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Review Article

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Left Septal Fascicular Block: Evidence, Causes, ECG/VCG Criteria, Differential Diagnosis and Proposal for New Concepts

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Abstract

The existence of a tetrafascicular intraventricular conduction system is widely accepted by researchers. In this review, we have updated the criteria for Left Septal Fascicular Block (LSFB) and the differential diagnosis of prominent anterior QRS forces. More and more evidence points to the fact that the main cause of LSFB is critical proximal stenosis of the left anterior descending coronary artery before its first septal perforator branch. The most important characteristic of LSFB that has been incorporated in the corresponding diagnostic ECG criteria is its transient/intermittent nature mostly observed in clinical scenarios of acute (i.e. acute coronary syndrome including vassospastic angina) or chronic ischemic coronary artery disease (i.e. exercise-induced ichemia).In addition, the phenomenon proved to be phase 4 bradycardia rate-dependent and induced by early atrial extra stimulus. Finally, we believe that intermittent LSFB has the same clinical significance as the "Wellens' syndrome" and the "de Winter pattern" in the acute coronary syndrome scenario.

Keywords: Left septal fascicular block; Etiologies; Electro-vectorcardiographic criteria

Introduction

The fact that the Left Bundle Branch (LBB) is divided into three fascicles or a "fan-like interconnected network" **[1]** in the vast majority of human hearts is supported by observations from many diagnostic modalities, including, including anatomy **[2-6],** pathological anatomy, histology, histopathology, cardiac microanatomy (micro-Computed Tomography (CT), using iodine contrast in three dimensional visualization) **[7],** electrocardiography **[8],** vectorcardiography **[9,10],** treadmill stress testing **[11-14],** electrophysiology [15-17], body surface electrocardiographic mapping **[18],** evidence from explanted isolated normal human hearts **[19-25],** from ungulate heart animal models **[26-28],** and canine heart **[29,30]** studies. The left His system is trifascicular consisting of a left superior or anterior fascicle (LAF), a Left Inferior or Posterior Fascicle (LPF), and a Left Septal Fascicle (LSF) or left septal Purkinje network in the left ventricular conduction system **(Figures 1A-D).** Consequently, the term "hemiblock" to describe the block of one of the left fascicles, established in the last century by Mauricio B Rosenbaum **[31]** and his research group, should be updated.

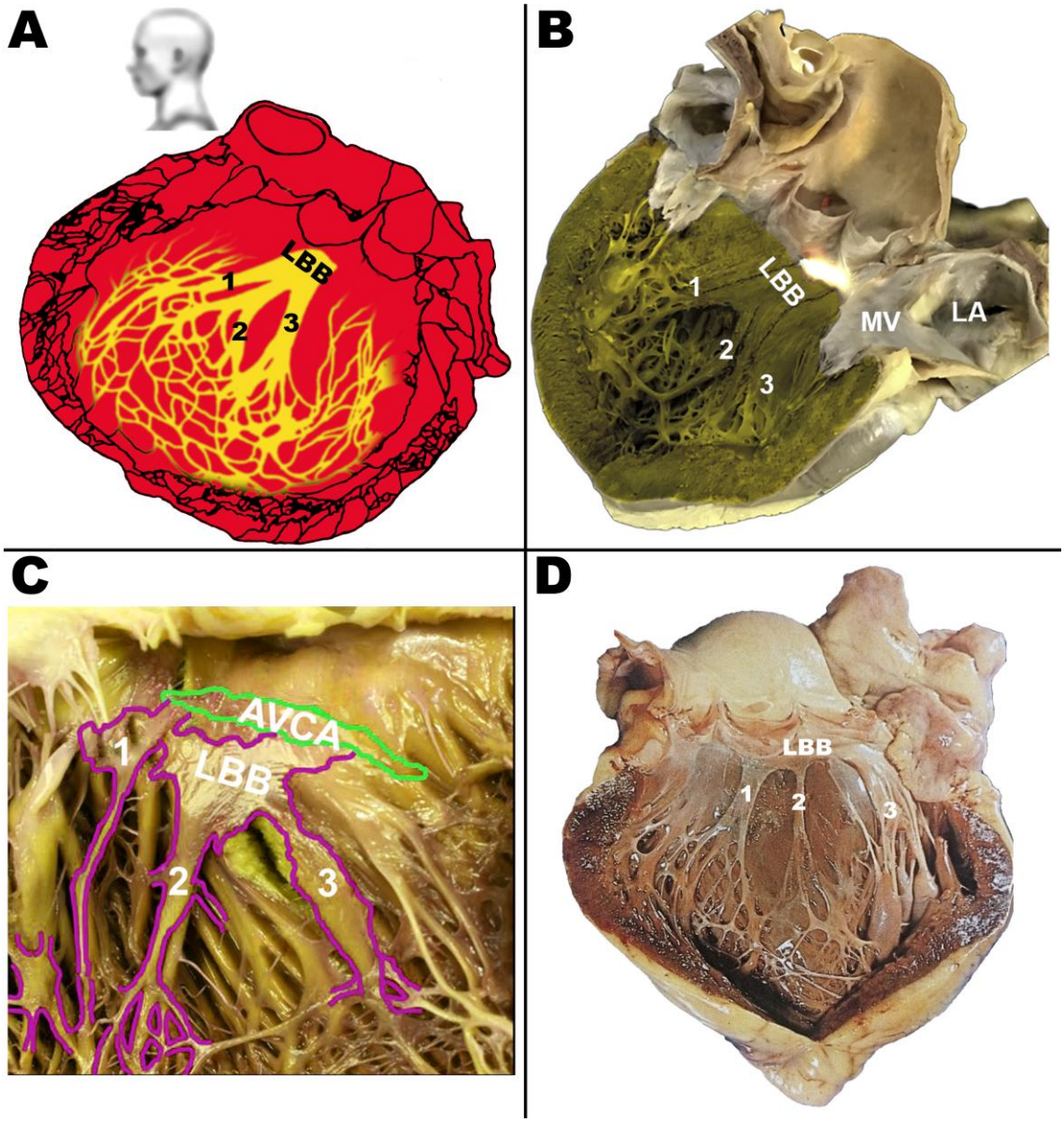

Figure 1: Anatomical studies demonstrating the trifascicular nature of the left bundle branch of His in the human heart. A) From the monograph of Sunao Tawara (1906). It has been rotated placing the image in a more attitudinally appropriate orientation. The reconstructions made by the authors of this article show the origin and distribution of the Left Bundle Branch (LBB) within the Left Ventricle (LV) with its three fascicles; [32] B)The highlighted area in dark yellow shows the limits of the endocardial position of the LBB of His and its 3 fascicles, the left anterior fascicle (1), the left septal or middle fascicle (2), and the left inferior or posterior fascicle (3) in a left posterior oblique view. Modified from [21] (MV, mitral valve; LA, left atrium). C) The fascicular arrangement of the LBB explored by micro-computed tomography. This figure demonstrates high-resolution ($73 \times 73 \times 73\ \mu m^3$) whole-heart micro-computed tomography data. A comparison between the visual anatomy in a macro photograph and the segmented atrioventricular conduction axis and left sided bundle branches of the heart is shown in this panel (AVCA: atrioventricular conduction axis). Modified from [33]; D) The heart in ‘‘Valentine’’ position showing the left Hisian system with its three fascicles in a myocardial dissection, courtesy from Dr. de Almeida.

The LBB originates at the crest of the muscular interventricular septum, immediately inferior to the membranous septum at the base of the interleaflet triangle between the right and noncoronaryaortic cusps. The neighboring structures of the LBB are: noncoronary and right coronary aortic valve cusps, aortic ring, membranous septum, subaortic septal endocardium, apex of the muscular septum, and the RBB. The close anatomical relation between the LBB and the aortic valve is the main reason for new Left Bundle Branch Block (LBBB) after Transcatheter Aortic Valve Implantation (TAVI).

