Hemiblocks Revisited

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Abstract*—**The trifascicular nature of the intraventricular conduction system and the concept of trifascicular block and hemiblock were described by Rosenbaum and his coworkers in 1968. Since then, anatomic, pathological, electrophysiological, and clinical studies have confirmed the original description and scarce advances have been developed on the subject. In the present study, we attempt to review and redefine reliable criteria for the electrocardiographic and vectorcardiographic diagnosis of left anterior and posterior hemiblock. One of the most important problems related to hemiblocks is that they may simulate or conceal the electrocardiographic signs of myocardial infarction or myocardial ischemia and may mask or simulate ventricular hypertrophy. Illustrative examples of these associations are shown to help the interpretation of electrocardiograms. The incidence and prevalence of the hemiblocks is presented based on studies performed in hospital patients and general populations. One of the most common causes of hemiblocks is coronary artery disease, and there is a particularly frequent association between anteroseptal myocardial infarction and left anterior hemiblock. The second most important cause is arterial hypertension, followed by cardiomyopathies and Lev and Lenègre diseases. The hemiblocks may also occur in aortic heart disease and congenital cardiopathies. Left anterior hemiblock is more common in men and increases in frequency with advancing age. Evidence is presented regarding the relationship of spontaneous closure of ventricular septal defects, which may explain the finding of this and other conduction defects in young populations. Isolated left anterior hemiblock is a relatively frequent finding in subjects devoid of evidence of structural heart disease. Conversely, isolated left posterior hemiblock is a very rare finding; its prognostic significance is unknown and is commonly associated with right bundle-branch block. The most remarkable feature of this association is that the prognosis is much more serious with a great propensity to develop complete atrioventricular block and Adams-Stoke seizures. (Circulation*. 2007;115:1154-1163.**)

Key Words: epidemiology ■ heart septal defects ■ myocardial infarction ■ heart block ■ bundle-branch block

Thirty-eight years have elapsed since Rosenbaum et al published their seminal work that put together diverse previous observations and brought about a rational and complete analytic approach to the trifascicular concept of intraventricular conduction.

These conclusive studies and an exhaustive review of the preceding knowledge of the subject were first published in the Spanish monograph,[1] and later a shorter updated version was presented to acquaint the English reader with the electrocardiographic manifestations and clinical correlation of the abnormal spread of ventricular excitation.[2] A series of articles published by the same authors between 1969 and 1973 also contributed to the expansion of the recognition of this research work. As stated by Herman N. Uhley,[3] this work triggered and influenced the work of anatomists, pathologists, electrophysiologists, clinicians, and students.

The anatomic, physiological, experimental, clinical, and pathological studies carried out in Argentina showed that, in fact, the intraventricular conduction system has 3, and not 2, terminals: 1 in the right ventricle and 2 in the left ventricle. On the basis of our own anatomic studies in different species, which included 60 human hearts and a careful review of the literature, it became clear that the 2 suspected left ventricular pathways were the anterior and posterior divisions of the left bundle branch (LBB).[1,2] Since then, the world of cardiology has become familiar with a new doctrine and even a new language: a conduction system that was essentially trifascicular and a whole family of conduction disturbances. As in other fields of science, the change in the paradigm had practical consequences that occupied a central position in interdisciplinary progress in the diagnosis and treatment of many cardiac patients.

The purpose of the present study is to briefly refresh various concepts related to the anatomy, pathogenesis, electrocardiographic and vectorcardiographic manifestations, the epidemiology, and clinical implications of blocks in the divisions of the LBB. One of the utmost clinical problems related to hemiblocks is that they can mimic or hide a myocardial infarction or a myocardial ischemia, left ventricular enlargement, or hypertrophy, or they can disguise or conceal a right bundle-branch block (RBBB).

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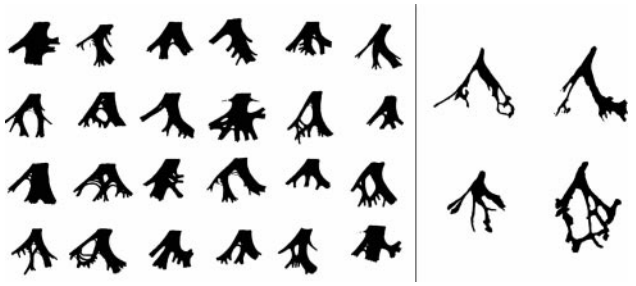

Figure 1. Left, Diagrammatic sketches of the LBB reconstructed from transverse sections of the LBB of human hearts.[5] Right, 4 prototypes of LBB dissected from adult normal human hearts.[1] Note that each of these prototypes has a similar pattern among those obtained histologically.

Anatomy of the LBB: The Middle or Septal Fascicle

Despite the fact that conduction disturbances that involve the anterior and posterior divisions of the LBB described as the hemiblocks were accepted by the cardiological community, some anatomic and electrocardiographic studies proposed that, besides the anterior and posterior fascicles, a middle or septal fascicle can be recognized in the left ventricular conduction system, which might have functional and clinical significance.[4–7] In fact, the existence of midseptal fibers cannot be disregarded, and as such the functional and, probably, clinical significance of a middle or septal fascicle cannot be totally ignored either.[7–9] In accordance with our studies on the anatomy of the LBB, the midseptal fibers are given off in most cases by the posterior division, less frequently by the anterior division or from both, and in few cases have an independent origin from the central part of the main LBB at the site of its bifurcation.[1,2] Figure 1 shows the LBB and its divisions from human hearts, obtained by macroscopic dissection and by reconstruction of the anatomy from serial histological sections of the left septal myocardium. As depicted in Figure 1, in many cases there are abundant interconnections among the main divisions (anterior and posterior) and the middle septal fibers. Although a conduction disturbance in the left septal fascicle may well occur, however, its electrocardiographic recognition is extremely difficult, particularly when present as a fixed pattern. As a matter of fact, its occurrence has been mostly proposed in cases of intraventricular aberrant conduction, transient morphological changes of the QRS, or comparison of ECGs taken before and after its probable occurrence.[7] Moreover, because electrocardiographic and pathological correlation of conduction disturbances is particularly difficult, histological studies of this conduction disturbance would most probably be inconclusive and misleading. In fact, bundle-branch blocks without histological lesions and vice versa have often been reported. Finally, the addition of left anterior hemiblock (LAH) or left posterior hemiblock (LPH) might make even more complex a recognizable change in the QRS configuration produced by left septal fascicular block.

