Gross Anatomy Associated with Patterns Called Left Posterior Hemiblock

By A. Wade Strickland, M.D., Leo G. Horan, M.D., and Nancy C. Flowers, M.D.

SUMMARY

From a compilation of 1411 gross dissections of the hearts from patients who had had recent electrocardiograms prior to death, 62 were found to have frontal QRS axes between +90 and +180°.

Thirty-eight exhibited an S\textsubscript{1}Q\textsubscript{3}R\textsubscript{3} pattern—the second screening criterion basic to consideration for the label of left posterior hemiblock (LPH). Twenty-two of these also had right ventricular free wall weights in excess of 70 g. Two others exhibited inferior myocardial infarction only. Of the remaining 14, six afforded mild clinical suspicion of increased hemodynamic loading of the right heart but did not have increased right ventricular weights. Four had right bundle-branch block (RBBB), and only one had a prolonged P-R interval.

The S\textsubscript{1}Q\textsubscript{3}R\textsubscript{3} pattern with right-axis deviation thus occurred in patients with or without right ventricular hypertrophy and with or without inferior wall myocardial infarction. Right bundle-branch block was a frequent occurrence in the spectrum of right-axis deviation (RAD) whether S\textsubscript{1}Q\textsubscript{3}R\textsubscript{3} was present or not.

The scatter of the frequent associates of RAD—inferior myocardial lesions, right ventricular hypertrophy, a clinical history of right ventricular loading diseases, and RBBB—suggests three alternative ways of viewing the S\textsubscript{1}Q\textsubscript{3}R\textsubscript{3} pattern with RAD: (1) LPH is a cause of S\textsubscript{1}Q\textsubscript{3}R\textsubscript{3} with RAD. It is a manifestation of left ventricular myocardial disease, but it may be a result of overt infarction, or may be mimicked by right ventricular disease. (2) LPH is the cause of S\textsubscript{1}Q\textsubscript{3}R\textsubscript{3} with RAD. It is the means by which diverse etiologies produce a distinctive electrocardiographic pattern (including left ventricular myocardial deficits, right ventricular enlargement, or a small group of unknown causes). (3) LPH is an artifact of convenience. Patients with RAD may or may not have S\textsubscript{1}Q\textsubscript{3}R\textsubscript{3}; they frequently have inferior wall myocardial infarction, right ventricular overload or enlargement, and RBBB.

Additional Indexing Words:
Left fascicular blocks Right-axis deviation

INTEREST IN the electrocardiographic concept of block in only one division of the left bundle branch has become abundant in the past few years.\textsuperscript{1-3} Less abundant has been an appreciation for where the hemiblock pattern fits in the surrounding spectrum of axis deviation and what its relationship is to the other causes of such axis deviation.\textsuperscript{4} Compounding the problem of understanding the functional significance of the hemiblock pattern in electrocardiographic interpretation is the growing divergence of opinion as to what is the usual distribution of the left bundle
branch and its ramifications in the human heart.5-8

We recently completed a prospectively planned inquiry into the relationship between gross myocardial lesions, myocardial hypertrophy, and electrocardiographic patterns of QRS morphology.9-11 Because of the pertinent opportunity, we have reexamined our data with respect to the pattern called left posterior hemiblock. We have sought in this report: (1) to view the data appearing in the spectrum of axis deviation between +90 and +180°; (2) to consider where the pattern labeled left posterior hemiblock fits into that spectrum; and (3) to consider the effect of other abnormalities (such as gross regional myocardial deficit, right ventricular hypertrophy, or the presence of right ventricular loading disease) upon relating the pattern of LPH to its neighbors in the band of RAD. This study which was begun 10 years ago, does not include a report of the histologic examination of the conduction system. It does present the occurrence of the LPH pattern against a background of RAD and gross morphology in a single, large series of patients in which there was a uniform prospective approach to anatomic categorization.