Left Septal Fascicle: Possible Anatomical Types/Variants

Anatomopathological studies showed that the LSF has diverse morphologies and considerable variability in its structure. Thereby, five basic anatomical variations can be described **[24,32-35].**

Type I: the LSF arises independently from the stem of LBB **(Figure 2).**

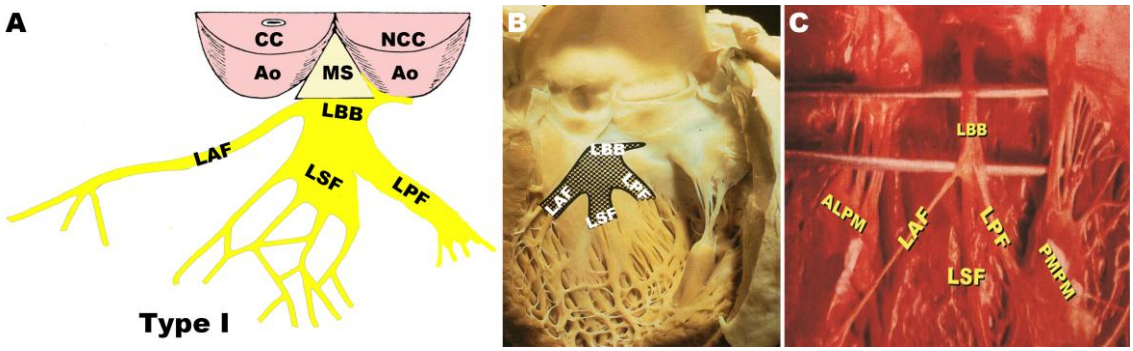

Figure 2: A) Type I: the LSF arises independently from the stem of LBB; B) Modified from **[36]** C) Lateral views of the endocardium of the interventricular septum in the human heart. In this example the LSF originates from the stem of LBB. Additionally, the LAF conducts to the ALPM of the mitral valve and the LPF straight to the PMPM of the mitral valve. Figure extracted from Rosenbaum's classical book **[37].** ALPM: Anterolateral Papillary Muscle; PMPM: Posteromedial Papillary Muscle; Ao: aorta; MS: Membranous Septum; CC: Coronary Cusp; NCC: Non-Coronary Cusp; LAF: Left Anterior Fascicle; LPF: Left Posterior Fascicle; LSF: Left Septal Fascicle; LBB: Left Bundle Branch.

Type II: the LSF originates from the LAF of the LBB **(Figure 3A).**

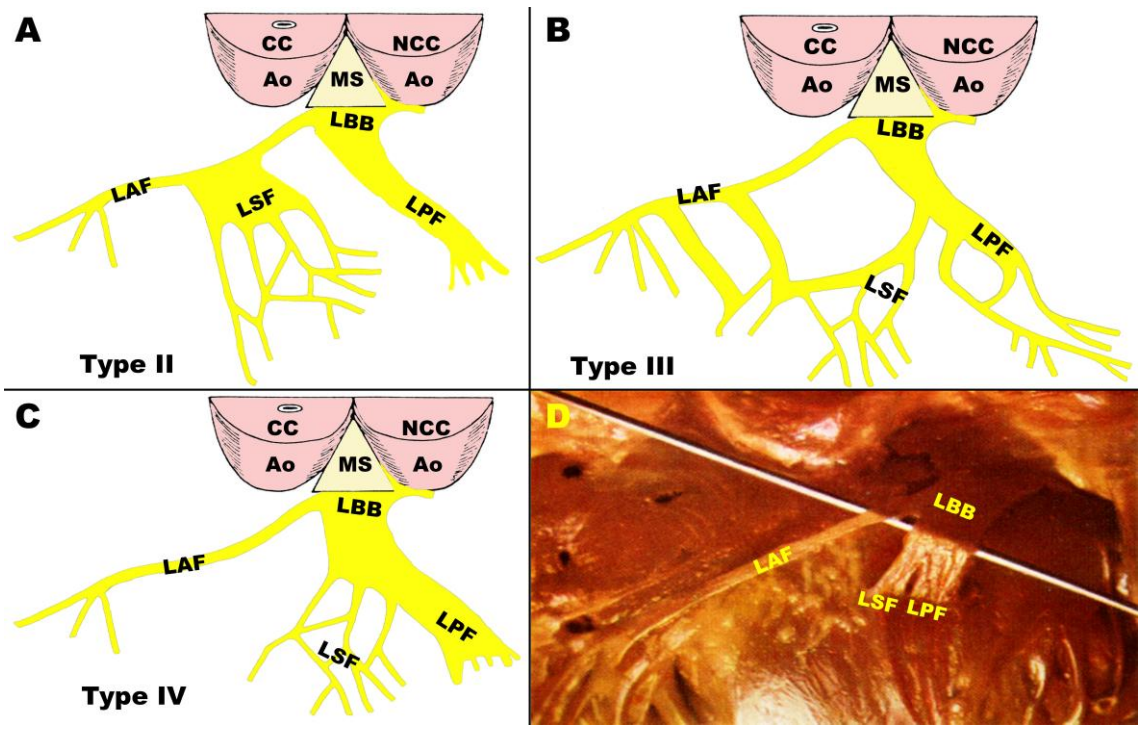

Figure 3: A) Outline of the type II LSF variant; B) Outline of the type III LSF variant; C) Outline of the type IV LSF variant, which represents the most frequent type. The left septal fibers are almost always present in mammals, emerging from the LPF at different points of its trajectory; **[38]** D) Demonstration of the type IV LSF anatomic variation. In this figure, extracted from the original book by Rosenbaum et al, the LSF originates from the LPF. Rosenbaum considered these as "false sinews or tendons" originating from the LPF **[37].** Studies of the histopathologic correlates of left posterior fascicular block have indicated the presence of lesions along the LPF and the LSF of the LBB **[39-42].** Ao: aorta; MS: membranous septum; CC: coronary cusp; NCC: non-coronary cusp; LAF: left anterior fascicle; LPF: left posterior fascicle; LSF: left septal fascicle; LBB: left bundle branch.

Type III: the LSF originates concomitantly with the other two fascicles (LAF and LPF) **(Figure 3B).**

Type IV: the LSF originates from the LPF **(Figure 3C)** and its respective anatomic demonstration (3D).

Type V: the LSF is represented as a “fan-like interconnecting network” **(Figure 4).**

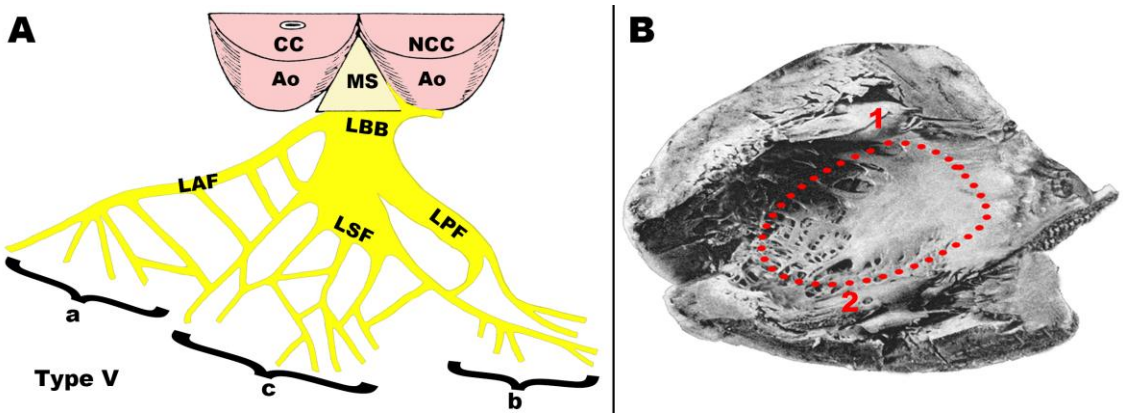

Figure 4: A) Outline of the type V variant with ventricular distributions of the three fascicles of the LBB (modified from Hecht 1973): **[43]** LAF is distributed in the anterolateral papillary muscle of the mitral valve, anterosuperior region of the septum and anterolateral LV wall (a). LPF is distributed in the base of the posteromedial papillary muscle of the mitral valve, posteroinferior region of the septum and laterobasal part of the LV (b).LSF is distributed in the apical and centroseptal region and low septum (c); B) Illustration of the type V LSF anatomic variation. The LSF is an interconnected network of Purkinje fibers that join the other two fascicles: the anterolateral papillary muscle (1) and the posteromedial papillary muscle (2) of the mitral valve. Ao: Aorta; MS: Membranous Septum; CC: Coronary Cusp; NCC: Non-Coronary Cusp; LAF: Left Anterior Fascicle; LPF: Left Posterior Fascicle; LSF: Left Septal Fascicle; LBB: Left Bundle Branch.

