Electrovectorcardiographic Diagnosis of Left Septal Fascicular Block: Anatomic and Clinical Considerations

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Several publications considering anatomical, histological, pathological, electrocardiographic, vectorcardiographic, and electrophysiologic studies have shown that the left bundle branch splits into three fascicles or in a "fan-like interconnected network" in the vast majority of human hearts. The left His system is trifascicular with a left anterior, a left posterior, and a left septal fascicle (LSF). Consequently, the classic term "hemiblock," to describe the block of one of the fascicles, established several decades ago by the Rosembaum's school, should be updated.

Electrovectorcardiographic changes resulting from conduction abnormalities of the left anterior and left posterior fascicles are commonly diagnosed, mainly by their changes in the frontal plane. However, the existence of conduction defects of the LSF remains controversial. The ECG/VCG hallmark of LSF block is prominent anterior QRS forces (PAF) on the horizontal plane. This ECG/VCG phenomena should be distinguished from other conditions that also produce anterior QRS shift in the HP as: normal variants, right ventricular enlargement, misplaced precordial leads, lateral myocardial infarction, right bundle branch block, Wolff-Parkinson-White, obstructive and nonobstructive forms of hypertrophic cardiomyopahty, diastolic left ventricular enlargement, endomiocardial fibrosis, Duchenne muscular dystrophy, and dextroposition.

The two highly frequent etiologies of LSFB are ischemia (coronary artery disease (CAD) with critical proximal obstruction of the left anterior descending coronary artery) and, in Latin America, Chagas' cardiomyopathy.

The aims of this review are to revise the evidence of the existence of a trifascicular left Hissian system and to help in the ECG/VCG recognition of the LSFB.

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heart conduction system; fascicular block; bundle of his; electrocardiography

INTRODUCTION

The three left fascicles of the left bundle branch (LBB) along with the right bundle branch (RBB), constitute the quadrifascicular structure of the intraventricular conduction system of the heart, coined by Dr. Uhley.[1,2]

Anatomical Considerations of the Left Intraventricular Conduction System

Anatomopathological studies showed that the left septal fascicle (LSF) has diverse morphologies and considerable variability in its structure. Thereby, six basic anatomical variations can be described[3–7]:

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- **Type I:** the LSF arises independently from the main LBB (65% of the cases).
- **Type II:** the LSF originates from the left anterior fascicle (LAF) of the LBB.
- **Type III:** the LSF originates from the left posterior fascicle (LPF). This type represents about 2.4% of all cases.
- **Type IV:** the LSF originates concomitantly with the other two fascicles (LAF and LPF).
- **Type V:** the LSF is represented as a "fan-like interconnecting network."
- **Type VI:** the LSF is absent; consequently, the left Hissian intraventricular system has only two fascicles: LAF and LPF. It occurs in approximately 15% to 40% of the cases.[3]

a. Blood Supply (modified from reference 8)

The blood supply of the human His bundle and its proximal branches have a dual origin, with anastomoses within the His bundle. The conduction system is supplied as follows:

(1) His bundle: it has a dual supply by the AV node artery from the right coronary artery (RCA) and the first septal branch of the left anterior descending artery (LAD) in 90% of the cases, and entirely supplied by the AV node artery in 10% of the cases.
(2) Proximal right bundle branch: it is supplied by both the AV node artery and the septal branch in 50% of the cases, and only by the septal branch in 40% of the cases. The AV node artery as a single supply occurs in about 10% of the cases.
(3) Left bundle branch: it is supplied by the AV node artery (ramus septi fibrosi) from the RCA (in 90% of the cases) and ramus septi ventriculorum superior and ramus critae, branches of the LAD.
(4) Left branch fascicles or divisions branches supply: see Table 1

The LSF is supplied exclusively by the septal perforating branches of the LAD.[9] Critical lesions of the LAD before the first septal perforating branch are the main cause of LSFB in developed countries, and it is a major determinant of predominant anterior forces (PAF) from V_1 to V_3 during acute myocardial ischemia.[10] In Brazil, where Chagas' disease is very common, coronary artery disease represents only 18% of all LSFB.

Table 1. Blood Supply of the Left Branch Fascicles or Divisions

Responsible system	LAF (%)	LPF (%)	LSF (%)
LAD branches	40	10	100
Double irrigation (LAD & RCA)	50	40	0
RCA branches	10	50	0

LAD = left anterior descending artery; RCA = right coronary artery.