The Diagnosis of Hemiblocks

Apart from the experimental approach and perhaps a few reliable electrocardiographic-pathological correlations, the most important and accurate evidence of all is the study of clinical cases in which conduction is only partially altered and allows intermittent conduction in the fascicle or fascicles under study. Thus, examples of intermittent LAH and LPH during sinus rhythm or intraventricular aberrant conduction of supraventricular premature beats constitute by far the best material for the study of the changes and effects on the QRS complex of any type of fascicular or bundle-branch block in the human ECG and/or vectorcardiogram (VCG).[1,2] Precisely, the first approach of Mauricio B. Rosenbaum to hemiblocks corresponded to a patient with permanent RBBB and an AQRS at −75°, who, a few weeks later, showed an AQRS at +110° and in whom both types of AQRS, in other tracings, were seen to alternate with each other (intermittent LAH and LPH). Thus, it was assumed that the 2 distinctive electrocardiographic patterns indicated the association of RBBB with blocks in the anterior and posterior division of the LBB, respectively.[1,2]

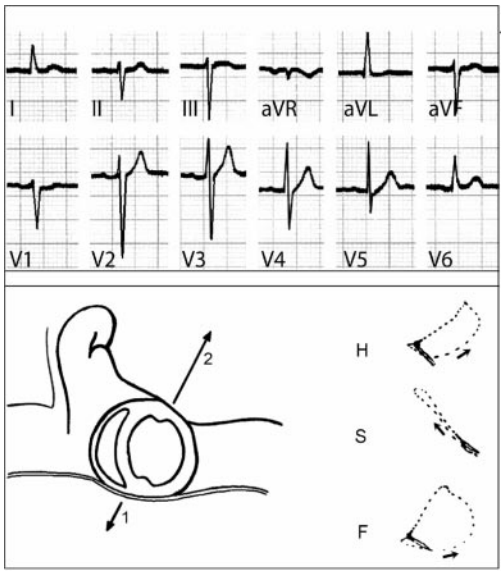

Figure 2. Top, ECG of a typical LAH. Bottom left, Drawing of a frontal section of the heart, which schematically depicts the magnitude and direction of the 2 main electrical vectors of LAH. Vector 1 corresponds to the initial activation of the posteroinferior wall of the left ventricle (R wave in II, III, and aVF). Vector 2 shows the activation process of the anterolateral wall oriented superiorly and to the left (deep S wave in II, III, and aVF; SIII is deeper than SII). Bottom right, The VCG of LAH.

Electrocardiographic and Vectorcardiographic Diagnosis of LAH

Figure 2 depicts the typical ECG and VCG changes produced by LAH in the human: (1) the first 0.02-second QRS vector depicts an inferior and rightward shift in the frontal plane (≈+120°) responsible for the presence of a small Q wave in leads I and aVL and a small and sharp R wave in II, III and aVF. (2) A shift of the main QRS forces superiorly and to the left, which elicits deep S waves in leads II, III, and aVF (note that SIII is deeper than SII). (3) LAH usually shifts the main forces of the AQRS to −45°, −60°, or even to −75° (complete LAH). Because the AQRS may be <−45° in cases of incomplete LAH, the degree of left axis deviation required

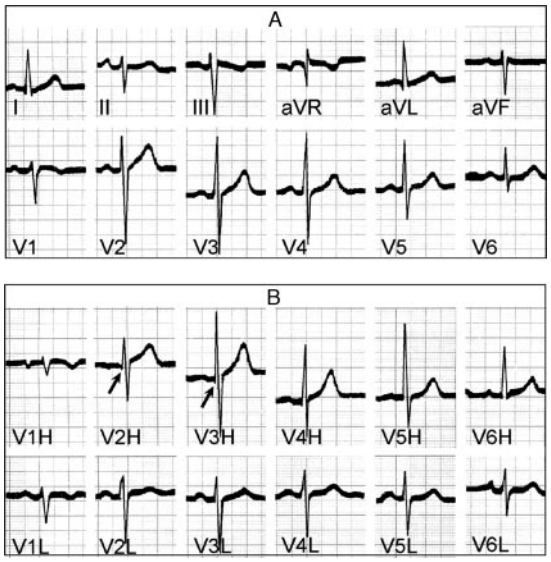

Figure 3. A, Typical LAH that shows the same features described in Figure 2. B, V1H to V6H were recorded 1 intercostal space above the conventional level, and V1L to V6L were recorded 1 interspace below. Note the small Q wave in V2H and V3H (arrows) that are not seen at the normal level (3A) and lower level.

for the accurate diagnosis of complete LAH is −45°.[1,2] (4) All these changes occur with a QRS that widens no more than 0.02 seconds in pure and uncomplicated LAH. The presence of myocardial infarction or left ventricular enlargement may produce a more substantial QRS widening.[1,2] Figure 2 (bottom right) shows the typical vectorcardiographic changes in the frontal plane: small initial forces oriented inferiorly and to the right and the main forces oriented superiorly and to the left with a wide open counterclockwise rotated loop. Although the main changes produced by LAH occur in the limb leads, some distinctive features may also be observed in the precordial leads. As shown in Figure 3, deeper S waves are recorded in leads V5 and V6 as a result of the superiorly directed forces. Accordingly, S waves tend to disappear in leads above the normal level and are deeper when the electrodes are placed below the normal level (Figure 3A and 3B). Both vertical differences in the placement of the recording chest electrodes as well as individual variations in the position of the heart (horizontal or vertical) may produce important changes in the QRS configuration, particularly in the precordial leads V4 to V6. The normal Q waves in the left chest leads may sometimes be absent in LAH, probably because of changes of left to right septal activation. Of note, small sharp Q waves that simulate an old anteroseptal infarction may be seen sometimes in the chest leads V2 and V3 at the normal level and in almost all cases when placed in a higher position. These Q waves correspond to the initial QRS forces directed inferiorly and to the right (Figure 3B, top panel).