The Concept of Left Septal Fascicular Block (LSFB)

Possible main causes of LSFB

- **In the setting of coronary artery disease: [44,45].**
- **Acute Coronary Syndrome (ACS)** as a consequence of critical proximal obstruction of Left Anterior Descending (LAD) coronary artery before its first septal perforator branch **[46-49].**
- **ACS as a consequence of critical proximal obstruction of left main coronary artery (LMCA) [50,51].**

A 72-year-old male patient was admitted to the emergency room with typical precordial pain that was relieved by intravenous nitroglycerin. Coronary angiography revealed Left Main Coronary Artery (LMCA) spasm and proximal critical obstruction of the Left Anterior Descending coronary artery (LAD). The patient was urgently submitted to Coronary Artery Bypass Graft Surgery (CABG), which was uneventful. Electrocardiogram (ECG) at admission is shown in **Figure 5A** and on the third day after successful CABG is shown in **Figure 5B.**

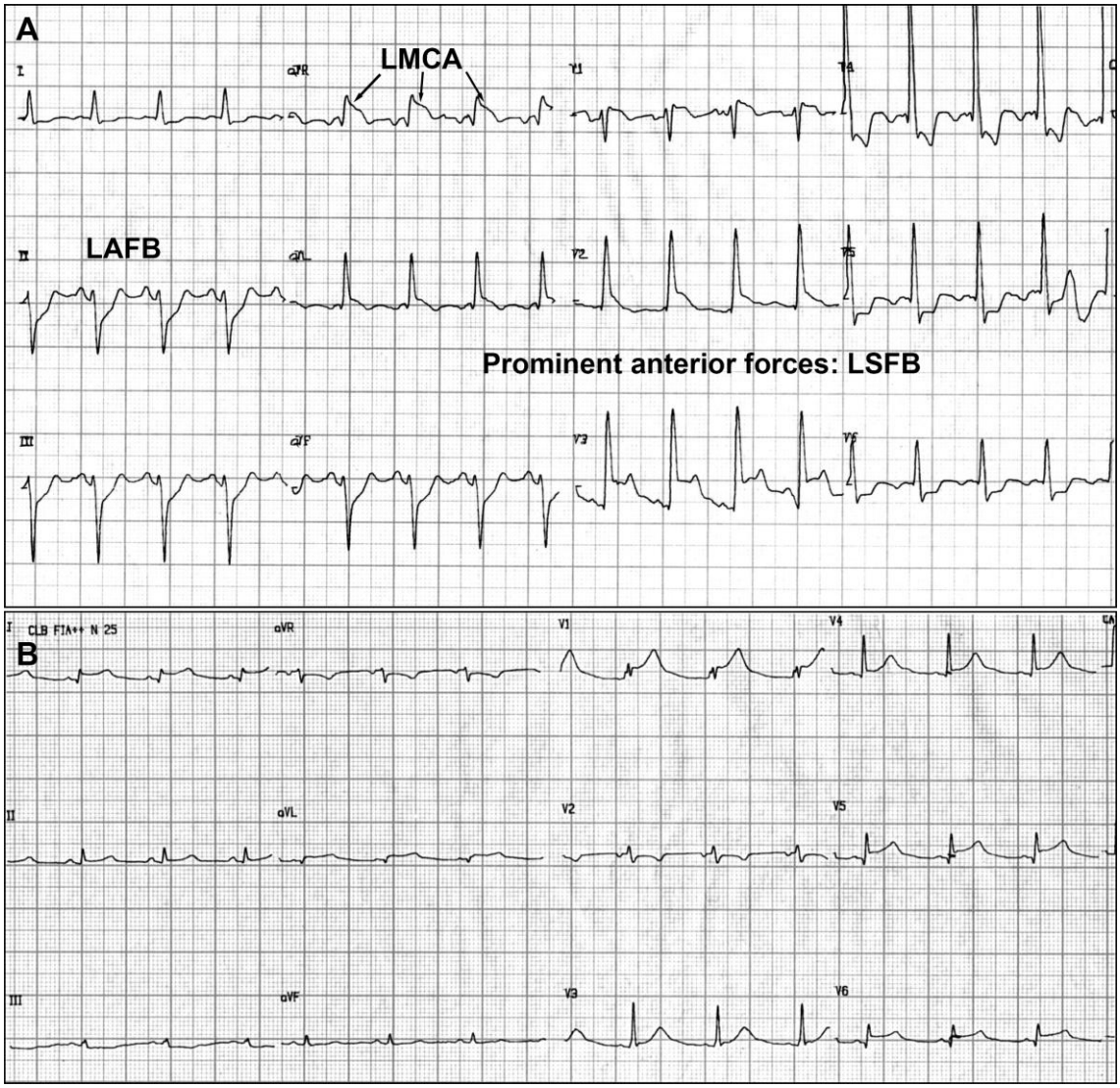

Figure 5: A) Left Anterior Fascicular Block (LAFB) and Left Septal Fascicular Block (LSFB): Prominent Anterior Forces and widespread ST depression accompanied by ST elevation in leads aVL, V1-V3 and aVR (aVR >V1) suggestive of obstruction in the left main coronary artery (LMCA). Laboratory: there was no increase of biomarkers of myocardial injury (creatine kinase- myocardial band/troponin) preoperatively. B) Both fascicular blocks have disappeared: extreme shift of the QRS electric axis to the left in the frontal plane (left anterior fascicular block is no longer present, and prominent anterior forces (left septal fascicular block) have disappeared**.** Postoperative ST elevations of "pericarditis" type are seen.

- **In the setting of stable effort angina during stress testing [52] (Figure 6).**

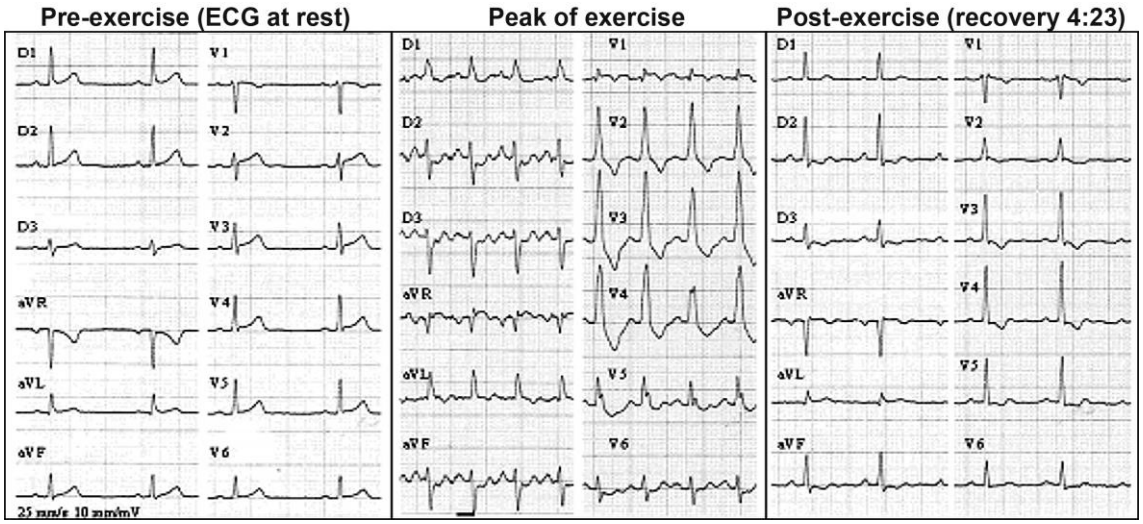

Figure 6: During peak exercise, there are tall R waves in V1-V4, which is the hallmark electrocardiographic finding in left septal fascicular block associated with left anterior fascicular block (left bifascicular block).

