Electrocardiographic Criteria for the Diagnosis of Left Anterior Fascicular Block*

Left Axis Deviation and Delayed Intraventricular Conduction

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The two current criteria for diagnosis of left anterior fascicular block (LAFB) were evaluated; they are marked left axis deviation (LAD) and a delay in the time of inscription of the intrinsicoid deflection (ID) in lead aVL asynchronous to V6. From 400 electrocardiograms with a LAD of −30° or greater, 62 percent showed asynchronous activation of the left ventricle. There was only a general relationship between the degree of LAD and delayed ID in aVL. The incidence of delayed ID in aVL was as follows: 2 percent with mean frontal QRS axis at 0°; 9 percent at −15°; 41 percent at −30°; 69 percent at −45°; 82 percent at −60°; and 100 percent at −75° or greater. The lack of correlation between both criteria in many instances questions their validity. The LAD alone should not be considered synonymous with LAFB. Recognition of delayed inscription of the ID in aVL is a useful supplemental criterion for diagnosis.

In recent years the left bundle branch has been viewed as having two major divisions, anterior and posterior. The concept of selective block of the subdivisions of the left bundle, the so-called fascicular blocks or hemiblocks, has been advanced to explain deviations in the frontal plane QRS axis. Rosenbaum and co-workers1,2 have considered left anterior hemiblock or left anterior fascicular block (LAFB) to be present in those instances when left axis deviation (LAD) occurs on a standard electrocardiogram. However, there has not been agreement on the degree of axis deviation necessary to make the diagnosis. Although LAD is generally considered to be due to block in the left anterior fascicle, other ventricular alterations can produce this shift of QRS axis.2-10

Medrano and co-workers4-11,12 have described both clinically and experimentally a delay in the time of inscription of the intrinsicoid deflection (ID) in lead aVL due to a regional delay in the high lateral left ventricular activation as a consistent finding in block of the anterior superior division. More recently, Gallagher et al13 have reported a regional delay in high lateral left ventricular activation due to experimental interruption of the left anterior fascicle.

This study was undertaken to determine whether there is a correlation between LAD and possible regional delay in intraventricular conduction, represented by a delay of the ID in aVL as compared to V6 determined from the standard clinical ECG. We examined specifically the time of inscription of the ID in lead aVL and its relationship to the mean frontal QRS axis (AQRS) in patients with and without LAD.

METHODS

The AQRS was calculated to the nearest 15° in the 12-lead standard ECG of 6,303 consecutive patients in a general hospital adult population. In 500 tracings, an axis deviation of −30° or greater was observed. Those having other known electrocardiographic causes for LAD, such as diaphragmatic myocardial infarction (DMI) or left bundle-branch block (LBBB), were eliminated. Tracings with criteria for left ventricular hypertrophy (LVH)14 were eliminated, unless the onset of the ID in aVL was delayed at least 10 msec or more than in V6. The remaining ECGs were separated into the following groups according to the AQRS: −30°; −45°; −60°; and −75°. Two separate groups of 100 consecutive
Table 1—Left Axis Deviation and Delayed Intraventricular Conduction

<table>
<thead>
<tr>
<th>AQRS</th>
<th>No. of Cases</th>
<th>Delayed ID in aVL</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>0°</td>
<td>100</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>-15°</td>
<td>100</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>-30°</td>
<td>160</td>
<td>66</td>
<td>41</td>
</tr>
<tr>
<td>-45°</td>
<td>158</td>
<td>109</td>
<td>69</td>
</tr>
<tr>
<td>-60°</td>
<td>44</td>
<td>36</td>
<td>82</td>
</tr>
<tr>
<td>-75°</td>
<td>38</td>
<td>38</td>
<td>100</td>
</tr>
<tr>
<td>Total</td>
<td>600</td>
<td>260</td>
<td>43</td>
</tr>
</tbody>
</table>

ECGs chosen at random were reviewed with AQRS between 0° and -15°.

In all tracings the time of inscription of the ID was measured in lead aVL and in V6; measurements were made from the onset of the QRS to the beginning of the downstroke of the R wave. A regional delay in the inscription of the ID was considered present when it measured 50 msec or more in aVL, and was greater than 10 msec in aVL, when compared to lead V6, thus suggesting a possible asynchronous activation of the left ventricle. The reliability and reproductivity of measurements were ascertained by the analysis of 100 randomized ECGs in "blind" fashion on three different occasions; 93 percent of measurements were accurate within 10 msec. The AQRS and synchronous intraventricular time of conduction were then compared. Those ECGs with right bundle-branch block (RBBB) and LAD were analyzed as a separate subgroup. All ECGs were recorded with a three-channel automatic electrocardiograph (Marquette 3000) at 25 mm/sec and a frequency response from flat at direct current to 100 hertz down -3 dB at 120 Hz.

RESULTS

Left axis deviation of -30° or greater was observed in 500 of 6,303 ECGs (7.9 percent). One hundred cases were eliminated, because they had other abnormalities that might result in LAD. This group included 20 patients with LBBB, 66 with DMI, and 14 with LVH. There were five other instances in which LVH was suspected by voltage criteria; but, because of an asynchronous delay of the ID between aVL and V6, they were included in the study. Of the remaining 400 ECGs, 249 (62 percent) showed a delay in the ID in aVL that was not detected in V6 ("regional delay").