It is worth mentioning that LAH may simulate left ventricular hypertrophy in the limb leads I and aVL and, conversely, it may conceal signs of left ventricular hypertrophy in the left precordial leads and may also hide signs of inferior ischemia (Figure 4A and 4B).

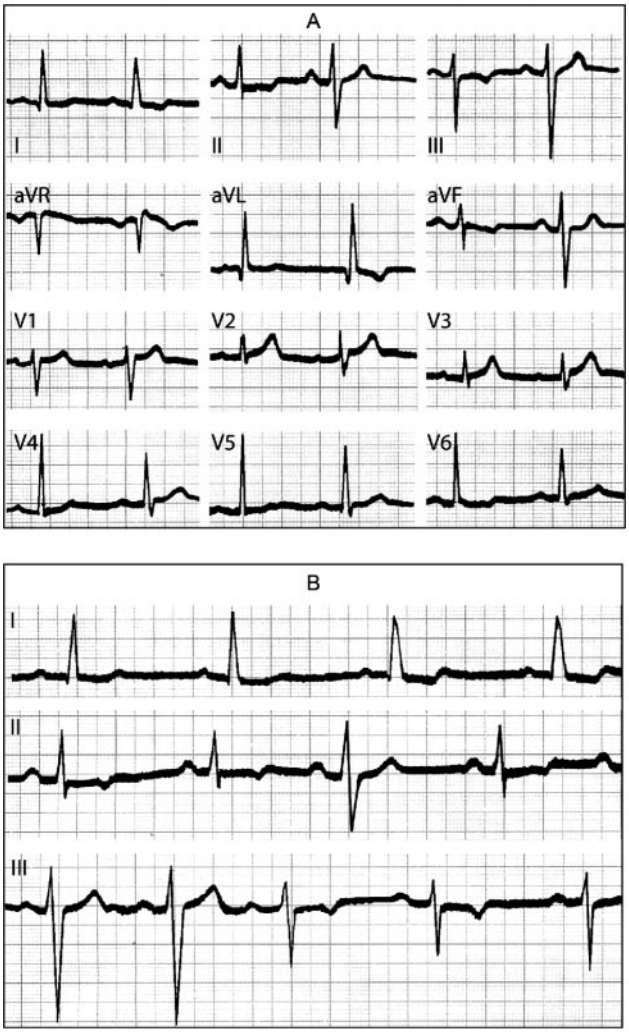

Figure 4. A, Intermittent LAH. In every lead the first beat shows normal conduction and the second beat LAH. B, Leads I, II, and III exhibit rate-dependent LAH. Note that LAH totally conceals the pattern of inferior ischemia in leads II and III, as well as the ventricular overload in the left precordial leads. LAH in aVL simulates a lateral infarction. See text for full description.

The Problem of Complete Versus Incomplete LAH

Different degrees of the conduction disturbance can be observed and recognized in cases of intermittent or transient LAH or during intraventricular aberrant conduction of premature supraventricular beats. When starting from a normal AQRS direction (for example +60°), progressively increasing degrees of LAH will shift the AQRS to +50°, +40°, +30°, +20°, etc, and cover the whole range to −60°. Consequently, in a given case that shows progressive left axis deviation of the QRS, it is easy to know when LAH is complete or incomplete and we may even diagnose incomplete LAH with a normal AQRS direction.

Diagnostic Pitfalls

It is difficult to draw a perfect dividing line between other causes of left axis deviation and left axis deviation caused by LAH, and so some overlap is unavoidable. A differential diagnosis must be considered with: (1) horizontal heart; (2) isolated left ventricular hypertrophy; (3) regional anterosupe-

rior right ventricular block; (4) straight back syndrome; (5) Wolff-Parkinson-White syndrome; (6) other causes of pitfalls in the diagnosis of LAH , such as hypertrophic subaortic stenosis, inferolateral myocardial infarction, emphysema, chest deformities, single ventricle, corrected transposition, or Ebstein disease. As a general rule, whenever the S wave in lead II is deeper than in lead III, the diagnosis of LAH is quite unlikely; the VCG with characteristic counterclockwise rotation of the QRS loop in the frontal plane can always help the diagnosis.[1,2]

LAH and Myocardial Infarction

The diagnosis of myocardial infarction in the presence of bundle-branch block (particularly left bundle-branch block [LBBB]) has always been difficult; this also applies to the association of myocardial infarction with hemiblock. The anterior division of the LBB is supplied by the septal branches of the anterior descending artery and its most proximal segment by the artery to the atrioventricular (AV) node in some cases.[1,2,10,11] Because the RBB shares the same pattern of blood supply, RBBB, LAH, or both together are likely to occur in anterior or anteroseptal infarction, an association that is exceedingly frequent during or around its occurrence.[1,2] The incidence of LAH in anterior and anteroseptal infarction has been estimated to be ≈7% to 15%,[12–16] and it may also take place transiently during severe anteroseptal myocardial injury.[1,2] Although hemiblocks may either simulate or hamper myocardial infarction, they do not hinder the diagnosis of an extensive necrosis regardless of its localization. A more localized necrosis can be concealed by the hemiblocks, however.

