Left Axis Deviation

A Reassessment

JOSEPH K. PERLOFF, M.D., NIGEL K. ROBERTS, M.D., AND WILLIAM R. CABEEN, JR., M.D.

SUMMARY This report deals with the ramifications of the concept of left axis deviation. In early life, the leftward shift of the frontal plane QRS axis is determined chiefly, if not solely, by the relative weights of the ventricles. Once adult ventricular weight ratios are reached, there is a long period of axis stability, then a gradual leftward drift of the QRS, governed principally by left anterior fascicular conduction. Thus, the normal QRS axis is age-dependent, and left axis deviation must be considered accordingly.

THE DIRECTION of the mean frontal QRS axis has been studied since derivation of the Einthoven triangle from bipolar leads I, II and III. With the advent of augmented unipolar limb leads, measurement of the QRS axis became more precise. Normal ranges were established, and deviations from normal designated "left" and "right," according to the direction of the shift. The normal QRS axis is age-dependent. Thus, left axis deviation exists when the mean frontal plane axis is equal to or less than 0° in very premature infants (weights below 1000 g), +90° or less in full-term newborns, +75° or less at 3 months, +45° or less in infants older than 6 months and in children, and −30° or less in adults.¹ In early life, the relationship between axis and age is related primarily to the relative weights of the ventricles. In the fetus, up to approximately 30 weeks gestation, the left ventricle outweighs the right.µ The right ventricle then grows disproportionately so that at 32 weeks it is about the same thickness as the left, and at 36 weeks, slightly thicker.¹ After birth, the right ventricle continues to gain weight, but at a much slower rate than the left. By the end of 4 weeks, the left ventricle is heavier; adult ratios are reached before age 6 months.²

The causes and clinical significance of left axis deviation have been controversial since the early days of electrocardiography. The concept of interruption of the anterior fascicle of the left bundle branch — commonly designated "left anterior hemiblock" — as a cause of left axis deviation dominated interest in the past decade.⁴ Evolution of the theory of fibrotic or degenerative left anterior fascicular block resulted in clarification of many misconceptions previously applied to the left superior QRS axis. Concurrently, the term "left axis deviation" was reapplied to several old designations and to some new ones not related to unifascicular block. This progress has generated a considerable amount of information on left axis deviation. Most, if not all, the various patterns can now be separated by applying strict analytical electrocardiographic criteria, thus avoiding the limitations inherent in inappropriate concentrations on left anterior fascicular block.

Despite lively interest, the diverse ramifications of acquired and congenital left axis deviation have not been condensed into a single current essay. It is to this end that our remarks are directed.

Historical Perspective

As early as 1937, Ashman and Hull ascribed left axis deviation to coronary artery disease and left ventricular hypertrophy,⁵ and a decade later Wilson reinforced the latter concept.⁶ Shortly thereafter, the idea of "peri-infarction block" was introduced,⁷ and attention was called to Wilson's comments on intraventricular block.⁸ The notion of peri-infarction block emphasized the importance of coronary artery disease as a cause of left axis deviation, and provided a seemingly rational explanation for it.¹¹ Although in 1951 Grant and Estes elaborated on focal intraventricular block of the peri-infarction type, they stated, "There is evidence that the left bundle bifurcates early

From the Divisions of Cardiology, Departments of Medicine and Pediatrics, School of Medicine, University of California, Los Angeles, California.

Address for reprints: Joseph K. Perloff, M.D., Department of Medicine, Division of Cardiology, Center for the Health Sciences, University of California, Los Angeles, California 90024.

Received August 29, 1978; revision accepted January 31, 1979.

Circulation 60, No. 1, 1979.

into two major sub-branches, one running anteriorly in the septum, and the other posteriorly toward the base of the left ventricle. Conceivably, one or the other of these primary branches of the left bundle could be independently blocked. . . . At the present, the QRS loop deformities which might be produced by one or the other of these types of left branch block are conjectural.13" In 1956, Grant published an elaborate electrocardiographic-pathologic correlation study of 672 cases in which the roles of left ventricular hypertrophy and myocardial infarction as causes of left axis deviation were reexamined in addition to the contributions of body build (kyphoscoliosis), chronic pulmonary disease and various types of "parietal block."14 Three years later, photographs were published of the septal surface of the left ventricle swabbed with cotton soaked in aqueous Lugol's solution, purporting to show that fibers of the left bundle divided into two branches.15 Then, an electrocardiographic-necropsy study of adults with marked left axis deviation called attention to proposed roles of altered anatomic and electrical positions of the heart, emphysema, left bundle branch block, and "factors associated with aging."16 These studies did not explain whether left ventricular hypertrophy or myocardial fibrosis was the principal etiologic factor in left axis deviation, but it was recognized that on rare occasions (four of 514 patients) the axis deviation occurred with neither clinical nor necropsy evidence of heart disease.17 Credence was soon lent to Grant's postulate14 that left axis deviation could be caused by block in the superior division of the left bundle; the acute occurrence of left axis deviation was observed at surgery when an aortic valve dilator passed through the outflow tract of the left ventricle, presumably injuring fibers of the superior division of the left bundle.18 It was later reemphasized, however, that left axis deviation occurred in a wide variety of clinical disorders, including myocardial fibrosis, infarction, cardiomyopathies, left ventricular hypertrophy, congenital or acquired interruption of segments of the conduction system, alterations in electrical resistance of pulmonary tissue (emphysema), and apparently in some healthy adult males.18 The increase in left axis deviation with age was attributed to the development of organic heart disease, especially coronary artery disease or systemic hypertension.19