Methods

Between the years 1961 and 1967, over 1600 hearts were examined by one or more of us: 1411 were from patients in whom standard electrocardiograms with normal sinus rhythm and QRS antegrade conduction had been obtained within the preceding 90 days at the Kennedy Veterans Administration Hospital, Memphis, Tennessee. The details of the method of dissection and its use in relating anatomic localization of myocardial lesions to electrocardiographic findings have been previously described.8-11 A brief recapitulation follows: The coronary arteries were serially sectioned transversely, then opened longitudinally. The septal and left ventricular free walls were laminated parallel to endocardial and epicardial surfaces at 5-cm intervals and, after fat removal, the right and left ventricles were weighed. The locations of myocardial infarctions and scars were recorded on large standard diagrams of the left ventricle. Coronary artery luminal encroachment was measured and site indicated. The degree of narrowing was rated on the basis of 0 to 4. The left ventricular myocardium was divided into septal, anterior, and posteroinferior thirds, and each third was further divided into five sectors: two basal, two central, and one apical. The occurrence of scar or infarction* in each sector was tabulated for each patient and filed with other morphologic data, including ventricular weights (fig. 1, table 1). Right ventricular enlargement was specified when the right ventricular free wall weight exceeded 70 g.11,12 Electrocardiographic voltages and time measurements were also tabulated for each lead of the most recent 12-lead electrocardiogram of each subject.

From these data, the mean frontal-plane QRS axis† was computed for each subject from the ten deflection (algebraic sum of R and S) in standard leads I and III. Computer sorting then selected for further study those electrocardiograms with a QRS axis between +90 and +180°; the presence or absence of S1Q3 pattern and whether R in lead III exceeded 5 mm in height was noted in each instance. Finally the presence or absence of right bundle-branch block (RBBB) and any degree of atrioventricular (A-V) block was also noted. The pertinent anatomic data were reviewed with particular attention as to whether gross evidence of myocardial deficit appeared in the inferoseptal zone (sectors 1, 2, 13, and 14 in fig. 2) or the lateral zone (sectors 8, 9, 11, and 12). This was done because: (1) the immediate region that the posterior portion of the left bundle may be expected to supply was the inferoseptal region; and (2) axis shifts, either right or left, may be expected from lateral infarction.

Results

Frequency Distribution of S1Q3R, Pattern between +90 and +180°

The QRS axis of a recent electrocardiogram in 62 subjects lay between +90 and +180°. The distribution peaked between +96 and +100°, rapidly trailing off toward 180° as seen in figure 1. The portion of the histogram above the zero line indicates the 38 patients with electrocardiograms of the S1Q3 pattern with an R3 greater than 5 mm. The remaining 24 (shown below the bold line) failed to have

*We will use the term myocardial infarction to refer to grossly visible fresh or subacute necrosis, the term myocardial scar to refer to grossly visible fibrosis, and myocardial deficit to include both infarction and scar.13

†Subsequent mention of QRS axis and axis deviation to the left refers to this conventional clinical estimate of mean axis as projected onto the frontal plane.
Histogram demonstrating frequency distribution of patients with electrocardiograms with right-axis deviation (between +90° and +180°). The ordinate indicates the number of patients and the abscissa, the degrees of frontal-plane axis. Patients with S\(_1\)Q\(_3\)R\(_3\) pattern are shown above the zero line; those without, below. The instances of right ventricular hypertrophy (with weight of free wall over 70 g) are shown by open blocks; two additional cases with inferior myocardial infarction only are indicated by the slanting lines (from southwest to northeast). Instances of clinically suspected right ventricular load without proven right ventricular hypertrophy are indicated by the bolder slanting lines (from northwest to southeast) and the remaining instances of probable LPH pattern are shown by the thin vertical lines. A star labels each instance of RBBB.