- **Concomitantly with the Wellens' Syndrome [53] (Figure 7A). Figure 7B** shows an ECG of the same patient performed one year before the clinical manifestations.

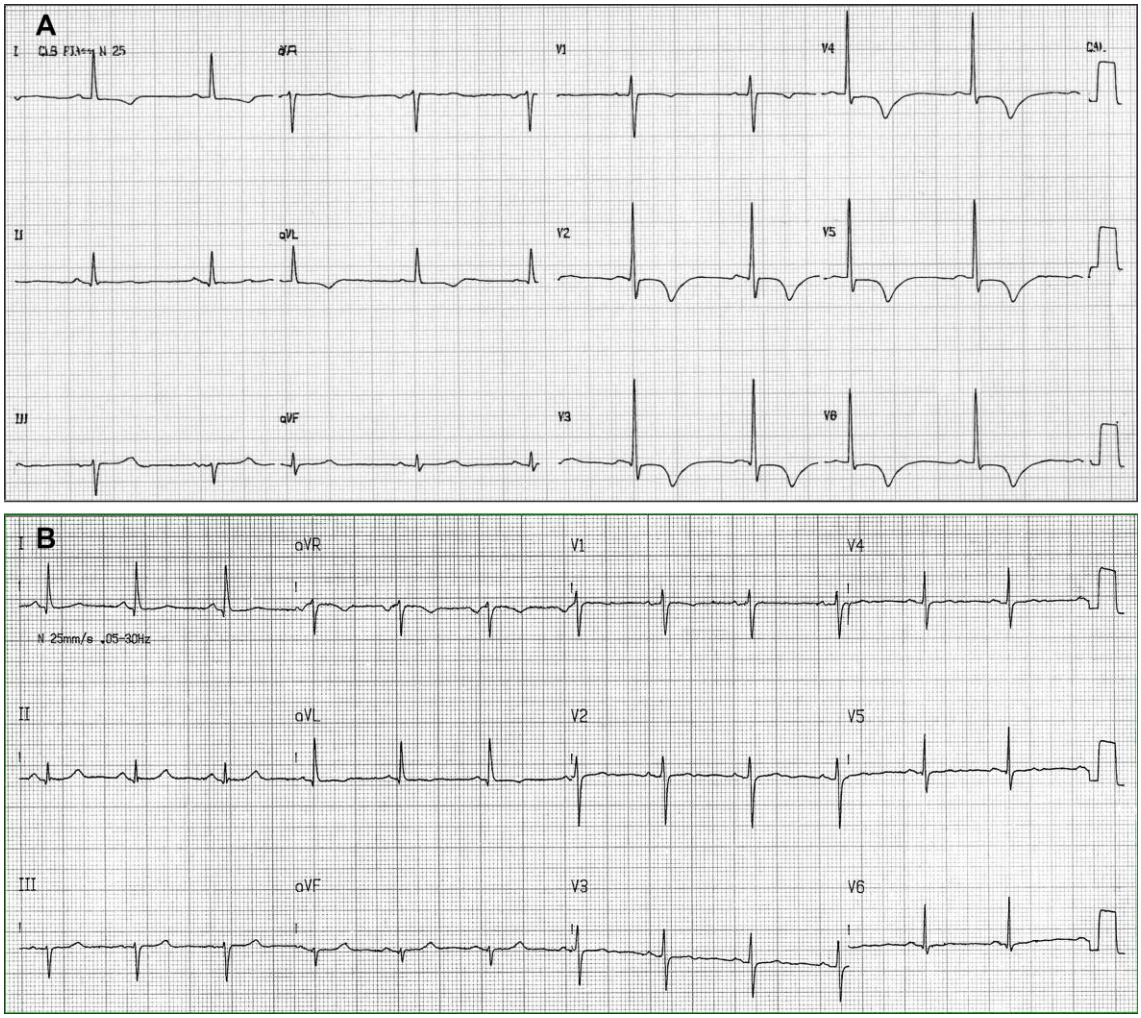

Figure 7: A) An ECG recorded during chest pain of a patient upon arrival to the emergency department. Deep negative and broad-based T-wave inversions in the precordial leads from V_2 through V_6, with high-voltage R wave in V_2 (R=18 mm). Initial small q waves are present in V_2-V_3. The normal left septal q waves in the lateral precordial leads are absent. R/S ratio in V_2> 2. S-wave depth in V_2<5 mm. **Conclusion:** Type 2 Wellens' pattern associated with prominent anterior forces: several left septal fascicular block criteria are present; B) There is a biphasic T-wave in most of the precordial leads and an rS -type QRS complexes in lead V2. Initial q waves are present in the lateral leads I, aVL, V5 and V6.

During episodes of Prinzmetal variant angina or vasospastic angina [51,54]. The phenomena occur only after many minutes of ischemia. Thus, R-wave voltage appears to increase only after coronary occlusions have lasted more than a few minutes. Ischemia lasting for at least 180 seconds was associated with a 53% increase in R-wave voltage size and a 135% prolongation of Ventricular Activation Time (VAT) or R-Wave Peak Time (RWPT) in the open-chest dog model **[55].** Gambetta and Childers, in experimental myocardial infarction **[56],** noted that conduction into the ischemic zone was accelerated at 30 seconds, thereafter becoming progressively prolonged. They attributed this sequence to the biphasic effects of local extracellular hyperkalemia, in which excitability and conduction are initially enhanced before entering the phase of deterioration **[57].** This conclusion is supported by observations in isolated guinea pig myocardium, where a biphasic change in conduction velocity was found with increases in potassium concentration **[57].** The enhancement of R-wave

height probably reflects the "evasion" of canceling vectors by the delayed activation process in the left septal fascicle area. Likewise, it could be postulated that initial shrinkage of the R wave is due to optimization of the canceling process by accelerated conduction. The duration of the first phase of ischemia is probably determined by the completeness of the coronary occlusion, influenced in turn by the presence or absence of collateral coronary blood flow.