Inferior Wall Myocardial Infarction Concealed by LAH

The typical QR or QS morphology in an inferior myocardial infarction in the inferior limb leads may be hampered by LAH. In a typical LAH, an R wave in leads II, III, and aVF is always observed because of early left ventricle activation of the inferior wall of the left ventricle supplied by the posterior division of the LBB. This R wave will totally or partially disappear whenever the necrotic zone comprises the areas of early activation. Conversely, if the infarction spares those areas, an initial R wave will occur in the inferior leads and the inferior infarction may be concealed (Figure 5, top). Moreover, the repolarization of secondary changes of LAH that produce a positive T wave in the inferior limb leads (Figure 4) also contribute to mask the signs of a diaphragmatic myocardial infarction. Accordingly, a negative T wave in II, III, and aVF in the presence of LAH is a strong sign of the coexistence of a significant inferior ischemia or in some cases a concealed inferior infarction as well. If this is the case, the VCG provides the typical pattern of inferior infarction in the presence of LAH.[17–19] The most characteristic finding of this association is found in the frontal plane of the VCG, where the initial 25-millisecond vector, instead of being oriented inferiorly and to the right, is inscribed to the right, usually with clockwise rotation. The remaining QRS loop rotates counterclockwise as expected in LAH (Figure 5). In some cases, like the case shown in Figure 6, a careful look at

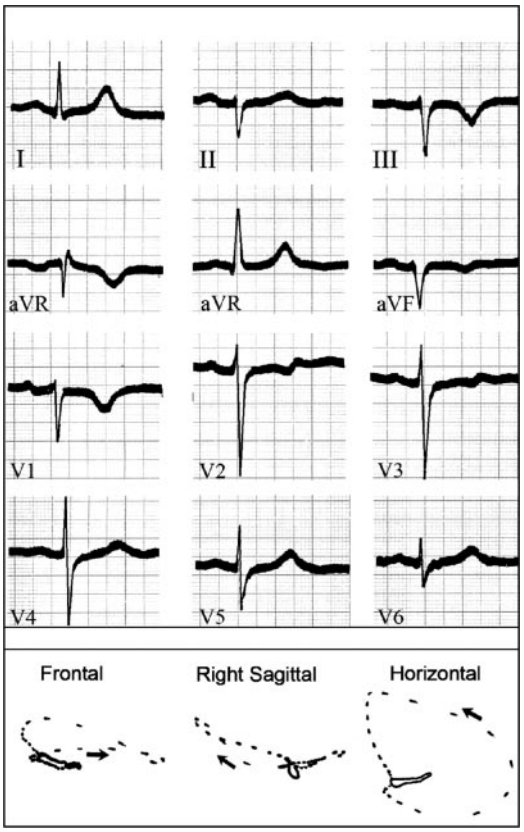

Figure 5. LAH and inferior myocardial infarction from a 50-year-old man. Top, The QRS does not show signs of myocardial infarction. However, the T wave is abnormal in leads III, aVF, and V1 to V3, which suggests myocardial ischemia. Bottom, The initial forces of the VCG in the frontal plane are oriented inferiorly and to the right with clockwise rotation. The remaining segments of the QRS loop show abnormal superior displacement as seen in inferior wall myocardial infarction that shows counterclockwise rotation, as expected in LAH.

the small R wave in the inferior limb leads may reveal minimal changes such as lower voltage in II, III, and aVF, RII lower than RIII, slurred small R waves in the same leads, or a tiny Q wave followed by a small R wave in LII, all of which suggest fibrosis or necrosis in the inferior wall. If the septum is preserved in an inferior myocardial infarction, the initial 25- to 30-millisecond vector of the frontal plane will be oriented first to the right and then superiorly and to the left with a typical inferior concavity; the final activation process shows counterclockwise rotation, and the maximum QRS deflection vector is oriented superiorly and to the left. When an anteroseptal and inferior infarction are present together, the initial 25- to 30-millisecond vector is initially oriented superiorly and then to the left with counterclockwise rotation.

Anterior Myocardial Infarction Concealed by LAH

If the chest leads are recorded below the normal level in cases of anterior infarction accompanied by LAH, a small R wave is recorded, which may conceal the Q waves. Likewise, LAH my also conceal an anterior myocardial infarction when the position of the heart is horizontal in a subject with a stocky build.[1,2]

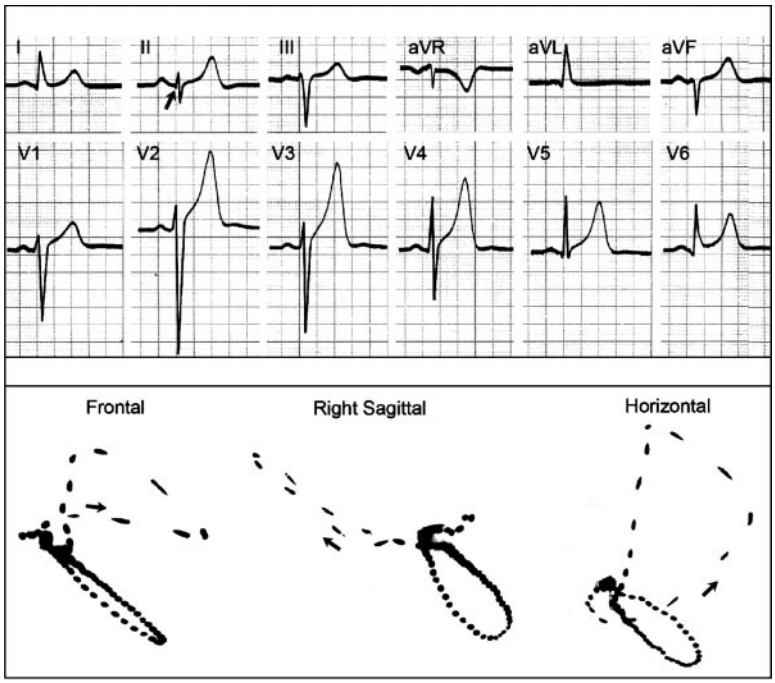

Figure 6. Top, LAH and inferior wall myocardial infarction in a 65-year-old man. Bottom, The frontal plane of the VCG confirms the diagnosis of inferior infarction associated with LAH.