Table 1

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (yr)</th>
<th>QRS axis(°)</th>
<th>RV weight (g)</th>
<th>RBBB</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. &quot;Probable&quot; LPH</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.J.</td>
<td>68</td>
<td>95</td>
<td>70</td>
<td>No</td>
<td>Coronary artery disease; prolonged P-R</td>
</tr>
<tr>
<td>W.F.H.</td>
<td>55</td>
<td>96</td>
<td>53</td>
<td>No</td>
<td>Esophageal carcinoma</td>
</tr>
<tr>
<td>O.L.J.</td>
<td>52</td>
<td>96</td>
<td>65</td>
<td>No</td>
<td>Intracranial neoplasm</td>
</tr>
<tr>
<td>W.E.T.</td>
<td>65</td>
<td>131</td>
<td>52</td>
<td>Yes</td>
<td>Coronary artery disease</td>
</tr>
<tr>
<td>C.N.</td>
<td>34</td>
<td>100</td>
<td>49</td>
<td>Yes</td>
<td>Lymphoblastoma</td>
</tr>
<tr>
<td>L.J.</td>
<td>45</td>
<td>94</td>
<td>34</td>
<td>No</td>
<td>Cardiac contusion</td>
</tr>
<tr>
<td>B.M.S.</td>
<td>47</td>
<td>96</td>
<td>53</td>
<td>No</td>
<td>Ulcerative colitis</td>
</tr>
<tr>
<td>S.B.</td>
<td>76</td>
<td>106</td>
<td>60</td>
<td>No</td>
<td>Coronary artery disease</td>
</tr>
<tr>
<td>II. &quot;Possible&quot; LPH</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.S.</td>
<td>42</td>
<td>98</td>
<td>52</td>
<td>Yes</td>
<td>Bronchogenic carcinoma</td>
</tr>
<tr>
<td>J.M.K.</td>
<td>65</td>
<td>93</td>
<td>50</td>
<td>No</td>
<td>Pulmonary tuberculosis</td>
</tr>
<tr>
<td>L.T.B.</td>
<td>47</td>
<td>97</td>
<td>53</td>
<td>No</td>
<td>Bronchogenic carcinoma</td>
</tr>
<tr>
<td>W.L.M.</td>
<td>54</td>
<td>103</td>
<td>62</td>
<td>No</td>
<td>Bronchogenic carcinoma</td>
</tr>
<tr>
<td>H.S.</td>
<td>67</td>
<td>98</td>
<td>65</td>
<td>No</td>
<td>Chronic obstructive lung disease</td>
</tr>
<tr>
<td>B.F.J.</td>
<td>74</td>
<td>94</td>
<td>56</td>
<td>Yes</td>
<td>Bronchogenic carcinoma</td>
</tr>
</tbody>
</table>

either an S\(_1\)Q\(_3\) configuration or a prominent R\(_3\). In 22 of the patients with the basic S\(_1\)Q\(_3\)R\(_3\) pattern, the right ventricular weight exceeded 70 g. Two further exclusions from the S\(_1\)Q\(_3\)R\(_3\) group were made because of inferior myocardial infarction or scar; 10
others with myocardial deficit previously had been excluded because of right ventricular hypertrophy.

This left 14 electrocardiograms to be considered eligible for the tentative label of LPH. Six of these (shaded with bold slanting lines and called "possible" LPH in table 1) would have ordinarily escaped such a label because of some degree of right ventricular loading suspected clinically. The actual right ventricular weight, however, did not exceed 70 g. Clinical diagnoses were bronchogenic carcinoma in four, pulmonary tuberculosis in one, and chronic obstructive lung disease in one. This contrasted with a diagnosis of severe obstructive lung disease in five of the patients in the corresponding group without the S_1Q_3R_3 pattern and without right ventricular hypertrophy. The sixth had rheumatic mitral stenosis. The stars in figure 1 indicate the accompaniment of RBBB: the frequency of occurrence of RBBB was approximately one of four whether the group considered was the possible LPH group, the probable total LPH group, the patients above the horizontal with S_1Q_3R_3, or all subjects with RAD.