- **With underlying Phase 4 LSFB (Figure 8).**

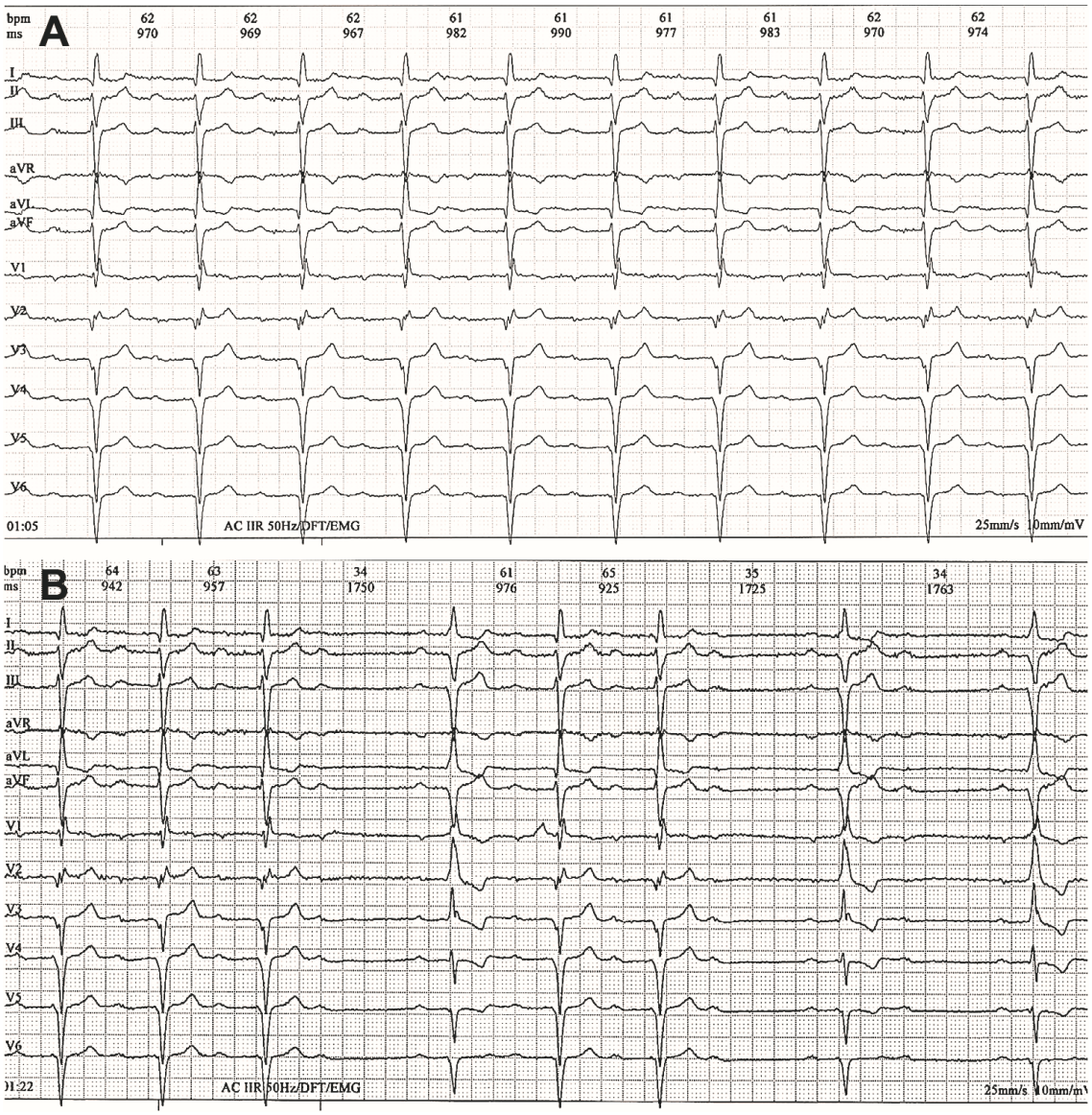

Figure 8: A) Surface 12-lead Electrocardiogram (ECG) during routine checkup; B) ECG depicting *2nd degree* Atrioventricular Block (AVB) and phase 4 or bradycardia-dependent Left Septal Fascicular Block (LSFB). In the present case, prominent anterior QRS forces are depicted in an intermittent fashion in an asymptomatic individual, associated with preceding pauses triggered by *2nd* degree AV block. Upon recovery of conduction in the AV node and resolution of pauses, prominent anterior QRS forces (transient LSFB) disappear. This behavior indicates a phase 4 or bradycardia-dependent mechanism. Phase 4 or bradycardia-dependent aberrancy or block occurs when a supraventricular or ventricular impulse reaches a diseased His-Purkinje system during phase 4 of the action potential at a time when sodium channels are inactive. The observations from the present case confirm previous observations of the existence of Phase 4 block and contribute to better understanding of the pathophysiology of the left His system.

Electrophysiological Study Evidence

Perrin et al, [58] when discussing LSFB, paraphrased Einstein: "Everything should be made as simple as possible, but no simpler." These authors presented a patient case, where they found that not all patterns of

ventricular conduction were explained by Rosenbaum's concept. A 43-year-old man underwent an electrophysiological study for premature ventricular complexes associated with Left Ventricular (LV) dysfunction. The baseline ECG showed intermittent Left Anterior Fascicular Block (LAFB) and absent septal Q waves. With the catheter nestled in the right aortic sinus facing the left/right commissure, a fascicular signal was recorded with presumed LBB/LAF-onset 28 ms pre-His and 35-ms pre-QRS. Pacing captured the fascicle without local myocardial capture. The resultant QRS was narrow (98 ms) with a normal frontal axis but prominent anterior QRS forces **(Figure 9).**

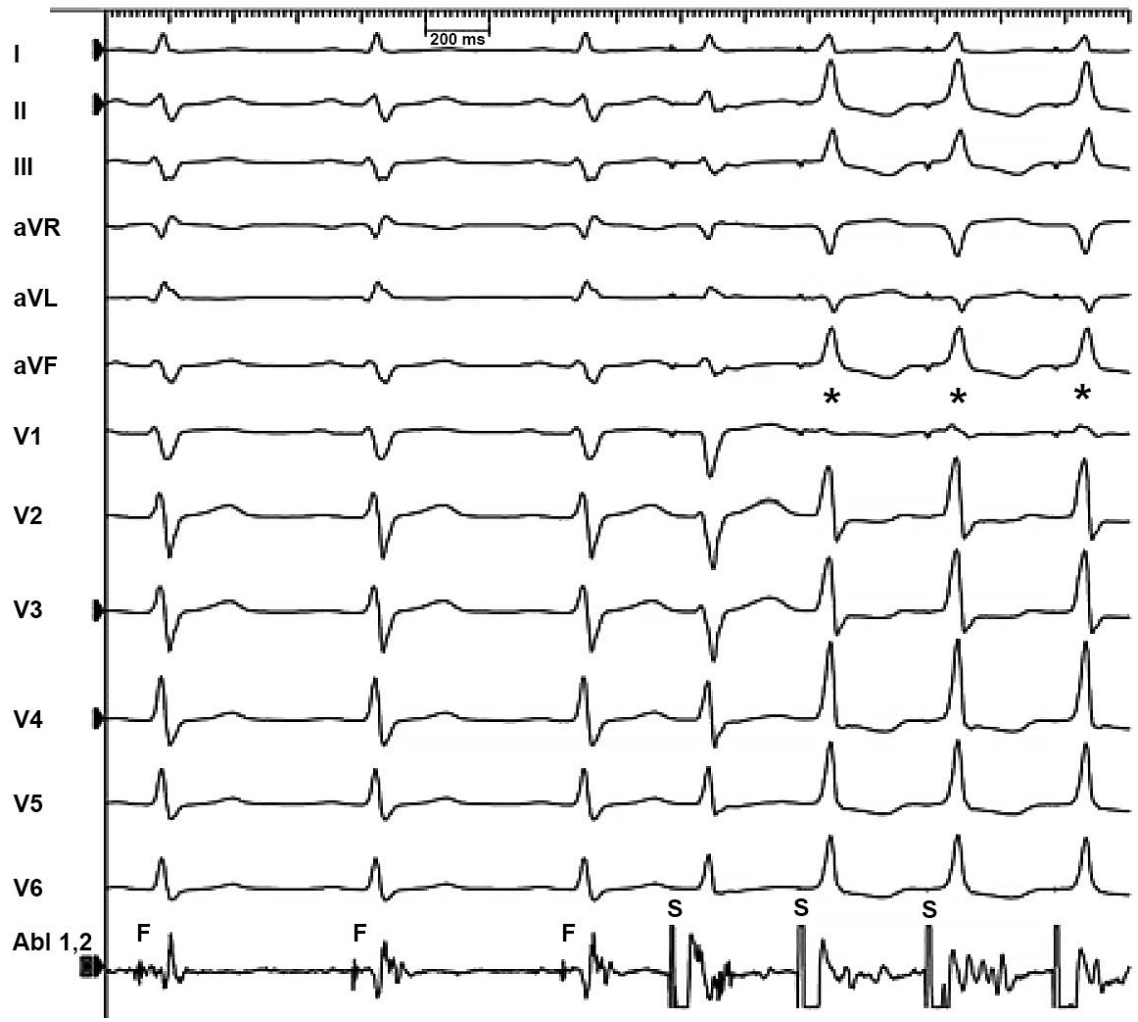

Figure 9: Electrocardiogram recorded while pacing (S) from the ablation catheter resting in the right coronary sinus. The first three sinus beats show a typical pattern of left anterior fascicular block (LAFB) with a QRS duration of 110 ms; each QRS is preceded by a fascicular signal (F). The final three beats (*) result from capture of the left septal fascicle - note prominent anterior QRS forces.