An early anatomical study of the normal human atrioventricular conduction system cast doubt on the presence of two major subdivisions of the left bundle branch,19 but other investigators, in a study of intraventricular block in the dog, selectively cut what was believed to be the anterior division of the left bundle branch, causing a superior shift in the terminal forces of the QRS.20 Lenegre21 drew attention to bilateral bundle branch block as a cause of complete atrioventricular block — an idea first reported in 191222 and demonstrated experimentally in 1926.28 Lenegre identified the fault as "sclero-degenerative replacement of both branches," but, importantly, emphasized that in the majority of cases the real cause of the lesion was not ischemia, but was in fact un-

unknown.21 Lev, who studied a similar group of patients with bilateral bundle branch block, found fibrosis of the fascicles at necropsy.24 These observations set the stage for the current view that isolated, age-related degenerative disease of the cardiac fibroskeleton can cause a variety of infranodal conduction defects that are unrelated to coexisting myocardial disease or coronary artery obstruction, which may be negligible or absent. Left bundle branch block with left axis deviation was attributed to proximal interruption of the anterolateral division of the left bundle (left axis deviation) with parietal conduction delay beyond that site (QRS prolongation resembling left bundle branch block).26 In such cases, the initial forces of the QRS were normal, and only the anterolateral division of the left bundle branch was blocked. Experimental laceration of the left anterior aspect of the primate (baboon) ventricular septum was then shown to produce marked left axis deviation ascribed to interruption of the anterior rami of the left bundle branch.28 Neither thickness of the left ventricular wall per se, nor electrical position of the heart, were considered important in the development of left axis deviation.29 An electrocardiographic-necropsy study of 96 adults reaffirmed that left ventricular hypertrophy in itself was not responsible for left axis deviation, and implicated instead a conduction defect in the anterior division of the left bundle branch.30

In 1966, a comprehensive study by Pryor et al. of the clinical significance of left axis deviation dealt with the roles of both acquired and congenital heart disease, emphasizing that the left axis shift occurred in endocardial cushion defect, congenitally corrected transposition of the great arteries, type B preexcitation, tricuspid atresia and single ventricle.28 These authors not only suggested hyperkalemia as a newly recognized reversible cause of left axis deviation, but also commented on "pseudo-left axis deviation" in pulmonary emphysema, and observed that surgical injury to the superior division of the left bundle could cause left axis deviation after operation for congenital discrete aortic stenosis or idiopathic hypertrophic subaortic stenosis.28 There was conflicting opinion as to whether two anatomically distinct left branches existed, but the authors argued compellingly that it was conceptually important to consider the fibers of the left bundle as if they were arranged in superior and inferior divisions in order to approach rationally the diseases that alter the sequence of depolarization of myocardium supplied by these proposed radiations.28 Other investigators soon considered the gradual leftward migration of the frontal QRS axis, concluding that this electrocardiographic trend was a common sequel of aging, independent of the population prevalence of coronary atherosclerosis.29 Soon thereafter, a study of 353 necropsy patients ages 19–94 years indicated that the lesion causing left axis deviation in most cases involved the anterior-superior division of the left bundle branch.30

In 1969 and 1970, the publications of Rosenbaum and colleagues stimulated interest in the three main fascicles of the intraventricular conduction system —
the right bundle branch and two divisions of the left. The concept of the hemiblocks was appealing, and the terms left anterior hemiblock, left posterior hemiblock, and trifascicular block were introduced and widely used. Different combinations and degrees of bifascicular and trifascicular block were formulated, and it was pointed out that each division of the left bundle branch could separately exhibit first-, second-, or third-degree block. Left anterior hemiblock emerged as a principal basis for left axis deviation. "Lenegre's disease" or "Lev's disease" was considered a degenerative process variably affecting the trifascicular conduction system, of which the left posterior division emerged as the least vulnerable.

