98 years, with a mean of 69.1 years.

Seven patients had a clinical history of acute myocardial infarction. Another four had no good history for acute infarction but did have angina pectoris. One patient had generalized occlusive vascular disease without clinical evidence of heart disease, and one was just obese.

Complete Heart Block

Two patients in this group with superior peri-infarction block and RBBB developed
Table 1

Right Bundle-Branch Block and Superior Intraventricular Block (Acquired Spontaneously)

<table>
<thead>
<tr>
<th>Cardiovascular disease</th>
<th>No.</th>
<th>CHB</th>
<th>Paced</th>
<th>Deaths</th>
<th>Necropsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery disease</td>
<td>27</td>
<td>5</td>
<td>3</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>And hypertension</td>
<td>10</td>
<td>3</td>
<td>0</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension and pulmonary disease</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>And pulmonary disease</td>
<td>14</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>16</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>And pulmonary disease</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pulmonary disease</td>
<td>12</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes or obesity, or both</td>
<td>11</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Myocardial disease</td>
<td>6</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Hyperkalemia</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Neuromuscular disease</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Ventricular septal defect</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Secundum atrial septal defect</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Primum atrial septal defect</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Endocardial cushion defect</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Normal</td>
<td>20</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>129</td>
<td>18</td>
<td>10</td>
<td>18</td>
<td>11</td>
</tr>
</tbody>
</table>

Table 2

Right Bundle-Branch Block and Superior Intraventricular Block (Surgical)

<table>
<thead>
<tr>
<th>Cardiovascular disease</th>
<th>No.</th>
<th>RBBB</th>
<th>LAD</th>
<th>RBBB &amp; LAD</th>
<th>CHB</th>
<th>CHB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular septal defect</td>
<td>7</td>
<td>4</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Endocardial cushion defect</td>
<td>6</td>
<td>5</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Single ventricle</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td>9</td>
<td>0</td>
<td>9</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 3

Right Bundle-Branch Block and Superior Peri-infarction Block

<table>
<thead>
<tr>
<th>Clinical state</th>
<th>No.</th>
<th>CHB</th>
<th>Paced</th>
<th>Deaths</th>
<th>Necropsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical myocardial infarction</td>
<td>7</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Angina pectoris (no infarction)</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Generalized atherosclerosis</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Obesity</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>2</td>
</tr>
</tbody>
</table>

CHB (15%). A 74-year-old diabetic, with a history of myocardial infarction, was found to have CHB when first seen for evaluation of seizures. His condition reverted to first degree block and BBBB on pacing. The second patient developed BBBB with an acute infarction in 1960. Nine years later he developed CHB and Stokes-Adams attacks, and pacing was employed.

Deaths

During the follow-up period, which ranged up to 100 mo, four deaths occurred in this group. Autopsy was performed on two of
Partial Bilateral Bundle-Branch Block

Table 4
Right Bundle-Branch Block and Inferior Intraventricular Block

<table>
<thead>
<tr>
<th>Cardiovascular disease</th>
<th>No.</th>
<th>CHB</th>
<th>Paced</th>
<th>Death</th>
<th>Necropsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery disease</td>
<td>8</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>And hypertension</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Hypertension</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aortic valve disease</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Diabetes or obesity, or both</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Normal</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>24*</td>
<td>5</td>
<td>3</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

*One patient had coronary artery disease and aortic stenosis.

these patients. An old myocardial infarction with involvement of the conduction pathways was found in one patient. The second patient had severe coronary disease with diffuse myocardial fibrosis but without an area of infarction. Thus, the pattern of peri-infarction block does not always indicate previous infarction.

Inferior Intraventricular Block (IIVB) and BBBB (Table 4)

Of the 24 patients with this type of BBBB, 19 were males and five were females. They ranged in age from 39 to 90 years with a mean age of 61.9 years. In none of these patients was the BBBB surgically induced.

Etiology

Ten of these patients had clinical evidence of coronary artery disease, two of whom also had hypertension. Another four patients with hypertension alone were seen with this type of conduction delay. Three patients had rheumatic disease of the aortic valve. One of the group with coronary disease had calcific aortic stenosis. Two patients were obese, one of whom had diabetes. One patient was felt to have myocarditis. The remaining five patients had no apparent abnormality which might predispose to their BBBB.

