Left Axis Deviation

A Spectrum of Intraventricular Conduction Block

Gopal Das, M.D.

SUMMARY The effects of left axis shift (> -30°) on intraventricular conduction time (IVCT) were studied in 63 subjects in whom electrocardiograms before and after appearance of the left axis shift were available. Subjects with electrocardiographic evidence of left or right ventricular hypertrophy, left or right bundle branch block, myocardial infarction, pre-excitation and those receiving antiarrhythmic drugs were excluded.

The IVCT increased by an average of 25 msec when the QRS axis shifted to -30° or beyond. When the individual AQRS was related to the IVCT a linear correlation (r = -0.8) was observed. It appears that a continuum relation between the AQRS and the IVCT exists throughout the counterclockwise range of +90° to -90°. In all leftward shifts in AQRS from +90° there is a prolongation in IVCT.

Our observations indicate that all leftward shifts in AQRS, including the extreme left axis shift (left anterior hemiblock), represent a spectrum of intraventricular conduction block.

MARKED LEFT AXIS DEVIATION, mean frontal plane QRS angle (AQRS) more negative than -30° in the absence of inferior wall myocardial infarction is commonly believed to result from an intraventricular conduction block in the anterior fascicle of the left bundle, and has been referred to in the past as parietal block of the superior division of the left bundle, left superior intraventricular block and more recently as left anterior hemiblock.1-4 Since the myocardium supplied by the blocked anterior fascicle depolarizes later in the sequence,5 it would expectedly result in lengthening in the total ventricular depolarization time. The results of studies analyzing the effects of acute and chronic appearance of the severe leftward axis shift on the intraventricular conduction time are conflicting and inconclusive.6-11

The present studies were specifically designed to evaluate critically the effects of naturally occurring severe left axis deviation on the intraventricular conduction time and the interrelation between the axis shift and the conduction time through controlled observations on the same patients before and after the spontaneous appearance of left axis shift.

Material and Methods

The electrocardiograms of 2,741 subjects with abnormal left axis deviation were reviewed. These comprise approximately 11% of the patients who had had an electrocardiogram performed at the VA Center in Dayton, Ohio during this period. The electrocardiograms bearing the diagnosis of left axis deviation were carefully reviewed and all patients whose electrocardiographic recordings met standard ECG criteria for left or right ventricular hypertrophy, left or right bundle branch block, initial force deformity consistent with myocardial infarction and pre-excitation syndrome, known to influence the intraventricular conduction time, were excluded.12 Similarly, subjects receiving antiarrhythmic agents at the time of electrocardiographic recordings, i.e., quinidine, procainamide, diphenylhydantoin or propranolol were excluded. All the electrocardiograms were recorded on standard electrocardiographic machines at a paper speed of 25 mm/sec. The definition of left axis deviation (LAD) for the purpose of this study was the characteristic biphasic or negative QRS complexes in lead II (AQRS ≥ -30°) and an initial QRS vector directed inferiorly as evidenced by an R wave in lead aVF.

In order to make a valid comparison, only those subjects who had had at least one complete electrocardiogram prior to the observation of left axis deviation were accepted for the study. Sixty-three subjects who met all these criteria formed the basis for this study. Each subject served as his own control. The paired t-test was used for statistical analysis.

Leads I, II and aVF were selected for the calculation of AQRS and the intraventricular conduction time as measured in the QRS duration. All 63 pairs of electrocardiograms (pre and post left axis deviation) were assigned random code numbers. The selected leads from the electrocardiogram were mounted and photographically magnified 4 to 5 times their original size (fig. 1). The mean AQRS was derived using the equations for a rectangle (fig. 2) and the area under the QRS complex in lead I and aVF,13 which were electronically measured with a digitizer (HP 9864A). The blank portion of electrocardiographic paper in each instance served as a standard to prime the digitizer prior to actual area measurements. The intraventricular conduction time (QRS duration) was similarly digitized from the onset of the initial deflection to the termination of the QRS complex marked by an independent observer in all three leads and was averaged. By utilizing all three leads in the measurement of intraventricular conduction time, errors arising from a rotational change in QRS vectors on intraventricular conduction time in a given single lead were minimized. All the AQRS and the intraventricular conduction times were measured in triplicate, averaged and rounded to the nearest integer by a technician well trained in the use of the digitizer. The accuracy of our measurement with photographification and the use of electronic digitizer was ± 2 msec. At the completion of the measurements, the electrocardiograms were decoded and the findings from the pre and post left axis deviation were compared.

Results

The clinical diagnoses among the patients are shown in table 1. Fifty percent of the subjects had coronary artery dis-
FIGURE 1. Photographic magnification of the actual ECG trace which is shown in the left lower hand corner. Leads I, II and AVF. 052 & 109 are random numbers assigned to the ECG trace.

FIGURE 2. The equations used in the calculation of mean frontal plane QRS axis. AVF = lead AVF, I = lead I, R = resultant, \( \theta \) = the angle between the calculated resultant QRS and lead I (AQRS).