LSFB can be also related to exercise-induced ischemia,[11] sometimes leading to *giant* R waves in the precordial leads.[12–15]

Sudden development of LSFB in critical LAD lesions indicates a proximal location of the lesion, and therefore, a worse prognosis.

b. Electrophysiology of the Left Intraventricular Conduction System

The electrophysiologic demonstration of the activation of the middle third of the left septal surface 5 ms before the anterosuperior and posteroinferior regions was made in 1970 by Durren et al.[16] They demonstrated in 870 intramural terminals of isolated human hearts, that three endocardial areas were synchronously excited from 0 to 5 ms after the initiation of the left ventricle (LV) activity potential. To demonstrate the time course and instantaneous distribution of the excitatory process of the normal human heart, the authors studied isolated human hearts from seven individuals who died from neurologic disease with no history of cardiac disease. The first LV areas excited were located in the following regions:

(1) High on the anterior paraseptal wall just below the insertion of the anterolateral papillary muscle (ALPM) where the LAF ends;
(2) Central on the left surface of the intraventricular septum (IVS), where the LSF ends. Septal activation started in the middle third of the left side of the IVS, in the anterior and lower third at the junction of the IVS and posterior wall.
(3) The LSF, the left middle septum surface, and the inferior two-thirds of the septum originate the first vector[17]

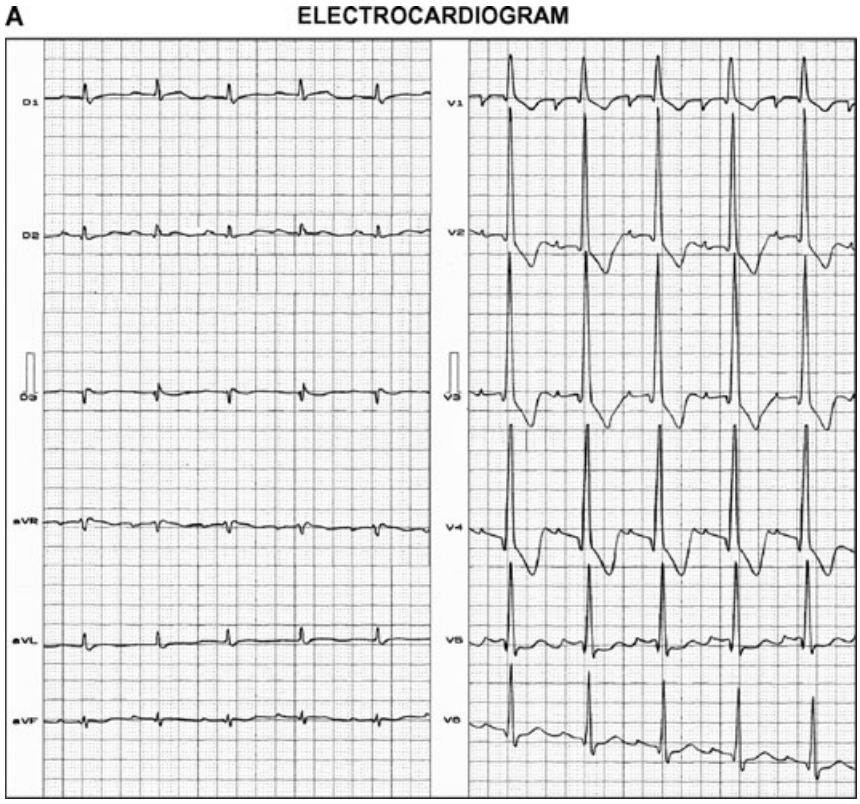

Figure 1. (A) This ECG belongs to a 75-year-old male with severe congestive heart failure and severe CAD. LAD: 100%; left circumflex: 100%, RCA: 90% obstructed. It depicts first degree AV block + LSFB + Anterior MI. Low QRS voltage only in the FP leads.

(4) Posterior paraseptal, about one third of the distance from the apex to the base, near the insertion of the posteromedial papillary muscle (PMPM), where the LPF ends. The posterobasal area is the last part of the LV to activate.

Rosenbaum et al.[18,19] postulated that the activation of the middle-septal region occurs in most cases, from the anterior "false tendons" that originate from the LPF. His group considered that the LPF in its final portion opens as a fan, and the anterior "pseudo-tendons" are those responsible for the activation of the middle-septal region. We think that in fact, only one of the anatomical variations of the LSF (type III), is precisely the one that originates on the LPF (2.4% of cases).

Another consideration should be done to the electrophysiologic explanation of the so called "atypical LBBB." There are cases of divisional or fascicular LBBB (LAFB + LPFB) that depict a Q wave in the left leads, turning the electrocardiographic pattern of LBBB "atypical." Alboni et al[17] called them "LBBB with normal septal activation."

Rosenbaum et al.[19] called the same phenomenon "left intraventricular blocks without changes in the initial portion of the QRS." His group did not provide an explanation for these cases, and stated in their traditional book, that they were "difficult to explain."