Complete Heart Block

Five of these 24 patients developed CHB (21%). Two of the five had coronary artery disease, and both had Stokes-Adams attacks. One patient had probable viral myocarditis. He had a constantly changing pattern of conduction defects, including left bundle-branch block at times, and finally developed permanent CHB. Two patients had no known underlying heart disease. One of them had BBBB for 10 years before developing CHB. Insertion of a permanent transvenous pacemaker relieved the syncope of three patients. One of the two patients for whom insertion of a pacemaker was deferred died suddenly 2 days following his last cardiac evaluation.

Deaths

Follow-up in this group of patients was for as long as 120 mo, with a mean period of 25 mo. There were three deaths. Autopsy was not obtained in either the previously mentioned patient with CHB who was not paced or in a 55-year-old obese, diabetic male with a malignant schwanoma. The third death was that of a 61-year-old male whose BBBB developed following an acute anterolateral myocardial infarction. Postmortem examination revealed a massive acute infarction involving the interventricular septum and the adjacent right and left ventricular myocardium.

Inferior Peri-infarction Block and BBBB (Table 5)

Twenty-five patients were seen with this type of BBBB. There were 20 males and five females. They ranged in age from 47 to 83 years with a mean age of 69.1 years.

Eleven of the 25 had a clinical history of myocardial infarction. Another four had generalized occlusive vascular disease without clinical evidence of coronary artery disease. Three were hypertensives. One patient had diabetes without evidence of vascular disease,
and one patient's only problem was obesity. Five patients were clinically normal.

**Complete Heart Block**

Three of these 25 patients (12%) developed CHB. An 83-year-old man had BBBB for 7 years before developing third degree block. He was successfully paced. A 63-year-old woman with syncope was found to have BBBB and then CHB, but refused insertion of a pacemaker. The third patient, an 81-year-old female, had RBBB and SIVB for 2 years. Her pattern then changed to one of RBBB and inferior infarction with peri-infarction block during a period of chest pain. She died suddenly, presumably with CHB, the same day. Necropsy revealed no coronary disease but there was diffuse fibrosis involving the conduction system as well as the general myocardium.

**Deaths**

Follow-up in these 25 patients was for as long as 96 mo. There were six deaths. Three of these were unrelated to heart disease, occurring in patients with myeloid metaplasia, fat embolism, and polycystic renal disease, respectively. Two patients died suddenly, including the previously mentioned woman felt to have CHB secondary to diffuse fibrosis of her entire conduction system. The other sudden death occurred in a 76-year-old man with congestive heart failure. Postmortem examination was performed in four of these patients. Interestingly, no infarction was found in two of these, though both had diffuse myocardial fibrosis.

**Discussion**

Left axis deviation has received much attention the past few years. It is now appreciated that LAD is in most cases secondary to disease involving the superior (anterior) intraventricular radiation of the left bundle of His. The anatomy of this conduction pathway has been well described previously. Experimental studies in animals have shown that section of this pathway results in an electrocardiographic conduction abnormality similar to that seen as LAD in humans.

In humans, fibrosis or infarction of the superior radiation can result in left axis deviation. One can get a pattern of superior intraventricular block (as described above) or superior peri-infarction block, depending on the presence or absence of necrosis or infarction. As seen in this report, peri-infarction block may occur without infarction if myocardial fibrosis is diffuse.

The most common etiology of LAD is coronary artery disease. In a review of necropsy data on 353 patients with LAD, Bahl and associates found that 299 patients had pathologic evidence of coronary artery disease. They also found LAD in patients with pulmonary emphysema, rheumatic heart disease, amyloidosis, congenital heart disease, cardiomyopathy, collagen disease, and hyperkalemia. Thirteen patients had no evidence of cardiac abnormality.