LAH Simulated by Extensive Inferolateral Necrosis

It has been stated that counterclockwise rotation of the QRS loop in the frontal plane is the most reliable sign that differentiates LAH from other conditions that may produce marked left axis deviation, which thus makes the VCG the main diagnostic tool for the recognition of this conduction disturbance. Figure 7 shows the ECG and VCG of a patient with inferior and anterolateral infarction. The AQRS is shifted superiorly and to the left to −60°, and except for the absence of initial R waves in II, III, and aVF (caused by an extensive inferior necrosis) and a Qr in lead II, it looks like a classic LAH. However, in the frontal plane the initial QRS forces are directed superiorly (inferior necrosis) and to the right (lateral necrosis) followed by a wide-open clockwise loop that shows a superior and leftward displacement of the main, final QRS forces. It has to be emphasized that the presence of Qr in lead II instead of QS implies that the last segment of the depolarization loop rotates clockwise, which falls in the positive hemifield of lead II. Thus, from the electrocardiographic point of view, a QS pattern in lead II allows the suspicion of LAH but not when a Qr complex is present. Besides the pattern of inferior necrosis in leads II, III, and aVF, abnormal Q waves are also present from V3 to V6.

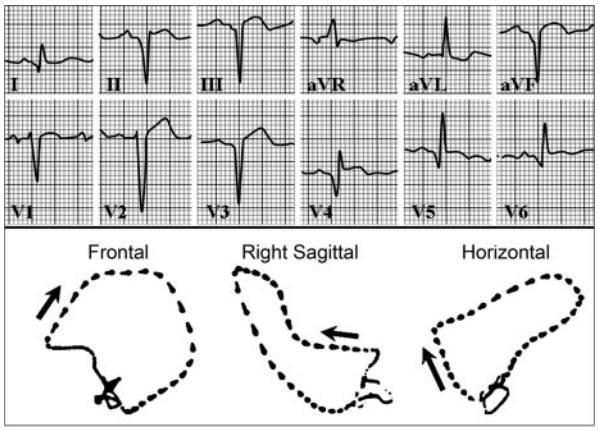

Figure 7. Anterolateral and inferior wall myocardial infarction that simulates LAH. Top, The ECG shows a QRS interval of 110 milliseconds. Bottom, Clockwise rotation of the QRS loop in the frontal plane rules out the presence of LAH.

LAH May Simulate a Myocardial Infarction

It has been mentioned that the shift of the initial forces inferiorly and to the right may produce a small Q wave in V2 and V3, which can fake the signs of an previous anterior or anteroseptal infarction (Figure 3B). In light of the frequent association of LAH and anteroseptal infarction, this finding may easily lead to erroneous interpretations. These small Q waves may be present if these leads are recorded above the conventional level and, even when correctly placed, in patients with vertical hearts, slender body type, or emphysema. If the Q waves are still recorded in the right chest leads at 1 intercostal space below the normal level, a real anteroseptal infarction is much more likely.[1,2] When LAH produces conspicuous Q waves in leads I and aVL, it may also imitate a lateral myocardial infarction, particularly if at the same time a T wave inversion, caused by secondary changes, is elicited (Figure 4A).[1,2]

LAH Obscures the Diagnosis of RBBB

The fact that LAH may partially conceal the diagnosis of RBBB by causing the S waves of RBBB to disappear from leads I, aVL, and in some cases from the left precordial leads has been shown and widely discussed under the name of *standard* and *precordial* masquerading RBBB.[1,2,20] When the S wave disappears in leads I and V5–V6, the configuration of RBBB becomes so atypical that it may be considered LBBB.

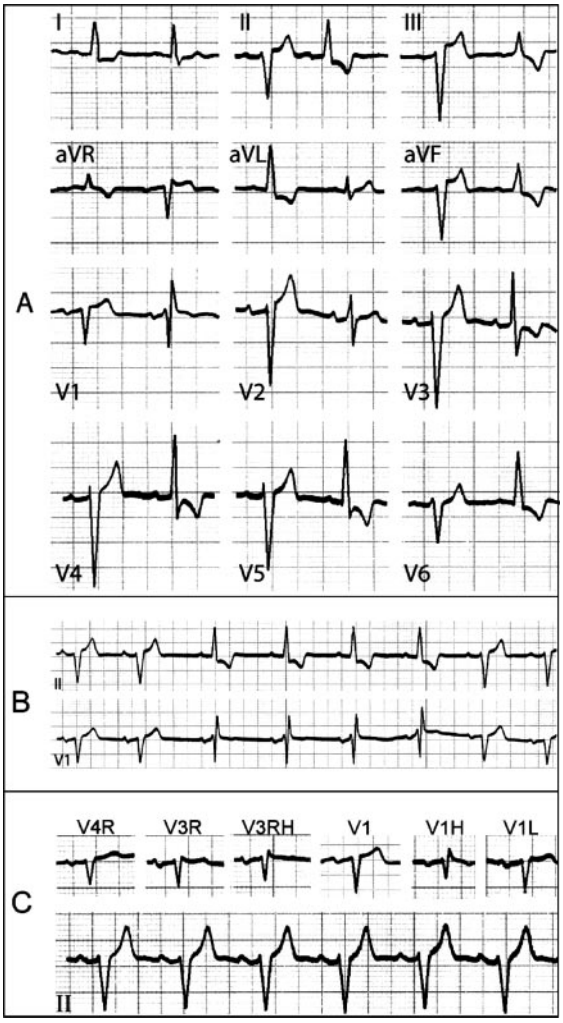

Figure 8. Permanent RBBB with intermittent LAH. The LAH conceals the signs of RBBB. A, In every lead, the first beat shows LAH (plus RBBB), and the second beat shows RBBB alone. B, Simultaneous recording of leads II and V1. LAH is seen only in the first 2 beats and the last 2 beats. When LAH is absent, a typical RBBB pattern is uncovered. C, The precordial chest leads recorded at the time when LAH was present (as seen in lead II) show the pattern of RBBB when V3R and V1 were recorded 1 intercostal space above the normal level (V3RH and V1H).