Medrano et al.[20] proposed that in these atypical LBBB cases, the fibers of the LSF would originate prior to the site or area of the block in the LPF and LAF, so the middle-septal activation is preserved (vector 1 or anteromedial or septal vector, representing the initial 10 ms of the left surface), originating the Q waves of leads V_5-V_6, and turning the LBBB into an atypical one.

Gambeta and Childers[21] also proposed the trifascicular nature of the left Hissian system. The authors observed the development of transient abnormal Q waves during exercise as the heart rate increased. This phenomenon was attributed to

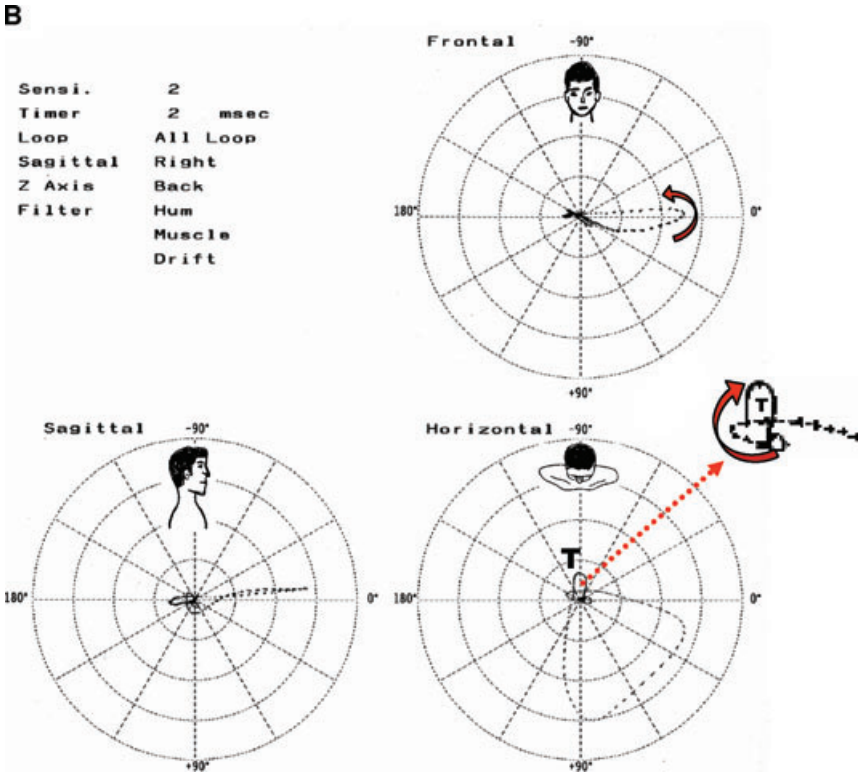

Figure 1. (B) Vectorcardiogram of LSFB. Frontal Plane: QRS loop with counterclockwise (CWW) rotation directed to left. Horizontal Plane: QRS loop with initial forces directed to back, the remained of QRS loop dislocated to the front and leftward (QRS loop predominantly located on left anterior quadrant) and CCW rotation: LSFB. Right Sagittal Plane: QRS loop directed to front: PAF.

transient tachycardia-dependent ischemic block in the LSF. The initial activation is conducted by the non blocked divisions (LAF and LPF). Since these fascicles have opposite directions and LPF activation predominates over LAF, produces the appearance of initial Q wave in intermediate precordial leads.

The same phenomenon was observed during the acute phase of myocardial infarction with the same electrophysiological explanation.[22,23]

Another strong argument for the existence of LSFB, was provided by several electrophysiological animal and human models. They showed the development of PAF (anterior shift of QRS loop) as a manifestation of intraventricular block, a consequence of intermittent intraventricular dromotropic disorder in the LSF region, during atrial extrastimuli.[24–30]

A delay in the LSF may explain certain type of ventricular aberration pattern observed by introducing premature atrial beats. This pattern consists of PAF with no or minimal QRS duration prolongation (≤20 ms) and without incomplete RBBB. Such delay is manifested as a narrow QRS with anterior shift in the HP, but no axis shift in the FP. It is important to recognize this aberration, because it may mimic the ECG findings of lateral MI (true posterior MI of the "old nomenclature") or RVH.[31]

In another study, aberrant ventricular conduction was induced in 44 subjects by introduction of atrial premature beats. The distribution of the patterns were RBBB (28); LAFB combined with RBBB (21); LAFB (17); LPFB combined with RBBB (12); LPFB (10); complete LBBB (10) and incomplete LBBB (6).