Left axis deviation in patients with pulmonary emphysema has been felt to be secondary to associated, but clinically unrecognized, coronary artery disease. However, LAD may occur with pulmonary disease alone. In these latter cases, however, the mean and terminal frontal plane forces are

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**Table 5**

**Right Bundle-Branch Block and Inferior Peri-infarction Block**

<table>
<thead>
<tr>
<th>Clinical state</th>
<th>No.</th>
<th>CHB</th>
<th>Paced</th>
<th>Death</th>
<th>Necropsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial infarction</td>
<td>11</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Generalized atherosclerosis</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes or obesity</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Normal</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>25</td>
<td>3</td>
<td>1</td>
<td>6</td>
<td>4</td>
</tr>
</tbody>
</table>
frequently more superior and rightward than in LAD without lung disease; this has been called "pseudo-left axis deviation" implying the presence of an extreme form of right axis deviation suggesting probable right ventricular enlargement.\(^5\)

In a general hospital, left axis deviation occurs in combination with right bundle-branch block in approximately 1% of patients having an electrocardiogram for any reason.\(^1,3\) In most reviews, the combination of LAD and RBBB is most frequently seen in patients with coronary artery disease.\(^1,3,6,23\) The combination is also fairly frequent in patients with hypertension or aortic valve disease.\(^1\) However, Lenègre,\(^24\) in his review of the pathology of the conduction pathways in 62 cases of bilateral bundle-branch block, noted that the majority of his patients who developed CHB following a prolonged period of bundle-branch block had a sclero-degenerative process of unknown etiology involving both bundle branches, rather than ischemic heart disease.

Watt and associates\(^25\) have sectioned the right bundle branch with a knife and ligated the anterior fibers of the left bundle branch in both canines and primates. The electrocardiographic patterns created by these lesions were similar to that of RBBB and LAD as seen in man, thus giving experimental confirmation that this pattern does represent bilateral bundle-branch block.

In this series coronary artery disease was the most common cardiac problem encountered clinically, as it occurred in 41% of all patients with RBBB and LAD. Hypertension was also a common underlying problem. Again, it is difficult to explain the 12 patients with pulmonary disease alone who had RBBB and LAD; a special attempt was made to exclude those patients whose tracings might be interpreted as pseudo-left axis deviation. It is still likely that most of them had clinically unrecognized coronary artery disease.

We see a considerable amount of congenital heart disease at this hospital, which probably explains the high incidence of congenital lesions in this study. The LAD deviation in these patients either was secondary to surgery or occurred in those types of lesions whose anatomic arrangement somehow causes abnormality of conduction (that is, forms of endocardial cushion defects). Right bundle-branch block followed surgery in almost all patients with congenital heart disease with RBBB and LAD.

Surgical creation of right bundle-branch block has been reported with repair of ventricular septal defect,\(^26,31\) tetralogy of Fallot,\(^27\) valvular pulmonic stenosis,\(^28\) and isolated infundibular stenosis.\(^29\) Left axis deviation has been reported to occur after surgery for both discrete fibrous subaortic stenosis and idiopathic hypertrophic subaortic stenosis\(^9\) and also after transventricular aortic commissurotomy.\(^30\)

To our knowledge, the surgical creation of both RBBB and LAD, as seen in nine patients in this group, has been reported previously to occur after closure of a ventricular septal defect, but no other lesion.\(^31\) In their report, Kulbertus’ group\(^31\) stated that of the 39 patients with RBBB and LAD either before or after surgery, 11 (28%) developed CHB postoperatively. This again illustrates the vulnerability of patients with this form of BBBB to development of CHB. Long-term follow-up of a large group of patients with congenital heart disease and BBBB will be necessary to determine the ultimate incidence of CHB in this setting.

That conduction abnormalities can involve the inferior intraventricular radiation of the left bundle of His has only recently been recognized. The anatomy of the inferior radiation has been well described.\(^5\) Experimental laceration of these fibers in canines and baboons has resulted in an inferior shift of the frontal plane terminal forces.\(^9,10,32\) In man, involvement of this conduction pathway can result from fibrosis or infarction. Pryor and Blount\(^5\) reported two cases of an electrocardiographic pattern suggesting this lesion, in which postmortem examination revealed fibrosis involving the inferior radiation.

It has been pointed out that a diagnosis of
inferior radiation block should be made only in those patients who are at least 40 years of age, who have medium to heavy body build, and who have no evidence for, or a reason to have, right ventricular hypertrophy or enlargement. \(^5\) Certainly, normal young people frequently have an inferiorly directed frontal plane axis, as do patients with right ventricular hypertrophy.

Those patients previously reported as having this conduction abnormality have usually had ischemic heart disease, hypertension, or diabetes, and many have had evidence for left ventricular enlargement. \(^5\) Thus, they appear to be of the same group of patients as are those with LAD.

Inferior peri-infarction block has been long recognized. The conduction abnormality associated with this electrocardiographic pattern is now felt also to represent disease involving the inferior radiation.