**Figure 2**

Diagrammatic representation of the left ventricular myocardium divided into 15 sectors for the purpose of localizing lesions. A diagram of the beginning of the anterior and posterior divisions of the left bundle is superimposed upon the diagram of the interventricular septum. The diagram is intended to indicate the limits of the divisional separation and does not show the many interconnections present in the human heart. Attention was focused upon lesions in the inferoseptal region (dotted) supplied by the posterior division and lesions in the lateral free wall (slanted lines). Sector 9 of the lateral free wall is between sectors 8 and 10 and hidden by the curve of the ventricular wall. See text for details.

**Figure 3**

Histogram showing the frequency distribution of patients with myocardial deficit (scar or infarction) involving at least the inferior wall of the left ventricle (open blocks) or outside the inferior wall (slanted lines). The ordinate and abscissa are as in figure 1. Patients with S_1Q_3R_3 pattern are indicated above the zero line; those without, below.
were extensive and ran into adjacent regions of the heart also. Only two formed solid transmural lesions in the inferior wall.

**Influence of Site of Deficit upon Axis Deviation**

In these 21 patients, the gross myocardial deficits of nine extended throughout zones 1, 2, 13, and 14 (the "inferoseptal" region) and nine more entered this region at least in part. By contrast, only four examples extended throughout the "lateral" region (zones 8, 9, 11, and 12, but 11 entered it partly). Table 2 compares the axis deviation for portions of the whole original group of 1411 dissections with and without such myocardial deficits and with and without ventricular hypertrophy. The means appeared not to shift remarkably with either major "inferoseptal" or "lateral" deficit.

**Discussion**

In figure 1 are plotted all cases of +90 through +180°; the 38 with S

1Q

3R

3 pattern (above the zero line) may be representative of the initial population in which to consider LPH. Note that the cases with posterior hemiblock patterns without right ventricular loading diseases are found in the company of cases that had right ventricles weighing greater than 70 g. These two groups can usually be easily separated since the ones with markedly enlarged right ventricles and atria had severe chronic lung disease and could be recognized electrocardiographically as well as clinically as having cor pulmonale. The difficulty with pure electrocardiographic diagnosis of posterior hemiblock arises in the range of between +90 and +110°. Here, we were unable to distinguish electrocardiographically the cases with suspected posterior hemiblock without right ventricular loading diseases from the cases with disease predisposing to such overload but without right ventricular hypertrophy.

According to Simonson,14 the upper limit for rightward deviation of the mean QRS axis in the age group of 40–59 years is +87.9° with a standard error of 2.91. It could be postulated that all cases with a mean QRS axis of +90° or greater associated with S

1Q

3R

3 pattern and relatively tall voltage in leads III and aVF represent impaired conduction in the region of the posterior ramifications of the left bundle. Indeed the distribution of the S

1Q

3R

3 pattern in the range of QRS axis between +90 and +180° was paralleled by the distribution of proven right ventricular hypertrophy, clinical right loading without hypertrophy, inferoseptal myocardial scar or infarction, and even RBBB. It may be further argued that the effect of conduction defect could be obtained by interfering with the delivery of conducted impulses from the Purkinje system by scar or infarction in the region supplied by the left posterior fascicle. Of incidental interest is the fact that one of the patients with S

1Q

3R

3 excluded from the final or possible LPH grouping because of right ventricular hypertrophy had calcific aortic stenosis and left ventricular hypertrophy. One patient included in the probable LPH group also had a history of cardiac contusion which could have been responsible for impaired conduction, and one

| Table 2 |

**Comparisons of Axis Deviation**

<table>
<thead>
<tr>
<th>Group</th>
<th>RV ≤ 70 g</th>
<th>RV &gt; 70 g</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Mean axis (°)</td>
</tr>
<tr>
<td>Myocardial scar of infarction:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Including inferoseptal region</td>
<td>46</td>
<td>26.7</td>
</tr>
<tr>
<td>Including lateral region</td>
<td>36</td>
<td>22.0</td>
</tr>
<tr>
<td>Excluding inferoseptal region</td>
<td>65</td>
<td>30.7</td>
</tr>
<tr>
<td>Excluding lateral region</td>
<td>85</td>
<td>24.7</td>
</tr>
<tr>
<td>Without myocardial scar or infarction:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>With LV ≤ 180 g</td>
<td>382</td>
<td>36.6</td>
</tr>
<tr>
<td>With LV &gt; 180 g</td>
<td>239</td>
<td>31.8</td>
</tr>
</tbody>
</table>