The main conditions for this pattern are: (1) high degree of LAH superimposed with RBBB and (2) left ventricular hypertrophy and/or left ventricular focal block caused by myocardial fibrosis or necrosis.[1,2,20] Together, these conditions neutralize the main forces of RBBB and thus diminish or eliminate the S wave of lead I (standard) and/or that of V5–V6 (precordial). Interestingly, LAH may eventually obscure the typical QRS changes of RBBB even in the right precordial leads in such a way that the electrocardiographic diagnosis may be mistaken and the RBBB totally missed,[20] which implies important diagnostic and prognostic connotations, when the fact that exclusion of the RBBB supposes a great clinical hazard is considered. Figure 8A through 8C shows the ECGs of a typical example in which LAH is intermittent and, when present, conceals the pattern of RBBB in the conventional right precordial leads. In Figure 8A, the first beat of every lead shows LAH plus RBBB whereas the second beat only exhibits RBBB. Note that LAH not only obscures the diagnosis of RBBB but also conceals a severe left ventricular hypertrophy in the left precordial leads. Likewise, the secondary T-wave changes induced by LAH in leads II, III, aVF, and from V3 to V6 reverse the deep negative T waves. Figure 8B shows the simultaneous recording of leads II and V1; LAH is only seen in the first 2 beats and last 2 beats. When LAH is absent, a typical RBBB pattern is uncovered. The precordial mapping may unmask the pattern of RBBB as shown in Figure 8C. Of note, this patient developed paroxysmal AV block and Adams-Stokes seizures 1 year later and required a pacemaker implantation.

Clinical Significance and Epidemiology of LAH

As a left ventricular structure, the anterior division of the LBB may be injured by diseases that involve primarily the outflow tract, the anterior half of the ventricular septum, and the anterolateral wall. Other pathologies that may also provoke LAH are hypertension, cardiomyopathies, aortic valve disease, Lev and Lenègre diseases, spontaneous and surgical closure of a ventricular septal defect, and other surgical procedures.[1,2,21–30]

Lenègre disease may be suspected on clinical grounds in many cases of RBBB with LAH that eventually develop AV block.[1,2,31] In most of our observations LAH preceded RBBB and the additional involvement of the LBB or its posterior division led to the final occurrence of complete heart block. At present, Lenègre disease has been considered a genetic hereditary disorder and it should be clinically suspected in middle-aged or slightly older people.[32] Commonly, concomitant coronary artery disease or myocardial disease is absent, and the histological study only shows scleradegenerative lesions and fibrosis of the conduction system.[2,31]

Lev disease corresponds to a process of sclerosis of the left side of the cardiac skeleton, which may cause intraventricular conduction disturbances caused by involvement of the branching His bundle, usually at the level of the pseudobifurcation.[1,2,33] Lev disease is responsible for most cases of RBBB with LAH seen in elderly people without other signs of cardiac involvement who eventually develop complete heart block after many years.[1,2,33]

Spontaneous closure of ventricular septal defects is an ignored cause of LAH. It should be stressed that the spontaneous closure of membranous or perimembranous septal defects is an overlooked cause of LAH that may account for a significant number of cases, particularly when found in young subjects without apparent structural heart disease. Among 14 documented cases of spontaneous closure of a septal defect and intraventricular conduction disturbances studied in our center (1 of them with postmortem histological study of the conduction system), LAH was found in 70% of the cases followed by RBBB.[26] Another study performed by Arcil et al[34] demonstrated that the spontaneous closure of a ventricular septal defect in newborns occurred in ≈50% of the cases in a 6-month follow-up and that 67% of the cases developed LAH.

Finally, small areas of myocardial fibrosis caused by subdued myocarditis may well be another cause of LAH as observed in 2 young patients (16 and 20 years old) studied in our center, who did not disclose any detectable cardiac abnormality after a typical episode of myocarditis, except for the conduction disturbance in the anterior division of the LBB.

Most studies on the incidence of LAH were performed from hospital subjects,[1,2,28,29] some were performed on general populations,[30,35] and very few were executed on selected populations of presumably healthy individuals who were previously screened and followed.[36,37]

Among 1658 consecutive patients treated in a cardiological service, LAH was found in 76 cases (4.58%); 53 cases showed RBBB (3.19%), and 17 cases showed LBBB (1.02%).[2] If we put together LAH and LBBB, it is clear that intraventricular conduction disturbances are more frequent in the left side than in the right one, which is congruent with the fact that left heart disease is far more common than right. The main clinical causes of LAH in a hospital population were arterial hypertension and coronary artery disease, which was unequivocally present in 41% of the patients,[2] and it is well known that LAH is commonly associated with anteroseptal or anterolateral infarction.[1,2,16,38–40]

In a retrospective study of the natural history, clinical significance, prognosis, and associated conduction disturbances, 247 cases of LAH were detected among a presumable healthy population of 8915 individuals engaged in civilian flying activities in Argentina (prevalence, 2.77%).[36] The age of the studied population ranged between 17 and 79 years. Of these 247 cases of LAH, 52 cases (21%) were detected in the first ECG, and 195 cases (79%) were detected after either a previous normal ECG or depiction of slight left axis deviation ($<-30°$). The ECGs and clinical data were retrospectively analyzed for a period that varied from 3 to 42 years (mean, 15.3 ± 10.2 years), and 115 men with LAH had records for >15 years. In 15 cases, LAH was associated with RBBB. There were 2 men who developed LBBB and posteriorly complete AV block. Of note, not a single case of LPH was found. Underlying heart disease was not detected in either of the men. Although LAH was observed at all ages from 17 to 70 years, it is striking that 152 of the 247 cases were found in patients who were 17 to 39 years old. Even more surprising was the low incidence of associated pathology: hypertension in 26 cases (10.5%) and coronary artery disease in 8 cases (3.2%), detected in subjects between 36 and 73 years (mean, 50.7 ± 9.1 years). This finding poses an important question about the etiopathogenesis of LAH that takes the low incidence of underlying manifest heart disease into consideration. In the Argentinian study,[36] all patients that presented with LAH underwent a careful medical examination, routine laboratory analysis that included serologic tests for Chagas disease (because it is endemic in Argentina), ECG stress test, M-mode, and 2-dimensional echocardiography. If the exercise testing was abnormal, thallium[201] myocardial perfusion studies and/or coronary arteriography were performed to rule out coronary artery disease. To be noted, these findings are in agreement with other epidemiological studies that also suggest that isolated or lone LAH may not have adverse prognostic implications.[28,41–43]