The combination of RBBB and IIVB has been described in only a few patients previously and is considered to be a form of bilateral bundle-branch block, just as is RBBB and LAD. \(^4\) \(^-\) \(^8\) It is suggested that the combination of RBBB and IIVB carries a more serious prognosis than does RBBB and LAD, since the vascular supply of the various conduction pathways would require more widespread involvement to result in the former than in the latter type of BBBB.

Bilateral bundle-branch block is the most common forerunner of complete heart block, and RBBB with LAD is the most common type of BBBB. \(^1\) \(^,\) \(^24\) \(^,\) \(^33\) Lasser and associates\(^1\) reported a 10% incidence of complete heart block in patients with RBBB and LAD. Our incidences of 13.6% for patients with RBBB and SIVB and 15% for those with RBBB and superior peri-infarction block, are of the same magnitude. Presumably, with more complete and longer follow-up, this incidence of complete block would be even higher.

There have been no previous reports of the frequency of complete heart block in patients with RBBB and block of the inferior radiation of the left bundle. Our figures of 21% for those with IIVB and 12% for those with inferior peri-infarction block are in general agreement with those for RBBB and LAD. This seems to confirm that RBBB and block of the inferior radiation is a real form of BBBB and may lead to CHB, as described in a few cases by others.

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PARTIAL BILATERAL BUNDLE-BRANCH BLOCK

Minuscule Reviews

Of Two Papers in Advances in Internal Medicine—Vol. 16—1970


The authors are known to have had a long-time interest in the nature of intraventricular block and their clinical insights and experimental data have helped to elucidate the problems thereof. Their review of the problem is clear and concise and after an introductory paragraph, there are sections, subtitled, Species Differences, Sites and Extent of Intraventricular Block, Nomenclature and Criteria, Definition, Divisional (Ramus, Fascicular) Block, “Bilateral Bundle-Branch Block,” Incomplete Bundle-Branch Block, and Conclusions. The most detailed review is contained in the section on Incomplete Bundle-Branch Block. Evidence is marshalled to indicate that the peripheral ramifications of the conduction mechanism behave as a syncytium and that there is no longitudinal isolation of fibers in the main bundles in the dog’s heart. There are no illustrations and this might blunt the interest of some readers, but in the 78 references listed, there are made available sources of graphic data. Since a brief historic perspective is given, I would have liked to have seen Fahr’s work mentioned (Arch Intern Med 25: 146, 1920) which clearly analyzes sequential vectors and right and left bundle-branch block are correctly designated.

At this time when there is renewed interest in intraventricular conduction aberrations, spirited discussions regarding nomenclature and the continuing clarification of issues through the recording of His bundle potentials and myocardial potentials revealing the excitatory wave fronts in the ventricles. This short review of Wennemark and Kossmann should engage the attention of, and be profitable reading to, many cardiologists.

H.B.B.

Burchell HB: Surgical approach to the treatment of ventricular pre-excitation (p 43).

One of the most difficult things for a person who has pioneered a technic or a form of therapy is to evaluate with full objectivity the overall results at a later date.

Dr. Burchell has presented the various aspects relating to the surgical treatment of symptomatic Wolff-Parkinson-White arrhythmia. This is of great importance since several reports have testified to the fact that surgery can be performed successfully in selected patients with disabling, and otherwise uncontrolled, tachycardias. However, emphasis on the potential causes of failure was necessary to prevent indiscriminate use of a procedure with definite risks. Surgeons should be aware that a “Kent” bundle need not always be present. Its anatomical location is not the same in all patients. For this reason Dr. Burchell stressed that ground rules had to be established to justify future surgical ventures.

Since the field of conduction disturbances is moving at a jet-age speed, certain points would have required more emphasis if the subject had been written at the same time as this review. The effects of atrial pacing, the value of His bundle recordings, and certain additional refinements in technic would have been emphasized.

Agustin Castellanos

Circulation, Volume XLII, December 1970
Right Bundle-Branch Block Associated with Left Superior or Inferior Intraventricular Block: Clinical Setting, Prognosis, and Relation to Complete Heart Block

PATRICK J. SCANLON, RAY PRYOR and S. GILBERT BLOUNT, JR.

_Circulation_. 1970;42:1123-1134
doi: 10.1161/01.CIR.42.6.1123

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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/42/6/1123

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