Diagnosis of Isolated and Combined Block in the Bundle Branches and the Divisions of the Left Branch

By Agustin Castellanos, Jr., M.D., and Louis Lemberg, M.D.

The His bundle is a narrow, cylindrical structure which connects the atrioventricular (A-V) node with the bundle branches (fig. 1) and is composed of a penetrating and a branching portion. The former begins in the A-V node and ends where it emits the posteroinferior fibers of the left bundle. The branching portion extends from the latter site to the origin of the right branch (which is also close to where the anterosuperior fibers of the left bundle are emitted).

Conduction disturbances occurring in the specialized tissues located between His bundle and ventricles can produce A-V block as well as QRS abnormalities.1 Full understanding of the morphological changes produced by isolated and combined block in the bundle branches and their divisions is important in view of their prognostic and therapeutical implications.2

Complete Block in One Bundle Branch or in One Division of the Left Branch

The following conduction disturbances are included in this section: (a) complete right bundle-branch block (CRBBB), (b) complete block in the left bundle branch, and (c) left anterior hemiblock (Lah)1 and left posterior hemiblock (LPH).1 The terms left anterior and left posterior fascicular block have been applied to (b) and (c).

Table 1 shows the most important electrocardiographic findings in “pure” and “complicated” LAH. In LAH, the initial QRS vectors point inferiorly and to the right (due to propagation emerging from the unblocked posteroinferior division). The maximal vector is oriented superiorly and to the left because of the inferosuperior propagation through the electrically predominant left ventricle. This sequence of activation produces the: (a) abnormal left axis deviation (beyond −30°); 3 (b) qR pattern in lead I; (c) rS complexes in leads II, III, and aVF. QRS duration is at the upper limits of normal or slightly prolonged. A normal P-R interval and the normal-to-high voltage of the QRS complexes rules out other causes of abnormal left axis deviation such as pulmonary emphysema, Wolff-Parkinson-White syndrome, hyperkalemia and right ventricular apical pacing.3-5 In addition, certain processes which affect the initial vectors can modify the classical hemiblock pattern. For instance, myocardial infarction of any location produces changes in the initial vectors, with or without early and late slurrings due to “focal” conduction disturbances. The latter occur beyond the Purkinje system, inside, or around the infarcted area and have been loosely classified as post-infarction, peri-infarction, and intra-infarction block, etc.5 These conduction defects, which are electrophysiologically and morphologically different from LAH or LPH, can cause a considerable increase in the duration of the QRS complexes.

Specifically, when LAH is complicated by an anterosetal myocardial infarction (AMI), the q wave disappears in lead I and QS complexes are recorded in the right chest leads. An extensive anterior infarction with lateral involvement causes a QR or QS pattern.
in lead I, so that the electrical axis is deviated superiorly (due to the hemiblock) and to the right (because of the infarction). When an extensive inferior wall myocardial infarction (IWMI) produces abnormal left axis deviation, leads III and aVF show QS complexes. A terminal r wave (Qr pattern) appears in lead II. On the other hand, an IWMI occurring in a patient with preexisting LAH abolishes the initial r wave in II, III, and aVF. QS complexes are recorded in these leads.

By definition, the association of LAH with incomplete left bundle-branch block (ILBBB) is not an "isolated" lesion since two sites of the same (left bundle) system are affected. Yet, ILBBB is included in table 1 to show that it modifies the hemiblock pattern in a similar, but not identical way as ASMI. Although the q wave disappears in lead I, QS complexes are generally not recorded in lead V2. At times it is difficult to differentiate between ILBBB, septal fibrosis, localized septal infarctions, and coexisting incomplete LAH + LPH.

According to Pryor and Blount, the presence of abnormal left axis deviation in a patient with CLBBB suggests that there is a coexisting block at the level of the bundle branch and in the superior division. We believe that the diagnosis of this double conduction disturbance cannot be made without serial electrocardiograms.