Rosenbaum argued that in left anterior hemiblock, there was delayed activation of the anterior wall of the left ventricle because the depolarizing impulse could reach that zone only via the intact posterior division. This delay resulted in reorientation of the direction of depolarization of the middle and late QRS forces (60 msec) to about −60°. Since depolarization of the segment of myocardium supplied by the blocked fascicle was delayed, prolongation of intraventricular conduction time was anticipated. Although Rosenbaum found the QRS duration within normal limits, he observed an increase of 10–20 msec in intraventricular conduction time in subjects with intermittent left anterior hemiblock. This increment is difficult to recognize without comparison with an unblocked tracing in the same patient. A more current investigation restudied this point and documented an increase of 10 msec or more (average 25 msec) in intraventricular conduction time in adults after natural occurrence of left axis deviation; ECGs from individual patients were compared with their own control (pre-left axis deviation) tracings.

Elegant pathologic studies of the left bundle provided support for Rosenbaum’s postulates. Despite some variation in the structure and branching patterns of the left bundle, there is, as a rule, a slender anterior division and paired, equal-sized components of the posterior division. Two discrete fascicles as such occur rarely, and in most cases there are many communications between the divisions. Significant morphologic changes within the fascicles of patients with left anterior hemiblock have not been consistently identified, but physiologically in both man and dogs there is support for the anatomic concept.

Complete block of conduction in the left anterior fascicle is not necessary to produce left axis deviation; presumably, all that is required is enough delay in anterior fascicular conduction to result in activation of the anterior left ventricular sole via the posterior fascicle. Thus, in "fascicular block," left axis deviation can be interpreted as either delayed conduction or complete block in the left anterior fascicle. The distinction between these alternatives may be important in judging the prognosis of left axis deviation associated with right bundle branch block.

Interest has recently been focused on conduction defects in the unbranched portion of the His bundle (longitudinal dissociation). A discrete intra-His bundle lesion producing delayed conduction or block in fibers destined to branch and reach the left anterior fascicle has been postulated as a cause of left axis deviation, analogous to infranodal left anterior fascicular block. Support for this thesis stems from observations on human subjects studied with His bundle electrograms and in dogs studied with microelectrodes. If these conclusions are correct, "fascicular block" can be intra- or infra-hisian.

Prevalence

The prevalence of left axis deviation according to age and sex has been studied extensively. In analysis of ECGs from 500 Royal Canadian Air Force members, left axis deviation was identified in 1.2%, and in a 10-year follow-up of the ECGs of 1000 healthy young male aviators (mean age 23.7 years), the QRS mean frontal plane axis shifted gradually to the left, though only 0.2% showed electrical axes of less than 0°. Other investigators confirmed and extended these conclusions, examining 1000 ECGs chosen by random selection for each 5-year segment of the adult population (less than 20 years to 45 years or older) from the records of U.S. Air Force flying personnel. There was a gradual shift of the frontal plane QRS axis with increasing age after 20 years. The leftward shift in each age group was independent of QRS amplitude or changes in body weight; a shift above 0° was found in only 3.9% of the entire group (8.6% of those 45 years or older), and a shift superior to −30° was present in only 1.1% (2.4% of those 45 years or older). When ECGs were recorded as part of a survey of 1303 men and 1348 women older than 15 years who belonged to a large religious isolate in northwestern United States and southwestern Canada, a progressive leftward shift of the frontal QRS axis occurred with age, but healthy persons, including those aged 60–90 years, did not reach a QRS axis of −30°; there was no significant sex difference in the prevalence of left axis deviation which, in the group as a whole, ranged from 1.7–4.9%.

In an investigation of the prevalence, associated diseases and prognosis of left axis deviation (defined as a mean frontal QRS axis of −30° or less) in 248 of 4678 persons older than age 20 years, the frequency of the axis deviation increased with age among both sexes, but men had a higher incidence than women. A population study of 8000 Japanese-American men aged 45–69 years distinguished left axis deviation from left anterior hemiblock (defining the difference as frontal QRS axes of −30° to −44° and −45° to −90°, respectively); left axis deviation was found in 2.6% and left anterior hemiblock in 1.5%. In fascicular disease, there is currently a uniform consensus that frontal plane axes above −45° are properly designated left anterior hemiblock, while axes below −30° are neither left anterior hemiblock nor left axis deviation. Opinions differ on the proper designation of axes between −30° and −45°, some authors holding that
the term left axis deviation is appropriate, while most believing that left anterior hemiblock is properly applied.

**Variations on the Theme of Left Anterior Hemiblock**

**Left Axis Deviation in Acquired Heart Disease (table 1)**

In discussing acquired left axis deviation, we deal with specific categories of disease (or presumed disease) and especially with the mechanisms (or presumed mechanisms) believed to be responsible for the axis shift. What causes the gradual leftward shift of the frontal QRS axis that occurs during the course of normal adult aging? Is this axis shift one end of a spectrum that culminates in frank left axis deviation as a benign concomitant of advancing years? Lenegre described progressive fibrodegenerative disease in the human atrioventricular conduction system, setting the stage for the current view that the amount of connective tissue in the cardiac fibroskeleton and specialized conduction pathways “normally” increases with age.