The authors reasoned that activation in the fascicle traveled both anterograde to the Purkinje network subtended by the LAF and retrograde to the bifurcation of the RBB and LBB and thence (anterograde) to the LPF and RBB. This hypothesis explains the narrow QRS (activation of the RBB) and normal frontal axis (activation of LAF and LPF), but not the appearance of prominent anterior QRS forces. The authors suspected that the patient, in addition, had a delay or block in his LSF accompanying the delay in the LAF at baseline. Pacing the LAF

proximal to its termination “compensated” for its slow conduction, but the LSF could only be activated by the signal passing retrograde into the LAF and then anterograde along the length of the LSF (where it was blocked or met significant delay). PAFs may have many causes, including LSFB. The case was presented in relation to the authors' comments: “We share the authors' desire that a tetrafascicular conception of intraventricular conduction should ultimately prevail. The trifascicular Rosenbaum's model is simple, but simpler than true”. Dr. de Pádua expressed this very succinctly: ***“If hemiblocks do exist, they are only two - if a third one is postulated, hemiblocks do not exist!”.***

After Self-Expandable Percutaneous Transcatheter Aortic Valve Implantation [59] (Figure 10).

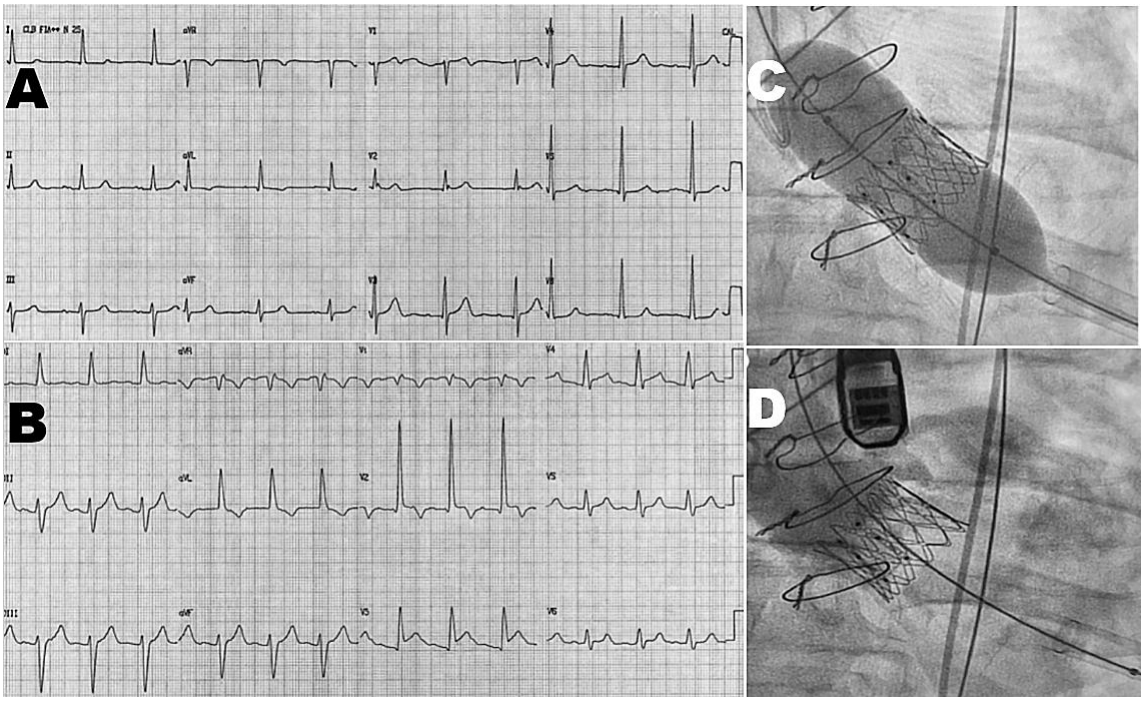

Figure 10: A) Electrocardiogram (ECG) before the procedure. P duration = 120 ms, bimodal aspect, PR interval = 220 ms, QRS axis +10°, QRS duration 110 ms, early precordial transition (between V1 and V2), embryonic initial q wave from V2 through V6. Conclusion: left atrial enlargement, doubtful first-degree AV block (elderly), embryonic q wave from V2 through V6; B) ECG performed immediately after the procedure. P duration = 120 ms, bimodal aspect, QRS axis −50°, QRS duration 130 ms, early transition in precordial leads, initial embryonic q wave from V2 to V4, tall R wave in V2: prominent anterior QRS forces (PAF), prolonged R-wave peak time in V1–V2 (≥35 ms), R wave in crescendo from V1 to V2 and decrescendo from V3 through V6, absence of initial q wave in the left leads (I, aVL, V5–V6) (by absence of first septal vector of the middle third of the left septal surface). ST segment elevation followed by negative T wave in the right precordial leads and lead aVL. Conclusion: left atrial enlargement, left anterior fascicular block and left septal fascicular block(= left bifascicular block) and ischemic changes in the anteroseptal wall. C) Catheter-balloon inflation and prosthesis opening; D) control aortography showing that the coronary ostia are intact, no significant aortic insufficiency.

In Chronic Chagasic myocarditis [60,61] (Figure 11A). ECG/VCG correlation in the frontal **(Figure 11B)** and horizontal planes **(Figure 11C).**