The separation of groups according to the degree of LAD is shown in Table 1. The relationship between AQRS and the onset of the ID in aVL is shown in Figure 1. Examples of the different forms of the ECG are shown in Figures 2 to 5.

Right bundle-branch block was associated with LAD in 35 cases. In every instance there was delayed onset of the ID in aVL. In contrast, not one of 20 ECGs with RBBB but without LAD had a delayed onset of ID in aVL.

DISCUSSION

There are two suggested criteria for the diagnosis of LAFB. Rosenbaum and associates proposed that a LAD of -45° or more may represent a hemiblock. This degree of LAD is usually accompanied by a Q3,S3 pattern. However, Rosenbaum et al. have also accepted a diagnosis of LAFB with a progressive leftward shift of the AQRS from a previously normal axis to minor degrees (less than -45°) of LAD. They suggest that these changes represent an incomplete form of hemiblock and that differentiation from normal variants is possible because of the availability of serial ECGs.
Figure 3. Electrocardiogram similar to that in Figure 2 but with shorter intervals. Time of inscription of ID in aVL is prolonged to 50 msec, a value greater than measurement in V6 of 35 msec.

Medrano and co-workers, after experimental studies in dogs, suggested that more specific criteria should be used. They believe that a delay in the inscription of the ID in lead aVL not present in lead V6 is a consistent finding in LAFB. These authors observed that, after the interruption of the anterior superior division of the LBBB, the activation process was delayed 20 to 30 msec in the upper third of the left septal mass and in high anterolateral regions of the left free ventricular wall. The delay was recognized by a prolongation of the intrinsic deflection time in direct epicardial leads, reaching 50 to 55 msec in high lateral recordings. These delayed forces are represented in the dog by a vector pointing upward and leftward in the vertical heart and upward and to the right in the horizontal heart. This explains the deep S waves in leads 2, 3, and aVF with slight LAD in vertical hearts. In the experimental dog with a horizontal heart, the R wave increases in voltage with a delay of the ID in leads 1 and aVL.

Activation studies following experimental posterior fascicular block in the dog revealed a consistent area of delay of 5 to 25 msec in epicardial surface maps that is confined to the lateral basal surface of the left ventricle. Transmural activation the same area revealed a 10 to 20-msec delay in the Purkinje and endocardial activation.

Most investigators agree that a LAFB will result in a 5- to 20-msec increase in the QRS duration in some leads of a standard ECG. Because the delay is regional, an asynchronous activation of the left ventricle occurs that may be recognized in the standard ECG. The inscription of the ID in lead aVL, the “exploring lead” over the affected high lateral ventricular wall, is delayed 10 msec or more when compared to the ID in lead V6, which explores the low free ventricular wall.

The present study was designed to evaluate the two suggested criteria, degree of LAD and regional prolongation of the QRS complex, for the diagnosis of LAFB. We found the two criteria satisfied in only 62 percent of cases with LAD of -30° or more. Thus, 38 percent considered LAFB by an axis criteria did not have a delayed ID. In contrast, 2 percent of cases with 0 axis and 9 percent with -15° axis had a significantly delayed ID in aVL compared to V6 but would not be recognized as possible instances of LAFB if the axis criterion alone was utilized.

The association between a regional delay in intraventricular conduction and extent of LAD suggests that there is an incomplete but direct relationship between both. The greater the leftward shift, the greater the possibility of a delayed ID in aVL. With an axis of -75° or greater, a regional QRS-ID delay was invariable.

A significant number of tracings with marked LAD did not have a “regional delay” in conduction. If both regional delay and LAD are necessary to establish LAFB, some other explanation for the LAD must be sought in these cases. There is no clear evidence to determine whether LAFB is or is not present in these tracings. Some could be explained by an electrical horizontal heart, obesity, or masked inferior myocardial infarction with an rS pattern in leads 2, 3, and aVF making diagnosis difficult by standard ECG. It is possible also that regional delay might not be manifest in aVL. Thus, in extreme horizontal hearts with LAFB, the delayed forces may be displaced rightward, so that the delay in the
ID may not be recognized in aVL but is present in aVR.\(^5\)

The value of the criteria suggested by Medrano et al\(^5\) is revealed by analysis of the data in patients with LAD and RBBB. All 35 cases with RBBB and LAD had delayed regional left ventricular activation. Regional delay was not seen in RBBB without LAD.

If, in some cases an LAD less than $-45^\circ$ may represent LAFB, whereas in other instances an LAD of more than $-45^\circ$ may not, we are faced with the problem of applying simple and conventional recognition criteria (left axis) that are neither sensitive nor specific. If the criteria suggested by Medrano et al are accepted as valid, it must be shown that they are specific. LAFB can be diagnosed utilizing these criteria in the presence of LVH, a condition that can produce LAD and a delay of the ID by itself but not in an asynchronous manner.\(^14\) Certainly LAD cannot be considered alone as an absolute criterion for the diagnosis, since many causes are known. It is not appropriate at the present time to consider LAD and LAFB synonymous. In our present state of knowledge, it seems reasonable to suggest a diagnosis of LAFB only when both an abnormal degree of left axis and a delay of conduction in the region of the affected fascicle can be detected. It should be recognized that the validity of the proposed criteria has not yet been demonstrated in the human heart.

REFERENCES