Other configurations could not be classified into the usual categories of intraventricular blocks. In 7 of them, the alterations only consisted on trivial modifications of the QRS contour. In the other 5, aberrant conduction manifested itself by conspicuous PAF of the QRS loop on the HP. The latter observation is worthy of notice, as it indicates that, in

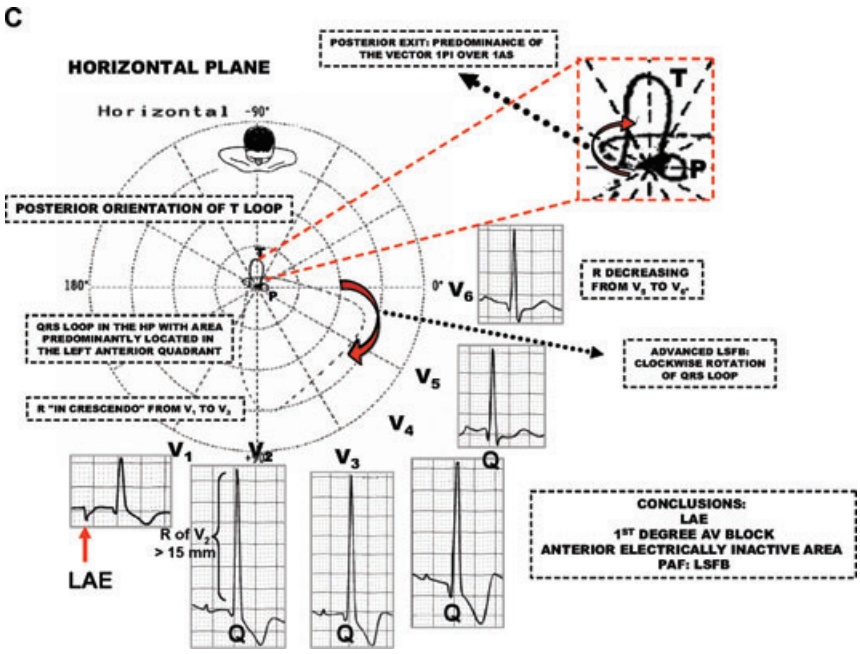

Figure 1. (C) ECG/VCG correlation in the horizontal plane. ECG/VCG diagnosis. Deep negative component of the P wave in lead V_1: Left atrial enlargement (LAE). First degree AV block. Initial Q wave in the anterior leads: anterior MI. PAF: V_2 R wave voltage >15 mm, R waves "in crescendo" from leads V_1 to V_3 and decreasing from leads V_4 to V_6: LSFB. Initial 10 to 20 ms vectors directed posteriorly. CW rotation of the QRS loop. QRS loop dislocated to front and leftward quadrant.

the differential diagnosis of the VCG pattern characterized by PAF, conduction disturbances should be considered a possible etiological factor in addition to RVH, and true posterior MI.[28–30]

Related the historical controversy about the bifascicular or trifascicular nature of the human left His system, we conclude that in most cases, the left His system is trifascicular. Consequently, the term "hemiblock," to refer to the block of the fascicles, should be avoided.[32–34]

NOMENCLATURE USED IN LITERATURE

There is a large variety of nomenclatures to name the LSF. This indicates the need of a consensus to unify terminology. This discussion should take place in an International or Worldwide Conference on Electrocardiology. The authors of this review strongly advocate for such a consensus.

In Brazil, a committee of experts in Resting Electrocardiology met in 2003 and developed the "Brazilian Guidelines for Interpretation of Resting Electrocardiogram." In this consensus, the diagnostic criteria of LSFB were determined and published.[35] However, it is important to remember different terminologies used in the literature: (1) left septal fascicular block (LSFB).[36–38,13,39–42] This is the currently accepted terminology and frequently used in more recent publications; (2) septal fascicle of the left bundle branch[43]; (3) focal septal block[22]; septal focal block[21]; (4) left parietal septal block[44,45]; (5) septal fascicular conduction disorders of the left branch[46]; (6) left septal Purkinje network block[47,48]; (7) left anterior septal block[49]; (8) anterior fascicular block[50]; (9) left septal subdivision block of the left bundle branch[31,51,52]; (10) left median hemiblock[53;54]; (11) middle subdivision block of the left bundle branch[55]; (12) middle fascicle block[17]; (13) block of the antero-medial division of the left bundle branch of His[15]; (14) anteromedial divisional block (AMDB)[12]; (15) block of the anterior median branch of the bundle of His; (16) blocking of the anterior-medial Ramulus[56]; (17) anterior conduction delay[29,31,52] and (18) intraventricular aberrant conduction.[26,57]