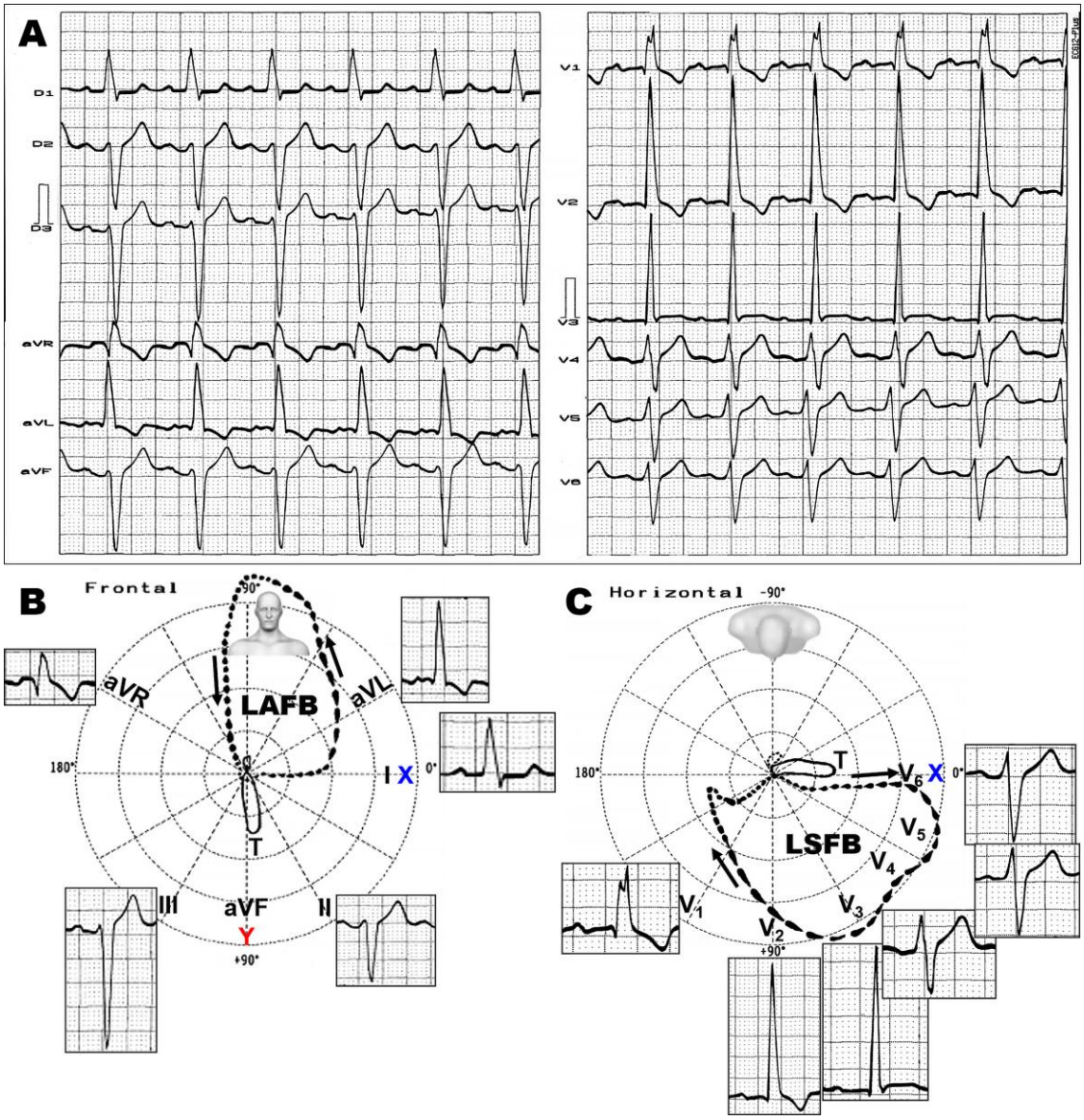

Figure 11: A) Clinical diagnosis: chronic chagasic myocarditis. Electrocardiographic diagnosis: sinus rhythm, heart rate: 88 bpm, P-wave duration: 100 ms, P-wave voltage: 1.5 mm, frontal P-wave axis: +60º, PR interval: 200 ms, frontal plane QRS axis (SÂQRS): -80º, QRS duration: 115 ms, QRS morphology in aVL: qR (with high voltage); II, III and aVF: rS (SIII>SII); V1 to V3: qR with PAF, V_4 to V_6: rS. Conclusion: Left Ventricular Hypertrophy (LVH), Left Anterior Fascicular Block (LAFB) and Left Septal Fascicular Block (LSFB): left bifascicular block. B)Frontal Plane:QRS axis (SÂQRS): -80º, SIII > SII, SIII > 15 mm: Left Anterior Fascicular Block (LAFB) type IV of Rosenbaum, QRS and T loop angle near 180º: Left Ventricular Hypertrophy (LVH) with strain pattern of repolarization. C) Horizontal Plane: prominent anterior QRS forces, apiculate aspect of R wave in V2-V3, R "in crescendo" from V1 to V2, absence of initial q waves in left lateral leads (absence of l_{AM} [anteromedial] septal vector), end-conduction delay: Left Septal Fascicular Block (LSFB) + some degree of Right Bundle Branch Block (RBBB).

Following Alcohol Septal Ablation for Hypertrophic Obstructive Cardiomyopathy [62] (Figure 12).

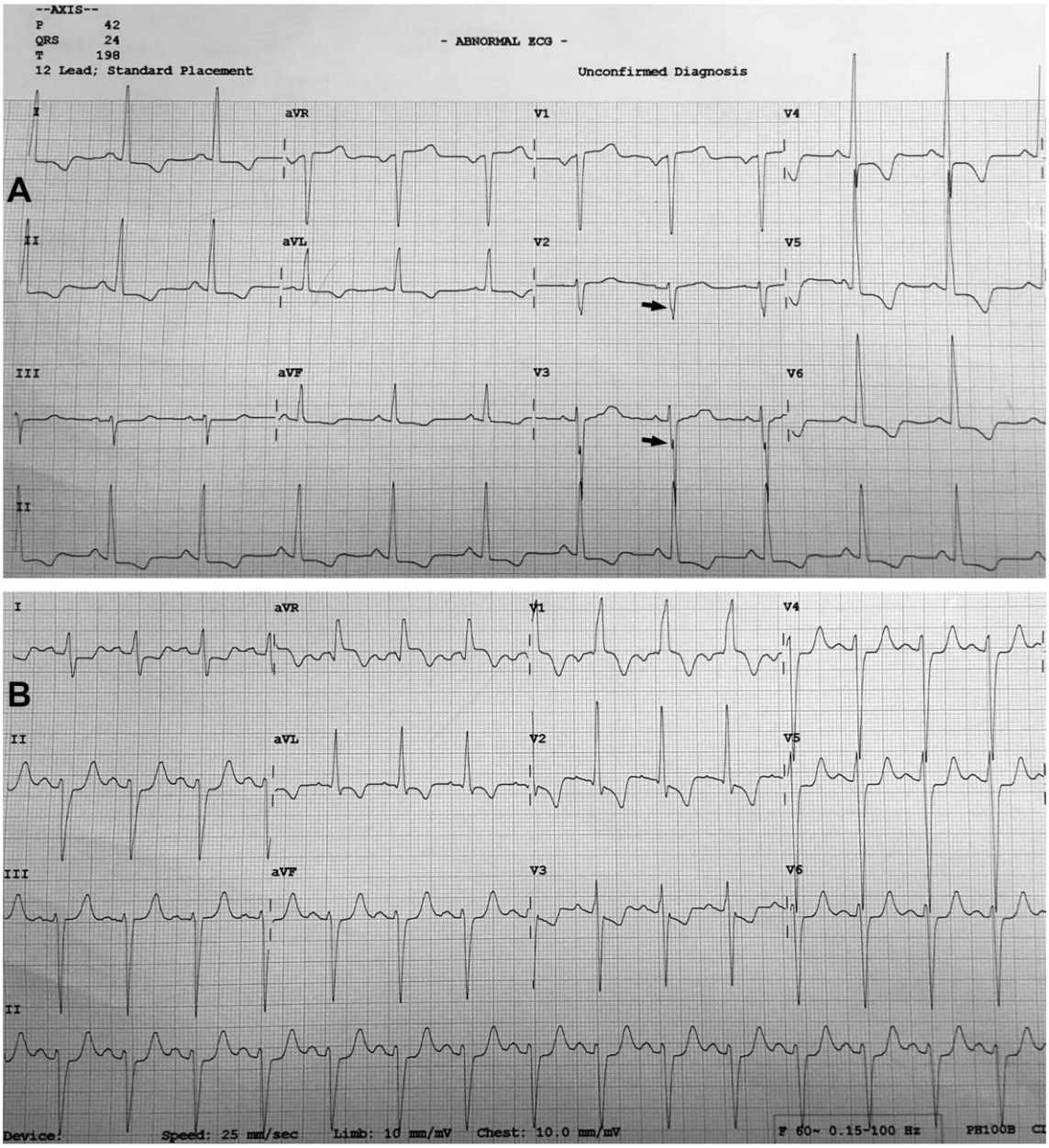

Figure 12: A) Electrocardiogram (ECG) performed before alcohol septal ablation (ASA). P-wave duration 120 ms, terminal mode negative of P in V1 > 40 ms, Morris index >0.04 mm x s, QRS axis +24°, QRS duration 85 ms, positive Sokolow-Lyon index >35 mm, strain pattern of repolarization, insinuation of fragmented QRS (arrows). Conclusion: Left Atrial Enlargement (LAE) and Left Ventricular Hypertrophy (LVH) and fragmented QRS (indicator of fibrosis in Hypertrophic Cardiomyopathy [HCM]).Note: The association of LAE and LVH + fQRS. This pattern is very frequent in hypertrophic obstructive cardiomyopathy. **B)** ECG obtained immediately after the ASA procedure. V1: LAE (Morris criteria), prolonged P duration 120 ms and QRSd 120ms andST-segment depression followed by symmetric negative T-wave, Complete Right Bundle Branch Block (CRBBB). V2: qRs pattern, prominent anterior QRS forces in the right precordial leads V1-V2, “R-wave in crescendo” from V1 to V2 and decrescendo from V2 to V6, prolonged R-wave peak time (> 40 ms) in V1-V2, R-wave