Can this process, in the absence of or apart from coexisting heart disease, impair conduction via the fascicles and ultimately result in left axis deviation due to left anterior fascicular delay or block? Is the mechanism underlying the moderate leftward shift in the frontal QRS axis (below −30°) the same or qualitatively different from axes above −45° due to left anterior hemiblock? Can we confidently distinguish the contributions of the effects of natural or physiologic aging from the effects of cardiac disease — occult or overt — that prevail in the same adult population? Although it is now generally accepted that marked left axis deviation (−45° or greater) can result from blocked conduction in the left anterior fascicle, it has not been proven that smaller degrees of leftward shift of the frontal QRS axis with aging represent a similar, though smaller, conduction defect. There is evidence, however, that a continuum exists in the relationship of the QRS axis and intraventricular (left anterior fascicular) conduction time through the range of +90° to −90°.

If these observations are correct, then any leftward shift in the QRS axis with normal adult aging, including left anterior hemiblock, would represent part of a spectrum of unifascicular conduction delay. In the study of 8000 Japanese-American men referred to earlier, more than 60% with frontal plane QRS axes of −30° to −90° (normal QRS duration) were believed to have no other cardiovascular abnormality. The incidence of coronary artery disease, fatal or nonfatal, in this group during observation periods of 3–6 years was not significantly different from control groups of normal men. Similarly, 50% of persons past age 20 years with mean QRS frontal axes of −30° or less had no evidence of heart disease and no excessive incidence of cardiac morbidity or mortality during an average observation period of 4 years.

In discussing the sequence or coincidence of atherosclerotic coronary artery disease and left axis deviation, electrocardiographic-pathologic correlative studies have identified frequent myocardial fibrosis and infarction attributed to coronary artery disease. It is rational to infer, in this setting, that ischemic heart disease in its own right causes fibrosis that partially or completely interrupts conduction in one or more fascicles. This mechanism presupposes a decrease in flow via the coronary circulation to or relatively near specialized atrioventricular conduction pathways, especially the infranodal system. The impendence could result from either reduced flow through the primary arterial supply or, if that source is obstructed, through collateral circulation. The sudden and sometimes transient occurrence of isolated left anterior hemiblock during acute myocardial infarction supports this thesis.

There are two chief sources of blood supply to the His bundle and proximal portion of its main branches — the atrioventricular node artery and the first septal perforator of the left anterior descending coronary artery. In addition, a variety of anastomoses provide important secondary sources from Kugel’s artery, from the first few septal branches of the posterior descending artery, from the second perforating branch of the left anterior descending coronary artery, from the posterior left and right atrial arteries, and branches to the crista supraventricularis from the right coronary artery. The relatively thin, vulnerable anterior portion of the left bundle branch can either be dually supplied by primary sources from the first septal branch of the left anterior descending and atrioventricular node artery, or be supplied entirely from the septal branch, or, rarely, by the atrioventricular node artery alone. Therefore, either anterolateral or inferior myocardial infarction could affect a portion of the blood supply to the left anterior fascicle. Left axis deviation has been found to be considerably more common in anterolateral infarction, a conclusion reached in 1956 by Grant, who attributed the axis shift to “peri-infarction block.”

An electrocardiographic-necropsy study also found that left axis deviation was more frequent with anterolateral infarction, but the incidence of inferior infarction was not low, although the majority of patients with inferior infarction had involvement of at least some portion of the anterior wall of the left ventricle. Although dual primary blood supply to the left anterior fascicle sets the stage for potential left anterior hemiblock with either anterolateral or in-

<table>
<thead>
<tr>
<th>Table 1. Left Axis Deviation in Acquired Heart Disease: Variations on the Theme of Left Anterior Hemiblock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aging of the cardiac fibroskeleton</td>
</tr>
<tr>
<td>Atherosclerotic coronary artery disease</td>
</tr>
<tr>
<td>“Peri-infarction block”</td>
</tr>
<tr>
<td>Left ventricular hypertrophy, concentric/asymmetric</td>
</tr>
<tr>
<td>Focal anatomic lesions</td>
</tr>
<tr>
<td>Neuromuscular disorders</td>
</tr>
<tr>
<td>Hyperkalemia</td>
</tr>
</tbody>
</table>
by patient fibrosis, especially leftward shift of left anterior hemiblock. It has been reaffirmed that a large inferior infarction can cause a left and superior QRS axis of less than $-30^\circ$ in the absence of left anterior hemiblock. However, the electrocardiographic recognition of inferior infarction in the presence of left anterior hemiblock is difficult, and diagnostic accuracy benefits from vectorcardiographic analysis.