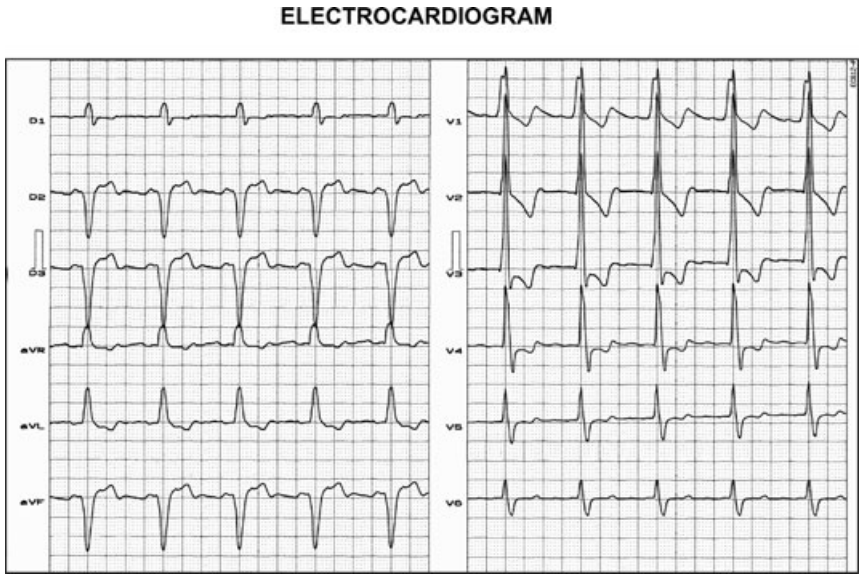

Figure 2. (A) This ECG depicts extreme left QRS axis deviation (-85°), SIII > SII, final S wave in left leads V_5 and V_6; all ECG features compatible with LAFB. PAF, R wave voltage "in crescendo" from leads V_1 to V_3 and decreasing from leads V_4 to V_6, small initial Q wave in leads V_1 toV_3, absence of initial Q wave in leads V_5 and V_6; all ECG features compatible with LSFB. The diagnosis is Left bifascicular block: LAFB + LSFB.

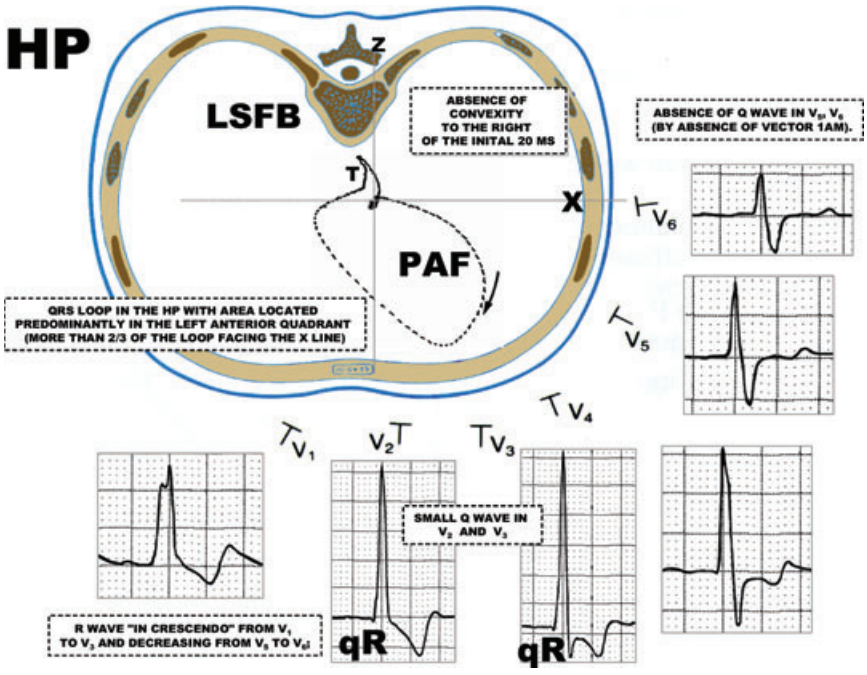

Figure 2. (B) ECG/VCG correlation in the horizontal plane. ECG/VCG diagnosis: ECG: R wave voltage "in crescendo" from leads V_1 to V_3 and decreasing from leads V_4 to V_6, small initial Q wave from leads V_1 to V_3, absence of initial Q wave in leads V_5 and V_6; all ECG features compatible with LSFB. VCG: QRS loop with initial QRS 10ms vector directed posteriorly and leftward, CW rotation and localized predominantly on left anterior quadrant: PAF. T loop directed to back.