FIGURE 3. Routine ECG before (1967) and after (1969) the development of the axis deviation. The QRS angle changed from 30° to –50° and the intraventricular conduction time increased from 73 to 110 msec.

Table 1. Clinical Diagnosis of Patients with Left Axis Deviation

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery disease</td>
<td>32</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>11</td>
</tr>
<tr>
<td>Primary myocardial disease</td>
<td>7</td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td>3</td>
</tr>
<tr>
<td>Chronic obstructive lung disease</td>
<td>1</td>
</tr>
<tr>
<td>Unknown (routine ECG)</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>63</td>
</tr>
</tbody>
</table>

58.8 ± 1.31 years (33–77). With the development of the left axis deviation the mean QRS angle changed from 23 ± 3.4 (79 to –26) to –40 ± 1.6 (–30 to –76) \( P < 0.001 \), while the mean intraventricular conduction time increased from 73 ± 1.5 (50–95) to 98 ± 1.4 (75–121) msec \( P < 0.001 \). Table 3 lists the number of subjects by the degree of their axis shift and the increment of increase in their intraventricular conduction times.

An electrocardiographic tracing of one patient is illustrated in figure 3. The intraventricular conduction time increased from 75 to 110 msec when the left axis shift appeared spontaneously.

A close linear correlation \( r = –0.8 \) between individual AQRS and the intraventricular conduction times was observed (fig. 4).

Discussion

The precise incidence of left axis deviation in the general population is not known. Among the healthy asymptomatic adult population an incidence of 0.2 to 1.2% has been reported. O'Reilly and Sokolow encountered a 4.7% incidence of left axis deviation among hospitalized patients. The relationship between the higher incidence of left axis deviation and the increasing age of the patients is well known.

Previous studies of left axis deviation have focused largely on the relationship of the AQRS to body build, height, weight, chest dimensions, anatomical position of the heart in the chest cavity, age, and etiology of cardiac disease. Studies evaluating the effects of naturally evolving left axis deviation on the intraventricular conduction time on the other hand are limited and inconclusive. More recent investigations have provided evidence that in the absence of severe wall myocardial infarction, severe left axis shift results from increased fibrosis and conduction delay in the anterior division of the left bundle branch, and has accordingly been termed left anterior hemiblock (LAHB) or left anterior division block.

Table 2. Age, QRS Axis and Intraventricular Conduction Time Before and After the Development of Left Axis Deviation

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pre-LAD (Mean ± SEM)</th>
<th>Post-LAD (Mean ± SEM)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>53.2 ± 1.62</td>
<td>58.8 ± 1.31</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Range</td>
<td>26 to 73</td>
<td>33 to 77</td>
<td></td>
</tr>
<tr>
<td>QRS axis</td>
<td>23 ± 3.4</td>
<td>–40 ± 1.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Range</td>
<td>79 to –26</td>
<td>–30 to –76</td>
<td></td>
</tr>
<tr>
<td>IVCT (msec)</td>
<td>73 ± 1.5</td>
<td>98 ± 1.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Range</td>
<td>50 to 95</td>
<td>75 to 121</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: LAD = left axis deviation; SEM = standard error of the mean; IVCT = intraventricular conduction time.
cardium supplied by the blocked fascicle is delayed, a prolongation in the total intraventricular conduction time would be expected.

In studies where severe left axis shift developed acutely, as during cardiac catheterization, or following cardiac surgery, or when it was induced experimentally, the QRS duration has been observed to increase by 10–20 msec. On the other hand, when compared by group analysis to those with normal QRS axis, His et al. found no increase in the intraventricular conduction time among subjects with left axis deviation. In 55 of 128 subjects (43%), Rosenbaum et al. also found the QRS duration to be within normal limits (60–80 msec). However, an increase of 10–20 msec in the intraventricular conduction time was observed among subjects in whom the axis shift (LASH) occurred intermittently. The present studies clearly delineate an increment of 10 msec or more in the intraventricular conduction time (average 25 msec) in 92% of the subjects, following the natural occurrence of left axis deviation, when individual patients with left axis shift were compared to their control (pre LAD) tracings.

Our results are in close agreement with the observations of Rosenbaum et al., Fernandez et al., Samson and Bruce, and Rothenberger and Winterberger in patients in whom severe left axis shift (LASH) developed either acutely or intermittently or where it was induced experimentally. It is of interest to note that in all of the above situations when an increment in the conduction time was detected, a preblock electrocardiogram was available for comparison. In the absence of such a baseline comparison, it would be difficult to delineate a 10–20 msec change in the intraventricular conduction time particularly since it varies widely in the normal population (50 to 110 msec).

While it seems well documented that severe left axis deviation results from an intraventricular conduction disturbance in man, it has not been demonstrated that lesser degrees of leftward axis shift represent a similar intraventricular conduction defect. From our studies, it appears that there exists a continuum in the relationship of AQRS and the intraventricular conduction time through the counterclockwise range of AQRS from +90° to −90° (fig. 4). These observations are consistent with the hypothesis that the occurrence of any leftward shift in AQRS, including the left anterior hemiblock represent a spectrum of intraventricular conduction delay.

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