Is peri-infarction block as a cause of left axis deviation still a viable concept? This idea, as proposed by First et al. in 1950 and elaborated upon by Grant, was based on the assumption that the block was intraventricular, i.e., near the zone of infarction. For example, it was argued that infarcts characterized by Q waves in leads I and aVL, the terminal forces, because of delayed activation of the adjacent left ventricular wall, would be unopposed and point in a direction opposite the initial QRS forces, thus, superior and to the left. However, the term “peri-infarction block” was rapidly qualified and, if any, forms of left axis deviation are considered the result of parietal (peri-infarction) block; instead, they represent block in the left anterior fascicle.

Left ventricular hypertrophy has long been considered an important cause of left axis deviation. In 1944 Wilson et al. reported that the axis shifted because the endocardial-to-epicardial QRS vectors produced a wavefront that was oblique and not perpendicular to the surface of the hypertrophied ventricle. Early evidence that left ventricular hypertrophy played a role in producing left axis deviation was circumstantial (guilt by association), but it became evident that the basic cause of the axis shift was not the hypertrophy itself, but rather the presence and location of associated myocardial fibrosis, especially of the anterolateral wall. Concentric left ventricular hypertrophy per se changes the amplitude, but not the direction of the QRS, while right ventricular hypertrophy adds new directional electrical forces to the QRS vector, causing shifts in the frontal plane axis to the right, culminating in frank right axis deviation. The current consensus is that the leftward shift of the mean frontal plane QRS axis in left ventricular hypertrophy results from delayed anterior fascicular conduction due to fibrosis.

Is this also the case with hypertrophic cardiomyopathy (asymmetric septal hypertrophy), in which a leftward shift of the QRS axis is relatively common and varies from slight to marked ($-90^\circ$)? Although disproportionate septal hypertrophy is, by definition, always present, the scalar ECG may show left ventricular hypertrophy without left axis deviation, and conversely, may reveal left axis deviation with or without hypertrophy. The major site of septal thickening is typically close to the infranodal conduction pathways. There is presumptive evidence that a leftward shift in the frontal QRS axis results from fibrosis involving the anterior division of the left bundle branch as in concentric left ventricular hypertrophy. However, it is not clear whether this is also the case in infants with asymmetric septal hypertrophy, especially the eccentric hypertrophy occasionally associated with 46 XX, XY Turner phenotype in which marked left axis deviation is the rule (beyond $-60^\circ$). Myotomy or myomectomy of the hypertrophied septum consistently results in alterations in conduction. The most common postoperative abnormality is complete left bundle branch block, but some patients develop an abrupt intraoperative leftward shift of the mean QRS axis characteristic of left anterior hemiblock.

Focal anatomic lesions caused by several cardiac disorders can interrupt conduction in the left anterior fascicle. For example, this mechanism is believed to be the basis for left axis deviation in congestive cardiomyopathies (acute, chronic or healed) and restrictive cardiomyopathies (infiltrative diseases). Left axis deviation caused by left anterior hemiblock in certain neuromuscular disorders should be considered separately. In myotonic muscular dystrophy, for example, the most common infranodal electrocardiographic abnormality is defective conduction in the left bundle branch system — left bundle branch block or left axis deviation. The dominant site of cardiac involvement appears to be the specialized conduction system — sinus node, atrioventricular node, infra-His and His-Purkinje system, analogous to disorders of impulse formation and conduction in much older subjects. Thus, the primary cardiac disturbance — premature degenerative disease of the cardiac fibroskeleton — may represent another of the non-myopathic manifestations of myotonic dystrophy, and may reveal itself in part as left anterior hemiblock.

In 1955 attention was drawn to a relationship between potassium concentrations and the frontal plane QRS axis in dogs. Subsequently, a number of observations confirmed that hyperkalemia as well as a sudden increase in serum potassium levels are sometimes accompanied by left axis deviation. The immediate cause of the axis shift has been ascribed to anterior fascicular block believed to stem from decreases in resting membrane potential and transmembrane potassium gradient which cause a decreased rate of rise of the action potential and slowed or decremental conduction or block. This same mechanism is held responsible for the generalized QRS widening with hyperkalemia. Thus, the overt focal disturbance in left anterior fascicle conduction results from preexisting occult fascicular disease made selectively manifest by hyperkalemia.

**Acquired Left Axis Deviation in Congenital Heart Disease (table 2)**

Anomalous origin of the left coronary artery from the pulmonary trunk is a congenital disorder in which acquired left axis deviation may develop. Infarction is accompanied by severe hypertrophy of uninfarcted zones of left ventricle. Thus, myocardial infarction and an eccentric form of left ventricular hypertrophy
coexist. In some survivors, left axis deviation occurs, although its cause is not clear. It has been proposed that the leftward shift of the frontal QRS axis is related in part to the disproportionate thickness of the posterobasal left ventricular wall. If an increase in muscle mass attracts the major vector of depolarization toward it, then posterobasal hypertrophy might shift the axis posteriorly, if not superiorly. However, the mechanism of left axis deviation is probably related less to eccentric hypertrophy than to infarction, causing left anterior hemiblock, analogous to adults with obstructive coronary artery disease. This view is supported by the changes in mean frontal QRS axis that sometimes occur after ligation of the anomalous left coronary artery in infants. Preoperative left axis deviation occasionally normalizes, but sometimes the superior and leftward axis develops after coronary ligation. These responses appear to reflect variations in coronary arterial flow to the left anterior fascicle, since operation, even if successful in diminishing myocardial ischemia, cannot affect the degree of left ventricular hypertrophy during the observed time course.