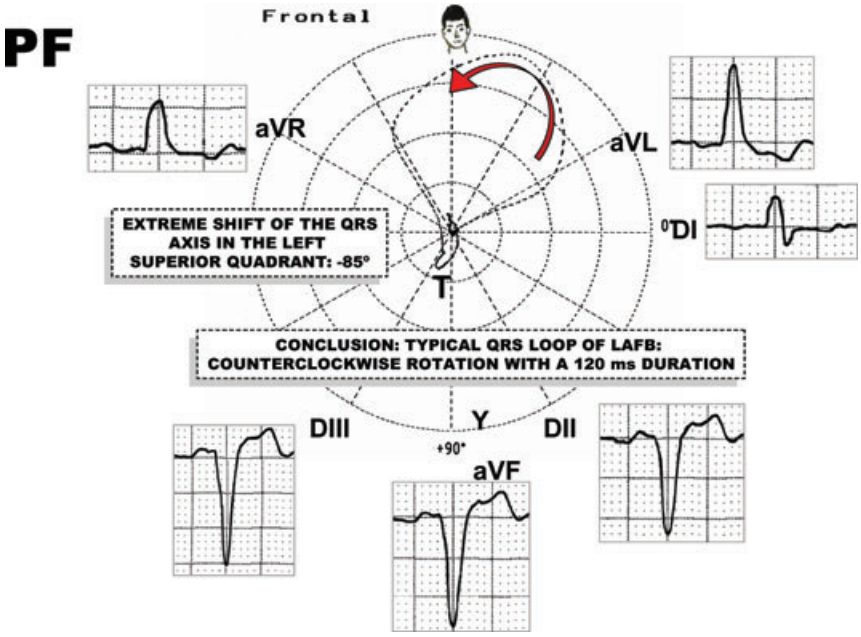

Figure 2. (C) ECG/VCG correlation in the frontal plane. ECG: Extreme QRS left axis deviation (-85°) SIII > SII; all features compatible with LAFB. VCG: QRS loop with CCW rotation and localized predominantly on left superior quadrant: LAFB.

We conclude that the various denominations only reflect the disparity of opinions on the existence of an anatomic structure defined as LSF. There are still some reasonable doubts on its electrophysiological properties and the effect of its disturbances (delay, block) on the surface ECG.

This group of authors does not consider themselves the "owners of the truth," and advocate for an international consensus to find a single terminology, based on a common interpretation of the literature. This will help to recognize this frequently misdiagnosed disorder and will help researchers to organize and focus their opinions on this issue.

LEFT SEPTAL FASCICULAR BLOCK: POSSIBLE ETIOLOGIES

The following list identifies possible causes of LSFB recognized in the literature:

(1) Ischemia: Coronary Artery Disease (CAD) with critical lesion of proximal LAD and/or its septal branches before the first septal perforating branch.[11]
(2) Chronic Chagas' Cardiomyopathy: main cause of LSFB in Latin America.[49]
(3) Non-Obstructive Hypertrophic Cardiomyopathy (NO-HCM).[58]
(4) Hypertrophic Obstructive Cardiomyopathy (HOCM).[59,60]
(5) Diabetes Mellitus.[46]
(6) Kearns-Sayre syndrome.[61]

LEFT SEPTAL FASCICULAR BLOCK: ELECTROCARDIOGRAPHIC CRITERIA[36,38,40–42,62,63]

(1) Normal QRS duration or minimal widening (up to 110 ms). If LSFB is associated with other fascicular or bundle blocks, the QRS could be wider than 120 ms.
(2) Frontal plane leads with normal QRS duration and amplitude.
(3) Increased ventricular activation time or intrinsic deflection in leads V_1 and $V_2 \geq 35$ ms.
(4) R wave voltage of lead $V_1 \geq$ than 5 mm.
(5) R/S ratio in lead $V_1 > 2$.
(6) R/S ratio in lead $V_2 > 2$.
(7) S wave depth in lead $V_1 < 5$ mm.
(8) Small Q wave in leads V_2, V_3 or V_1 and V_2.
(9) R wave of lead $V_2 > 15$ mm.