Substantial differences were observed with regard to the prevalence of LAH in other studies that involve selected populations in which the diagnostic criteria were well defined. Krivisky et al[37] found a prevalence of 1.03%, and in the 15 cases that showed LAH the average age was 24 ± 4 years. Rabkin et al[41] reported a prevalence of 6.2% in their population of 3933 airmen, which is almost triple the prevalence referred to in the Argentinian study, a fact that is hard to explain.[36] In summary, the prevalence of LAH in the normal population varies according to series from 0.9% to 6.2%. These differences may be attributed to nonidentical diagnostic criteria for LAH, ethnic diversity, or dissimilar incidence of associated pathologies. The prognosis of these patients is basically related to the characteristics of the associated pathology. It should be noted that the studies on LAH based on hospital cases or autopsy cases reflect a relationship between left ventricular disease secondary to coronary heart disease, hypertension, or cardiomyopathy.

Some authors have proposed that the prognosis of isolated LAH in acute myocardial infarction is not worse than in those patients without this conduction disturbance.[38–40] However, another study reported a slightly higher death rate among those patients with isolated LAH and myocardial infarction.[16] Likewise, in a recent study by Biagini et al[44] of 1187 patients with suspected coronary artery disease referred for stress testing, LAH, found in 159 patients, was associated with increased risk of cardiac death in a 6-year follow-up ($P = 0.004$).

In conclusion, it is our belief that isolated LAH does not imply by itself a risk factor of cardiac morbidity or mortality, and in a healthy population it must be regarded as an incidental electrocardiographic finding, which is not the case in hospital populations.

Left Posterior Hemiblock

On the basis of the intrinsic anatomic characteristics of the intraventricular conducting system, it is reasonable to believe that conduction block is more likely to occur in a thin and longer than in a short and thicker fascicle. Accordingly, we have proposed a theoretical order of anatomic and physiological vulnerability, with the RBB as the most vulnerable followed by the anterior division of the LBB, the posterior division of the bundle branch, and, finally, the main LBB. However, the clinical presentation of conduction disturbances in order of decreasing incidence is LAH, RBBB, LBBB, and last, LPH.[1,2] This rank depends not only on the intrinsic, anatomic, genetically determined differences among branches and fascicles but also on the manner in which the intraventricular conduction system is exposed to the various pathological processes of the surrounding cardiac structures. In fact, the posterior division is the least vulnerable segment of the whole system because it is short and wide, it is located in the inflow tract of the left ventricle, which is a less turbulent region than the outflow tract, it has double blood supply (from the anterior and posterior descending coronary arteries), and it is not related to structures that are so potentially dangerous.

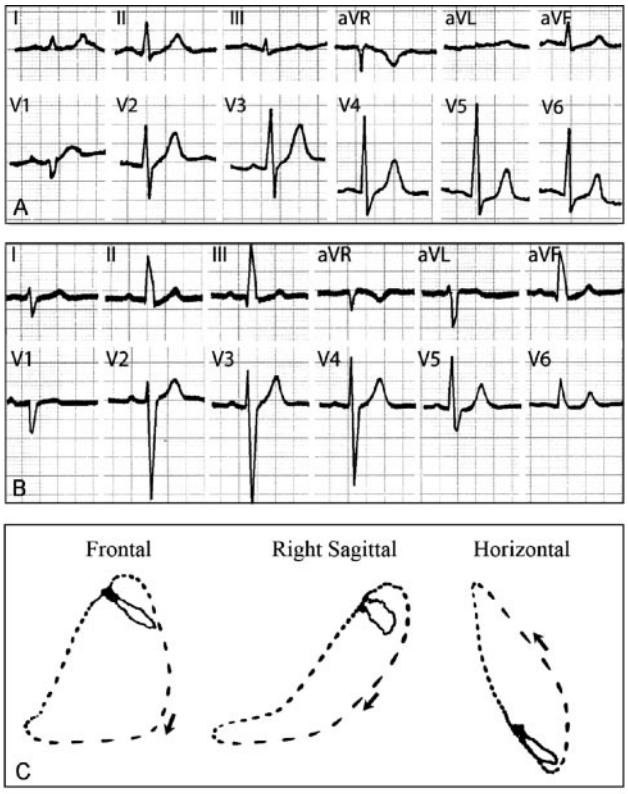

Figure 9. Development of pure LPH in a 62-year-old man without apparent heart disease (Lenègre disease?). A, Normal ECG. The AQRS is ≈+50°. B and C, ECG and VCG were recorded 1 year later. The AQRS has shifted to the right at ≈+110° with a tracing that is otherwise normal.

Electrocardiographic and Vectorcardiographic Diagnosis of LPH

The electrocardiographic recognition of LPH emerged from the study of patients with permanent RBBB and intermittent LAH and LPH, intermittent or progressive isolated LPH, aberrant conduction of premature supraventricular beats, and the experimental production of LPH in dogs and monkeys.[1,2]

The electrocardiographic criteria for pure uncomplicated LPH are (1) AQRS direction of ≥100°, commonly in the presence of severe left heart disease and in the absence of right ventricular hypertrophy, a vertical heart in slender subjects and a large lateral infarction; (2) S1Q3 pattern in the limb leads; (3) QRS duration <110 milliseconds; (4) rS morphology in leads I and aVL; and (5) qR pattern in leads II, III, and aVF (Figure 9A through 9C).[1,2]

Figure 9A through 9C illustrates a typical example of pure LPH. The patient had a previous ECG with an AQRS direction of ≈50° (Figure 9A). A year later, the AQRS shifted to +110°, and except for the change of the electrical axis, the ECG may be considered normal (Figure 9B). The VCG depicts the most typical changes of the QRS loop (Figure 9C). The initial forces oriented superiorly and to the left, responsible for the small R waves in leads I and aVL and the small Q waves of leads II, III, and aVF, are caused by early activation of the anterolateral wall of the left ventricle. In the frontal plane, the main and terminal forces of the QRS loop are oriented inferiorly and to the right (≈+100°) with a wide-open clockwise-rotated loop (Figure 9C). Actually, the ECG and VCG of LPH is the exact mirror picture of LAH in the standard and unipolar leads. Because isolated LPH is extremely rare, when present it is almost always associated with RBBB.