Rarely, left axis deviation develops when a normal neonatal left ventricle is abruptly exposed to a massive volume load that results in acute dilatation. This may occur when a large congenital pulmonary arteriovenous fistula suddenly becomes functional with expansion of the lungs at birth. This is another form of congenital heart disease in which left axis deviation may be acquired. Pulmonary arterial blood preferentially flows via the low-resistance fistula, so that a large volume is abruptly imposed upon the left heart. The frontal QRS axis may shift as far leftward as 0° (left axis deviation in the mature neonate). The cause of the shift is unclear. It is difficult to assign the cause to fibrosis of the left anterior fascicle, but if transient ischemia were the cause, surgical abolition of the fistula would be expected to normalize the axis, and this is not the case.

Intracardiac repair of Fallot's tetralogy is accompanied by an electrocardiographic pattern resembling complete right bundle branch block (whether or not the correction involves a right ventriculotomy), but the occasional development of left axis deviation after surgery for this anomaly is relevant. Direct interruption of the anterior division of the left bundle branch is believed to account for the axis shift.

Accordingly, left axis deviation in postoperative Fallot's tetralogy is a variety of acquired left anterior hemiblock in congenital heart disease.

Uncomplicated primary endocardial fibroelastosis of the dilated type is typically seen in infancy and is characterized by diffuse opaque, gray thickening of the endocardium due to proliferation of collagenous and elastic tissue. The left ventricle is exclusively or dominantly involved. The QRS axis in the frontal plane is usually normal, but occasionally leftward shifts occur. The mechanism of the leftward axis is not known, but fibrosis in the left ventricular wall may impair conduction in the left anterior fascicle. If so, the relative infrequency of left axis deviation may relate in part to the shortened life span of the average patient.

### Acquired Left Axis Deviation Unrelated to Left Anterior Hemiblock (Table 3)

A simple way to illustrate left axis deviation unrelated to left anterior hemiblock is the common observation on right ventricular ectopic rhythms, primarily those in response to transvenous right ventricular pacemakers. Pacing from the apex of the right ventricle (standard location for permanent transvenous pacemakers) typically results in a superior and leftward wavefront — left axis deviation — not present in the unpaced ECG. Occasionally, a right ventricular paced beat provokes retrograde atrial activation, then a reciprocal or echo beat that does not have the left axis deviation and left bundle branch block of the paced beats.

Pulmonary emphysema as a cause of left axis deviation was recognized as early as 1952. The observation that lung disease can be responsible for a leftward mean frontal QRS axis — sometimes as marked as <−90° — has been reconfirmed, but the mechanism remains unsettled. In 1956, Grant speculated that the axis shift could be related to reduced lateral electrical conductance of the hyperinflated lungs. He reasoned that the electrical field surrounding the heart might be concentrated vertically so the limb leads would record principally the superiorly (or inferiorly) directed components of the electrical activity. Thus, QRS forces pointing toward the left arm would be recorded as strictly superior (or conversely, QRS forces directed toward the left leg would be recorded as strictly inferior).

These postulates were supported by Spodick, who pointed out that a QRS axis of −90° (or +90°) in the frontal plane is an “axis-illusion,” since the sagittal projection shows a much less superior (or inferior) orientation. A better explanation than Grant’s has not been offered, and the term “pseudo-left axis deviation” has been applied. Severe kyphoscoliosis with left axis deviation is probably a variation on this. Since the majority of patients with

### Table 2. Acquired Left Axis Deviation in Congenital Heart Disease

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomalous origin of the left coronary artery from the pulmonary trunk</td>
</tr>
<tr>
<td>Large pulmonary atrioventricular fistula</td>
</tr>
<tr>
<td>Surgical repair of Fallot’s tetralogy</td>
</tr>
<tr>
<td>Endocardial fibroelastosis (dilated type)</td>
</tr>
</tbody>
</table>

### Table 3. Acquired Left Axis Deviation Unrelated to Left Anterior Hemiblock

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right ventricular rhythms</td>
</tr>
<tr>
<td>Pulmonary emphysema (an axis illusion)</td>
</tr>
<tr>
<td>Anatomical lie of the situs solitus heart</td>
</tr>
</tbody>
</table>
kyphoscoliosis have curvature to the right, it is the left lung volume that increases and may, accordingly, reduce lateral conductance in that direction analogous to emphysema.