In LPH the initial vectors point superiorly and to the left, and the maximal vector is oriented inferiorly and towards the right. In consequence, the electrical axis is deviated to the right, and lead I shows an rs complex. A

**Table 1**

| Criteria for the Diagnosis of Pure and Complicated Left Anterior Hemiblock (LAH) |
|-------------------|---|---|---|---|---|---|---|---|
| **Electrical axis** | **QRS duration** | **Lead I** | **Lead II** | **Lead III** | **Lead aVF** | **V1** | **V2** |
| LAH               | > - 30°  | N or SP | QR | rS | rS | rS | rS | rS |
| LAH + ILBBB       | > - 30°  | N or P  | R  | rS | rS | rS | rS | rS |
| LAH + ASMI        | > - 30°  | N or P  | R  | rS | rS | rS | QS | QS |
| LAH + ALMI        | > - 30°  | N or P  | QR | rS | rS | rS | QS | QS |
| LAH + IWMI        | > - 30°  | N or P  | R  | QS | QS | QS | rS | rS |
| Pure IWMI (with ALAD) | > - 30° | N or P  | R  | QS | QS | QS | rS | rS |

*See text for definition.

Abbreviations: ILBBB = incomplete left bundle-branch block; ASMI = anteroseptal myocardial infarction; ALMI = anterolateral myocardial infarction; IWMI = inferior wall myocardial infarction; ALAD = abnormal left axis deviation; N = normal; SP = slightly prolonged; P = prolonged.
Table 2

Criteria for the Diagnosis of Pure and Complicated Left Posterior Hemiblock (LPH)

<table>
<thead>
<tr>
<th>Electrical axis</th>
<th>QRS duration</th>
<th>Lead I</th>
<th>Lead II</th>
<th>Lead III</th>
<th>Lead aVF</th>
<th>V1</th>
<th>V2</th>
</tr>
</thead>
<tbody>
<tr>
<td>LPH</td>
<td>&gt; + 120°</td>
<td>N or SP</td>
<td>rS</td>
<td>qR</td>
<td>qR</td>
<td>qR</td>
<td>QS</td>
</tr>
<tr>
<td>LPH + ASMI</td>
<td>&gt; + 120°</td>
<td>N or P</td>
<td>rS</td>
<td>qrR</td>
<td>qrR</td>
<td>qrR</td>
<td>QS</td>
</tr>
<tr>
<td>LPH + IWMI*</td>
<td>&gt; + 120°</td>
<td>N or P</td>
<td>rS</td>
<td>qR</td>
<td>qR</td>
<td>qR</td>
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</tbody>
</table>

*Clinical, enzymatic, and evolutionary St-T changes required for this diagnosis.
Abbreviations as in table 1.

Figure 2

Vectorcardiogram (Frank system) and electrocardiogram in a case with left anterior hemiblock and complete right bundle-branch block. H = horizontal plane; F = frontal plane; S = sagittal plane.
Figure 3
Vectorcardiogram (Frank system) and electrocardiogram in a case with left posterior hemiblock and complete right bundle-branch block. Initial slurring in V2 is an artifact. H = horizontal plane; F = frontal plane; S = sagittal plane.

qR pattern is recorded in II, III and aVF (table 2). Since the initial vectors have an anterior and leftwards orientation, a negative deflection might be observed in V1 (but not in V2) in the absence of ASMI. The pattern of LPH can be produced by other processes (right ventricular hypertrophy, pulmonary disease, extremely vertical hearts, extensive lateral infarction). Therefore, the diagnosis of LPH cannot be made from the electrocardiogram or vectorcardiogram alone. Clinical, X-ray, angiographic, or postmortem studies are required for this purpose.

When LPH is associated with ASMI, the initial vectors point to the left and posteriorly and predominant negative deflections are recorded in the right chest leads. The presence of IWMI can be recognized by a large and wide Q wave in aVF. However, wide Q waves can be observed in pure LAH in the absence
of IIMI. In these cases, the diagnosis of IIMI can only be made by the evolutionary ST-T wave changes.