LPH Obscures the Diagnosis of Myocardial Infarction

When LPH occurs in the initial stages of an inferior myocardial infarction or in cases of transient injury of the inferior wall, the QRS forces after the initial milliseconds shift inferiorly and to the right because of arborization block or left posterior fascicular block that conceals the signs of an inferior myocardial infarction. Figure 10A and 10B illustrates an example of pure transient LPH that conceals the signs of an inferior myocardial infarction. During an anginal episode, the appearance of LPH veils the pattern of inferior myocardial infarction. Note the subtle ST-segment elevation in lead III and marked ST-segment depression in leads I and V5–V6 that denote the severe inferolateral myocardial injury.

Clinical Significance of LPH

Isolated LPH is extremely rare and is almost invariably associated with RBBB, in that LPH and RBBB share etiology, pathogenesis, and prognosis.[1,2] LPH plus RBBB in acute myocardial infarction is associated with a high mortality rate (80% to 87%) during the first weeks after the coronary event.[16,45] Likewise, the risk of progression toward complete AV block (a form of trifascicular block) is also considerable (42%), and ≈75% of these patients die from pump failure.[16]

Coronary artery disease is less common in RBBB with LPH than in RBBB with LAH or pure LAH, and it is apparent that myocardial disease and cases of unknown origin (probably Lenègre disease) occur more often. In Argentina, and very likely in other Latin American countries where Chagas disease is endemic, LPH with or without RBBB may be found in cases of chronic chagasic myocarditis.[1,2] Because the posterior division of the LBB is the least vulnerable segment of the intraventricular conduction system, the occurrence of LPH with RBBB is a forerunner of complete heart block. In fact, when the conduction system lesions are so extensive that they alter conduction in the posterior fascicle, it is nearly axiomatic that the RBB, the anterior division of the LBB, or both, are also involved. This is the reason for the high incidence of AV block in RBBB with LPH. A strong argument that supports this assumption is that, among 29 cases of RBBB with LPH, direct evidence of LAH was observed in 7 cases (intraventricular trifascicular block). Thus, RBBB with LPH, particularly when accompanied by AV conduction disturbances, constitute a variety of the trifascicular blocks.[1,2] AV conduction disturbances were observed in 24 of 29 cases of RBBB with LPH (82.7%). The PR interval was prolonged in 26 of the 29 patients. Eighteen of the patients exhibited complete or high-degree AV block during follow-up, and Adams-Stokes seizures were documented in 17 of the 29 cases (58.6%). To be noted, a study by Dhingra et al[46] in 21 patients with RBBB and LPH with a follow-up period of 671±68 days demonstrated that 6 pa-

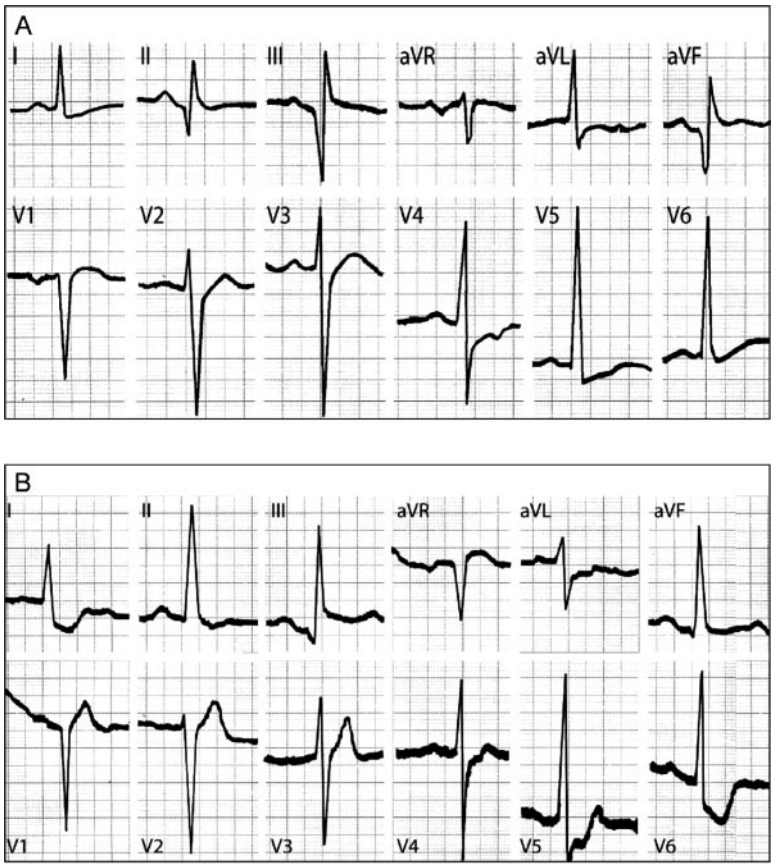

Figure 10. Transient LPH caused by subepicardial inferior wall injury greatly conceals the pattern of inferior wall myocardial infarction in a 62-year-old patient with unstable angina. A, Clear-cut signs of inferior infarction. B, During an episode of angina, the ECG shows a transient LPH, which almost completely conceals the signs of the inferior wall myocardial infarction.

tients had prolonged His Purkinje conduction time intervals, and only 1 of the 3 who needed a permanent pacemaker had definitely related trifascicular disease. Notwithstanding, because AV block becomes one of the most important features of the electrocardiographic picture of RBBB with LPH, in symptomatic patients the implantation of a permanent pacemaker must be carefully evaluated.

Conclusion

Although the existence of hemiblocks is widely accepted and its recognition well known by cardiologists all over the world, there still are some points related to its etiopathogenesis and clinical presentation that deserved to be reappraised. In the present review, we have presented evidence of some not so well known epidemiological, etiopathogenic, and diagnostic aspects of these conduction disturbances.

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Disclosures

None.

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