The QRS axis is not significantly dependent upon the anatomical lie of the situs solitus heart, even though variations in the heart’s anatomical lie are sometimes associated with minor changes in mean frontal plane QRS direction; consider the slight to moderate leftward shifts that occur when pregnancy or ascites elevates the left hemidiaphragm. Disturbances such as pneumothorax or pleural effusion usually cause a pronounced shift of the heart to the opposite side of the chest, but do not necessarily affect the frontal plane QRS axis.

Congenital Left Axis Deviation (table 4)

In congenital forms of left axis deviation, the mechanisms are more complex, varied and ill-defined, and may or may not be due to left anterior hemiblock. The purest form of congenital left axis deviation can be said to exist if an infant is born with isolated left anterior hemiblock and no other cardiac or vascular disease. It is not certain whether this occurs, but in 1965 Gup et al. identified what they thought was isolated congenital left axis deviation in six young children in good health and without evidence of heart disease. There is evidence from other sources that congenital (or at least genetic) abnormalities occur in the specialized atrioventricular conduction tissues. The combination of familial ostium secundum atrial septal defect with congenital PR prolongation and left axis deviation has been described, and occasionally, members of these families have first-degree heart block and left axis deviation without an atrial septal defect.

In the Wolff-Parkinson-White syndrome, a right-sided bypass tract (so-called type B) may result in preexcitation of the posterior atrioventricular margin near the septum. Late activation of the anterior and superior portions of the heart then results in a negative inferior field, directing the mean frontal QRS axis upward and to the left (left axis deviation). In addition, bypass tracts to the posterior left ventricle can produce a left superior QRS axis. Type B preexcitation occurs as an isolated disorder, but is found in 15–25% of ECGs from patients with Ebstein’s anomaly. All known examples of preexcitation accompanying this malformation have been type B; some have resulted in left axis deviation, based on the above mechanisms.

In 1956, Toscano-Barbosa described the ECG associated with malformations of the endocardial cushions. Comparing the tracings to those from patients with ostium secundum atrial septal defects, he stated, “The similarity that may exist in the precordial leads may lead one astray from the differences that almost universally exist in the extremity leads which are of real discriminatory value. Expressed most simply in the traditional interpretation of the standard leads as left axis deviation, the characteristic features may be more completely portrayed by vectorial analysis.” The mechanisms of left axis deviation in endocardial cushion defects appear to be related to congenital alterations of excitation pathways into the ventricles, as originally proposed by Toscano-Barbosa et al.

Anatomic investigations of the atrioventricular conduction system in man and in the dog have clarified this QRS pattern. The atrioventricular node is posteriorly and inferiorly displaced in relation to the coronary sinus; the His bundle is shorter than normal and posteriorly displaced; the left bundle branch is also relatively posterior, and arises from the common bundle soon after it enters the ventricular septum (early origin); the left anterior division of the left bundle branch has fewer fibers than normal (hypoplasia) and an increased length, while the posterior division is shorter than normal and may provide small branches to the posterobasal wall of the left ventricle; the right bundle branch is abnormally long. These anatomic patterns have been correlated with electrophysiologic observations.

Short HV intervals are in accord with data from more elaborate studies that showed early activation of the posterobasal left ventricular wall both in man and in a dog with congenital endocardial cushion defect. Abnormally early posterobasal activation is consistent with posterior displacement of the bundle branch and a short posterior division that gives rise to branches to the posterobasal wall; delayed activation of the anterior superior wall is in accord with hypoplasia or increased length of the anterior division, which does not have interrupted conduction as such. Accordingly, the anatomy of the atrioventricular conduction system in endocardial cushion defect, together with electrophysiologic observations on infranodal conduction, are acceptable explanations for the left axis deviation. A similar mechanism applies to the left axis deviation in common atrium (complete absence of the atrial septum), which necessarily means that the septum primum is deficient, so that elements of the endocardial cushion malformation coexist.

Left axis deviation accompanying tricuspid atresia was discussed in 1929 by Rihl, and the diagnostic value of this axis was reemphasized by Taussig in 1936. An abnormal leftward shift of the frontal plane QRS axis occurs in 75–85% of such patients, but the cause of the left axis deviation remains an

TABLE 4. Congenital Left Axis Deviation

| Congenital left axis deviation | Wolff-Parkinson-White type B (isolated) | Type B Wolff-Parkinson-White in Ebstein’s anomaly | Endocardial cushion effect | Tricuspid atresia | Single ventricle | Congenitally corrected transposition of the great arteries | Double outlet right ventricle with infraeristral ventricular septal defect |
enigma. In one study of the conduction system in tricuspid atresia, the atioventricular node and His bundle were normally located, but the course of the bundle branches differed from normal in that the left bundle branches originated unusually close to the node/bundle junction (early arborization), and the right bundle branch was markedly elongated in its course to the right septal endocardium. However, it is difficult to establish a relationship between either of these anatomic patterns and the frontal QRS axis.