*Circulation, Volume XLVI, August 1972*
patient died with lymphoblastoma. Lymphomas have also been reported to cause impaired conduction in the posterior fascicle.6 Watt and Pruitt confirmed the development of a Q wave, increased ratio of R to S, delayed intrinsic deflection time, and disappearance of the S wave in leads III and aV\textsubscript{F} following the production of posterior hemiblock in primates.2 The S wave reappeared with the addition of RBBB. Examples of posterior hemiblock with RBBB and without RBBB have been shown. All of our four cases with the posterior hemiblock pattern plus RBBB did not have an S wave in lead III; two of the remainder with posterior hemiblock and without RBBB had a small S wave in lead III. Watt and Pruitt also found that the QRS-axis shift was greater for left anterior hemiblock than for left posterior hemiblock. From their experimental work, it would appear that it is not necessary to acquire an extreme QRS-axis shift (+120°) before suspecting posterior hemiblock. However, they suspected that the QRS change may be so slight that a previous baseline tracing may be needed to make the diagnosis of disturbed conduction in the left posterior fascicle. On the other hand, Medrano et al. concluded from their study in dogs that posterior hemiblock could be recognized clinically from its characteristic electrocardiographic finding.

Rosenbaum et al.1 noted disturbed A-V conduction in 83% of his cases with RBBB and posterior hemiblock. In the nine patients with RBBB and RAD observed by Castellanos et al.7 seven had long P-R intervals, and eight exhibited periods of type II Mobitz block. If all 14 of our cases are true posterior hemiblock, then we did not find a strong association between disturbed A-V conduction and the combination of RBBB and posterior hemiblock, since in our four cases with RBBB and posterior hemiblock none had 1° A-V block. One patient without RBBB did have 1° A-V block.

Thus, the cases listed in table 2 are shown in figures 1 and 3 from a group quite different from that encountered by Rosenbaum et al. and by Castellanos et al.1,4 We believe the difference arises from the means of selection: the previous reports concerned patients seen in the context of life and the emergency situation, whereas our report reviews patients who have come to autopsy with subsequent reexamination of the electrocardiogram. Our infrequent finding of A-V block even in the patients with S\textsubscript{1}Q\textsubscript{3}R\textsubscript{3} pattern and myocardial infarction probably relates to the fact that many of the infarctions in this group were not acute, and that any degree of block associated with the original episode had already been survived. In addition, the electrocardiogram chosen for tabulation and sorting was the one taken nearest the time of death, and, although others retained in the patient’s folder had been selected as representative from the total file, previous episodes of A-V block may have been overlooked.

This study confirms the rarity of the electrocardiographic pattern called left posterior hemiblock after the exclusion of those with anatomically documented right ventricular enlargement has been applied to the S\textsubscript{1}Q\textsubscript{3}R\textsubscript{3} pattern with RAD. Sufficient empirical evidence exists to suggest that, in adults, axis deviation beyond +90° without other evidence of right ventricular enlargement should be viewed with suspicion as possibly representing delay in the region of the left posterior fascicle. It probably does not matter whether the posterior portion of the left bundle is a discrete anatomic pathway1,3,6 or is the boundary of a diverging but interconnected network7,8 as long as it functions as an approximate “division.” It is important prospectively and therapeutically to recognize possible additional involvement of the left bundle when the S\textsubscript{1}Q\textsubscript{3}R\textsubscript{3} pattern with RAD occurs in the presence of RBBB, since a significant number of these patients have been shown to develop complete A-V block.1,3,4,16

References


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