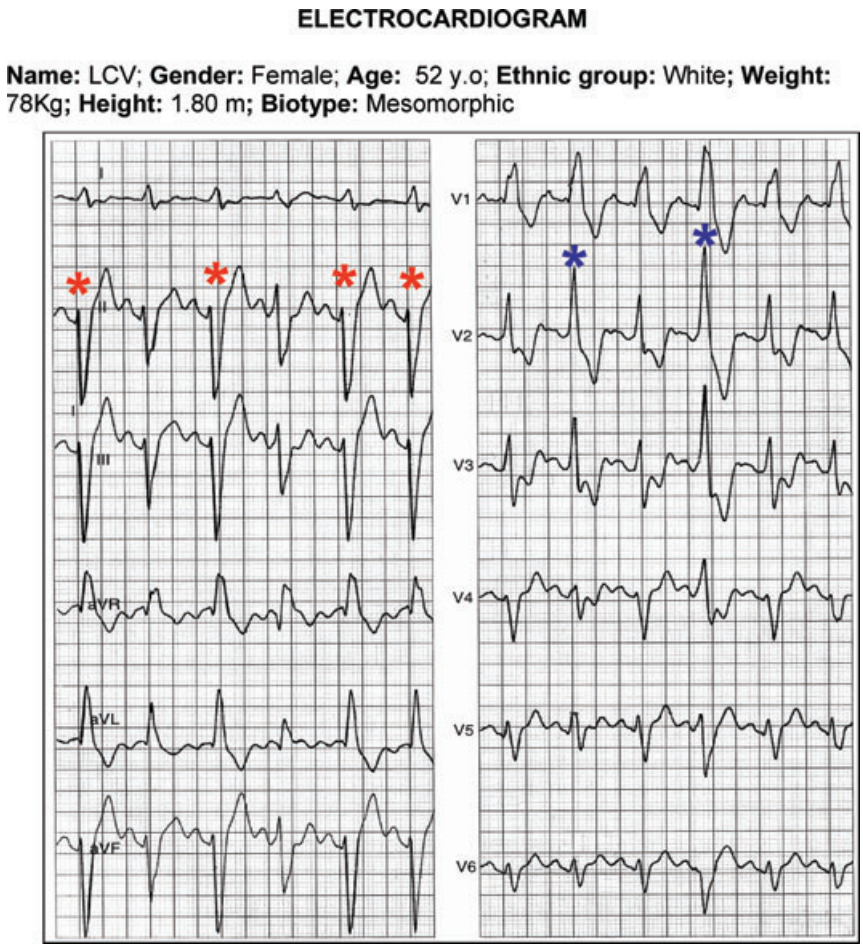

Figure 3. This is a case of a 59-year-old female with Chagas' cardiomyopathy; depressed left ventricular ejection Fraction (35%), LV end diastolic diameter of 74 mm. The 12-lead ECG shows complete RBBB, LAFB, and LSFB. FP: first, third, fifth and sixth beats (red asterisk) show higher degree of LAFB. HP: second and fourth beats (blue asterisk); LSFB is associated with RBBB. Conclusions: (1) Variable degree LAFB; (2) RBBB; (3) Intermittent LSFB; (4) Intermittent trifascicular block.

(10) RS or Rs pattern in leads V_2 and V_3 (frequent rS in V_1) with R wave "in crescendo" from V_1 through V_3 and decreasing from V_5 to V_6.

(11) Absence of Q wave in left precordial leads V_5, V_6 and I (by absence of vector 1 or anteromedial or septal vector, representing the initial 10 ms of the left surface).

(12) Intermittent PAF during hyperacute phase of myocardial infarction,[63] exercise stress testing in patients with severe myocardial ischemia[12,11] and during early atrial extrastimuli.[29]

(13) Intermittent rate-dependent Q wave in leads V_1 and V_2.[21]

The authors have provided a classification of the above ECG diagnostic criteria in Major and Minor criteria according to their relevance in:

I) Major Criterion

1) Intermittent PAF: intermittent or transient increment in the R wave voltage in intermediary precordial leads and intermittent or transient anterior displacement of the QRS loop on HP.

II) Minor Criteria

All the other criteria mentioned above.

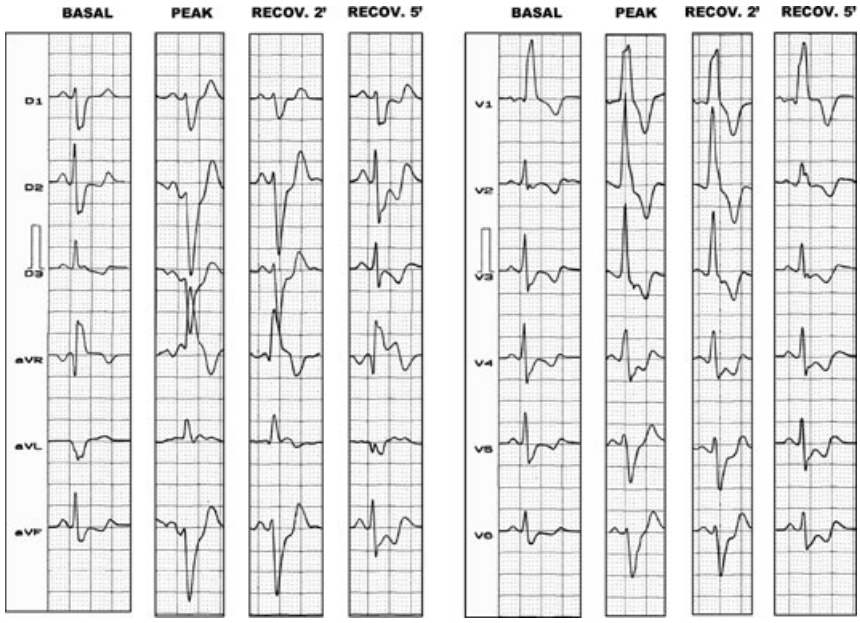

Figure 4. Exercise-induced LSFB. Transient ischemic bifascicular block. This case (stress test) belongs to a 60-year-old man, with prior MI for evaluation of stable chronic angina. Exercise induced transient LAFB, LSFB, and complete RBBB.