Left anterior hemiblock has also been considered as a mechanism of the left axis deviation. This proposal is not supported by the observation that in one case of tricuspid atresia with typical counterclockwise left superior axis, right axis deviation developed postoperatively, but the patient never had complete left bundle branch block, implying that the preoperative left axis deviation was not the result of permanent block in the left anterior fascicle. Plausible though unlikely alternative explanations are alternating left anterior and left posterior hemiblock, or initial left axis deviation due to anterior fascicular delay rather than block. Consideration has also been given to the idea that left axis deviation in tricuspid atresia results from unopposed left ventricular electrical forces. This hypothesis is supported by the tendency for left axis deviation to occur when the right ventricle is markedly hypoplastic, and for a normal or right axis to occur when the right ventricle is relatively well-developed, as in tricuspid atresia with large ventricular septal defect.

Left axis deviation sometimes occurs in single ventricle, a congenital anomaly characterized in its most typical form by absence of the right ventricular sinus (inflow portion), leaving a morphologic left ventricle (or primitive ventricle) that contains at its base a small outlet chamber (the right ventricular infundibulum). The conduction system has been studied in detail, but the differences from normal do not provide an explanation for the left axis deviation. However, a loose relationship exists between the anatomic position of the outlet chamber and the frontal electrical axis and QRS pattern. The mean QRS axis in the frontal plane has a tendency to point toward the major muscle mass of the single ventricle and away from the location of the infundibulum. Thus, when the infundibular chamber is located at the right basal aspect of the ventricle (noninverted position), the QRS axis is usually directed to the left, pointing either upward and to the left — left axis deviation — or downward and to the left.

Still another variety of left axis deviation occurs in congenitally corrected transposition of the great arteries, an anomaly in which a morphologic right atrium joins a morphologic mitral valve and left ventricle that everts into the pulmonary trunk, while a morphologic left atrium joins an anatomic tricuspid valve and anatomic right ventricle that everts into the aorta. Since the ventricles are reversed (inverted), the conduction system is the reverse of normal; the anatomic “left” bundle lies on the right side of the interventricular septum and vice versa. The anatomy of the conduction system in this anomaly has been carefully studied and though abnormal, does not clarify the mechanism of left axis deviation. The direction of the frontal QRS electrical axis correlates chiefly with pressure in the venous (morphologic left) ventricle. When venous ventricular pressure is low, left axis deviation is the rule.

Although the mechanism of left axis deviation is not entirely clear, it is related in some way to the electrically unopposed arterial (morphologic right) ventricle, i.e., an anatomic right ventricle in the systemic location and a low-pressure anatomic left ventricle in the venous location. Clearly, this form of left axis deviation is unrelated to left anterior hemiblock, since the left bundle branch is in the right heart.

Right ventricular origin of both great arteries (double outlet right ventricle) with infracristal ventricular septal defect and no pulmonic stenosis is associated with an ECG that characteristically displays left axis deviation. In this anomaly, the penetrating portion of the His bundle, after passing through the central fibrous body, comes to lie on the left side of the septum along the posterior wall of the ventricular septal defect. Although the branching left bundle is short and may cause asynchronous ventricular depolarization, the mechanism of left axis deviation is unknown. The axis tends to become more vertical in the presence of progressive pulmonic stenosis, even though right ventricular mass does not increase. However, the initial forces usually remain counterclockwise in the frontal plane, perhaps because of the abnormal distribution of the left bundle.

Acknowledgment

The authors express their appreciation to Sherri Bell for preparation of the manuscript.

References

11. First SR, Bayley RH, Bedford DR: Peri-infarction block:
electrocardiographic abnormality occasionally resembling bundle branch block and local ventricular branch of other types. Circulation 2: 31, 1950
42. Goldberg AN, Kurzynski TW, Hellerstein HK, Steinberg AG: Electrocardiographic findings among the total adult population of a large religious isolate. Circulation 41: 257, 1970
82. Gigli G, Scotti G: Sur un singulier trace electrocardiographique sans un cas de coeur pulmonaire chronique. Acta Cardiol 7: 206, 1952
100. Taussig HB: The clinical and pathological findings in congenital malformations of the heart due to defective development of the right ventricle associated with tricuspid atresia or hypoplasia. Bull Johns Hopkins Hosp 59: 435, 1936
Left axis deviation: a reassessment.
J K Perloff, N K Roberts and W R Cabeen, Jr

Circulation. 1979;60:12-21
doi: 10.1161/01.CIR.60.1.12

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1979 American Heart Association, Inc. All rights reserved.
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/60/1/12

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally
published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the
Editorial Office. Once the online version of the published article for which permission is being requested is
located, click Request Permissions in the middle column of the Web page under Services. Further
information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/