**Complete Block in the Right Branch and in One Part of the Left Bundle System**

This heading was used to avoid possible misunderstandings which could arise from the interpretation given to the terms "bilateral" or "bifascicular" blocks. Rosenbaum and co-workers described 30 different combinations which could be included in this section. Therefore, for the sake of simplicity, only those instances of **permanent** block in the sites mentioned before will be described:

1. **Complete block in the right and left bundle branches (complete bilateral bundle-branch block).** In these cases the blocked P waves reach the His bundle but fail to activate the right branch (RB) or the left branch (LB).  
2. **CRBBB + ILBBB (incomplete bilateral bundle branch block).** This diagnosis can be made if a CRBBB pattern appears in the surface electrocardiogram and the His bundle recordings show a prolonged H-V interval (due to a delay in the LB potential). In the presence of CRBBB, an ILBBB should produce an A-V conduction disturbance rather than morphological changes in the initial portions of the ventricular complexes. Prolonged P-R intervals in patients with CRBBB are not necessarily indicative of a conduction disturbance in the left bundle system since the block can occur at the A-V nodal level. His bundle recordings are **essential** to localize the site(s) of conduction delay.  
3. **CRBBB + LAH, with or without myocardial infarction, and normal H-V intervals.** See figure 2.  
4. **CRBBB + LPH, with or without myocardial infarction, and normal H-V intervals.** See figure 3.  

In the latter two instances, LAH, LPH, and myocardial infarction can be diagnosed as in tables 1 and 2, and CRBBB by the classical criteria. The presence of the latter does not interfere with the recognition of LAH or LPH since, in surface recordings, CRBBB affects only the terminal vectors. In consequence, the fundamental features of hemiblocks are maintained when they are associated with CRBBB.

**Complete Block in the Right Branch and in Two Sites of the Left Bundle System**

The following combinations can occur if the block is permanent in the three sites:

1. **CRBBB + LAH + LPH** (complete trifascicular block) produces complete A-V block. His bundle recordings show that the blocked P waves are followed by His bundle deflections and LB potentials.  
2. **CRBBB + LAH + a conduction delay in the posterior division (incomplete trifascicular block) produces a pattern of CRBBB + LAH with a prolongation of the H-V interval (at the expense of the LB-V subinterval).**  
3. **CRBBB + LPH + a conduction delay in the anterior division (incomplete trifascicular block) produces a pattern of CRBBB + LPH with a prolongation of the H-V interval (at the expense of the LB-V subinterval).**  
4. **CRBBB + a conduction delay (of more or less the same degree) in both divisions of the left branch (incomplete trifascicular block) produces a CRBBB pattern and a prolonged H-V interval (at the expense of the LB-V subinterval).**  
5. **CRBBB + LAH + ILBBB** produces a pattern of CRBBB + LAH and a prolonged H-V interval (due to a delay in the LB potential).  
6. **CRBBB + LPH + ILBBB** produces a pattern of CRBBB + LPH and a prolonged H-V interval (due to a delay in the LB potential).

**The Concept of “Electrical Axis”**

Proper interpretation of the criteria for the diagnosis of LAH and LPH requires an understanding of the fact that the term “electrical axis” has been used loosely and interchangeably with the QRS, the “mean” axis, and the “major” axis. While the QRS is determined by calculating areas in the proper
leads, the “mean” axis can be estimated roughly by using the amplitude of the positive and negative deflections. The “major” axis is obtained by using the magnitude of the largest deflections in the corresponding leads. We believe that the “major axis” of QRS is more useful than the $A_{QRS}$ for the diagnosis of complicated LAH and LPH. For instance, in a patient with CRBBB and an RS complex in lead I (in which the amplitude of these waves is the same) the $A_{QRS}$ can be to the right of +120°, while the maximal deflection axis is only around +90°. The use of the $A_{QRS}$ could lead to the associated diagnosis of LPH, which would not be considered probable from the “major axis.”

References

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Circulation. 1971;43:971-976
doi: 10.1161/01.CIR.43.6.971

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/43/6/971.citation

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