The diagnosis of LSFB could be done with 1 major criterion or with 2 minor criteria.

The Brazilian Guidelines for Interpretation of Resting Electrocardiogram[35] defined the following ECG criteria for diagnosis of LSFB:

(1) QRS duration <120 ms, (closer to 100 ms). The development of LSFB does not increase QRS duration by more than 25 ms, due to multiple interconnections between the fascicles of the LBB ("passage way zone" as defined by Rosenbaum). The QRS complex is slightly prolonged (between 100 ms and 115 ms). Thus, LSFB pattern with a prolonged QRS duration indicates the presence of additional conduction disturbances such as other fascicular blocks, RBBB, MI, focal block, or a combination of any of them;
(2) R wave in leads V_1 or V_2 and V_3 of 15 mm;
(3) Increased R wave voltage in all intermediary precordial leads and decreased voltage in leads V_5 and V_6;
(4) Early transition from lead V_1 to V_2;
(5) Absence of QRS axis shift;
(6) Predominantly negative T waves in the right precordial leads (due to ischemia involving the proximal third of the LAD).

All the above mentioned criteria are valid in the absence of RVH, septal hypertrophy or lateral MI, and other causes of PAF.

LEFT SEPTAL FASCICULAR BLOCK: VECTORCARDIOGRAPHIC CRITERIA (ALL IN THE HP)[53,54,15]

QRS loop in the HP with an area predominantly located in the left anterior quadrant (>2/3 of the loop facing the orthogonal X lead: 0° to ±180°);

(1) Absence of normal convexity to the right, of the initial 20 ms of the QRS loop.
(2) Discrete dextro-orientation with moderate delay of the vector from 20 to 30 ms.
(3) Anterior location of the vector from 40 to 50 ms;
(4) Posterior location with a reduced magnitude of the vector from 60 to 70 ms.
(5) Maximal vector of the QRS loop located to the right of +30°.
(6) Intermittent or transient anterior displacement of the QRS loop.
(7) T loop with posterior orientation tendency (useful for the differential diagnosis with posterior MI).

(8) The QRS loop rotation may be:
  (8a) Counterclockwise: incomplete LSFB.
  (8b) Clockwise: advanced or complete LSFB or in association with complete RBBB, LAFB, or LPFB.

Typical examples of LSFB are shown in Figures 1A–C, 2A–C. Figures 3 and 4 show examples of intermittent or transient exercise-related LSFB.

DIAGNOSIS OF LSFB

The differential diagnoses of LSFB include all possible etiologies of PAF.

The electrocardiographic diagnosis of PAF can be made when the R wave voltage in any anterior or anteroseptal precordial leads from V_1 (+115°) through V_4 (+47°) is greater than the normal upper limit for gender and age. Electrovectorcardiographic criteria of PAF should be age and gender-related.[64]

Causes of Prominent Anterior Forces (PAF). Differential Diagnosis of LSFB.

In the presence of PAF in the right and/or middle precordial leads V_1 through V_3 or V_4, the following clinical and/or electrovectorcardiographic differential diagnosis should be accounted:

1. Normal variant: PAF are observed in about 1% of normal subjects.[64] Two main types can be distinguished: Normal variant with counterclockwise (CCW) rotation of the heart around the longitudinal axis and athlete's heart, described predominantly in black athletes.[65]
2. LSFB (see above).
3. Misplaced precordial leads.[66]
4. Strictly posterior, postero-lateral-inferior MI (from the "old" nomenclature)[67] or lateral (from the "new"nomenclature).[68]
5. Right ventricular hypertrophy (RVH).[69]
6. Diastolic LVH.[70]
7. Complete RBBB.
8. Ventricular preexcitation (Wolff-Parkinson-White syndrome), with accessory pathway located in the posterior region (Type A).[71]
9. Hypertrophic cardiomyopathy: HOCM and NO-HCM forms.[72]
10. Duchenne muscular dystrophy.[73]
11. Endomyocardial fibrosis.[74]
12. Dextroposition.[75]

CONCLUSIONS

There is conclusive evidence of a left human trifascicular His system. The isolated left septal fascicular block has been described by several authors using different terminology inducing confusion in clinicians and researchers. Traditional teaching does not include the concept of a trifascicular left system. The authors provided with the current acceptable terminology and definitions for electrovectorcardiographic diagnosis of left septal fascicular block. Additionally, a call is made to the international societies to generate a position paper or consensus to unify nomenclature and definitions.

The authors have declared no Conflict of Interest.

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