voltage > 15 mm and embryonic initial q wave in V1-V2 absence of q in I, V5-V6 as a consequence of absence of first septal vector. Conclusion: LAE, LVH, atypical Left Anterior Fascicular Block (LAFB), Left Septal Fascicular Block (LSFB) and RBBB (type of trifascicular block previously not reported in the literature: RBBB + left bifascicular block).

Associated with the Type 1 Brugada Electrocardiographic Pattern [63]

A male Asian patient, 58-year-old, had approximately three days previously experienced several episodes of constrictive precordial pain, each of short duration (≤10 min), relieved during rest. During some of the episodes, pain radiated to the mandible and the inner part of the left upper arm and to the wrist. Medical history: hypertension diagnosed five years ago, familial type II dyslipidemia controlled by rosuvastatin, smoking since adolescence (stopped two years previously). Family history: a brother and a first-degree cousin (29 and 35 years old, respectively) from his father's side had experienced sudden death during nighttime sleep. In both, a molecular autopsy was performed with the Illumina HiSeq 2500 sequencing system that revealed a novel mutation, R376H, in the first pore segment in the *SCN5A* gene **(Figure 13).**

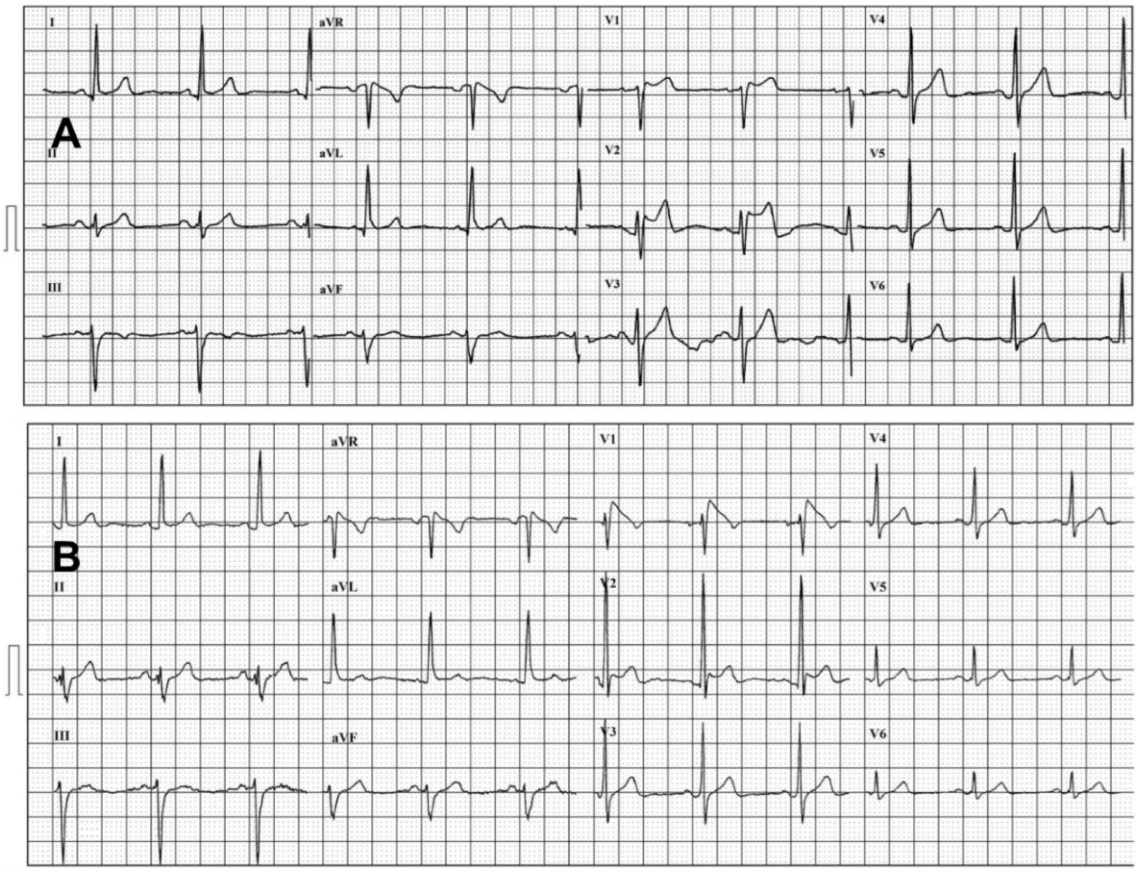

Figure 13: A) Sinus rhythm, normal P-wave parameters, normal PR interval duration (148 ms), first degree AV block (124 ms), QRS axis at −25°, normal QT-QTc, and type-2 Brugada pattern: wide β angle (angle formed by the ascending ramp of S and the descending of r′ in V2), ST segment with saddle-back appearance followed by plus-minus biphasic T wave. B) Sinus rhythm, heart rate 75 bpm, normal P wave, PR interval duration 144 ms, QRS axis - 45°, SIII > SII, absence of initial q wave in the lateral leads I, aVL, V5–V6. In the precordial leads, type-1 BrP, wide initial Q wave and prominent QRS anterior forces in V2–V3 and absence of q wave in V5–V6.

Conclusion: Left Anterior Fascicular Block (LAFB) and Left Septal Fascicular Block (LSFB) (left bifascicular block) andtype-1 Brugada pattern and probable electrically inactive forces in the septal wall. Percutaneous Coronary Intervention (PCI) was immediately performed with successful drug-eluting stent deployment in the LAD. Sequential ECGs showed constant type 1 Brugada pattern, but prominent anterior QRS forces disappeared immediately after PCI. Conclusion: coronary artery disease and BrS. Transient LSFB was caused by critical proximal obstruction of the LAD before its first septal perforator branch.

Kearns-Sayre syndrome: It is a neuromyopathic disorder associated with mitochondrial abnormalities and characterized by the triad of chronic external ophthalmoplegia, atypical pigmentary retinopathy, and progressive conduction system disorders. Ragged red muscle fibers that seem to contain an excess of altered mitochondria are observed. The disease affects both sexes during the first or second decade. The following manifestations are observed: central bilateral sensorineural deafness, pyramidal signs, ataxia, asymmetrical ptosis, external ophthalmoplegia, and progressive muscular weakness secondary to myopathy associated with a significant increase of proteins of cephalorachidian liquid.[10]Cardiac involvement in the Kearns-Sayre syndrome may manifest by different progressive degrees of atrioventricular block through the His-Purkinje system, giving rise to bundle branch block (right or left), frequently associated with left fascicular blocks.

Case report: A 23-year-old man of African descent was referred to a cardiologist for evaluations based on identified alterations in retinal examination that are commonly associated with cardiac disease **(Figure 14).**

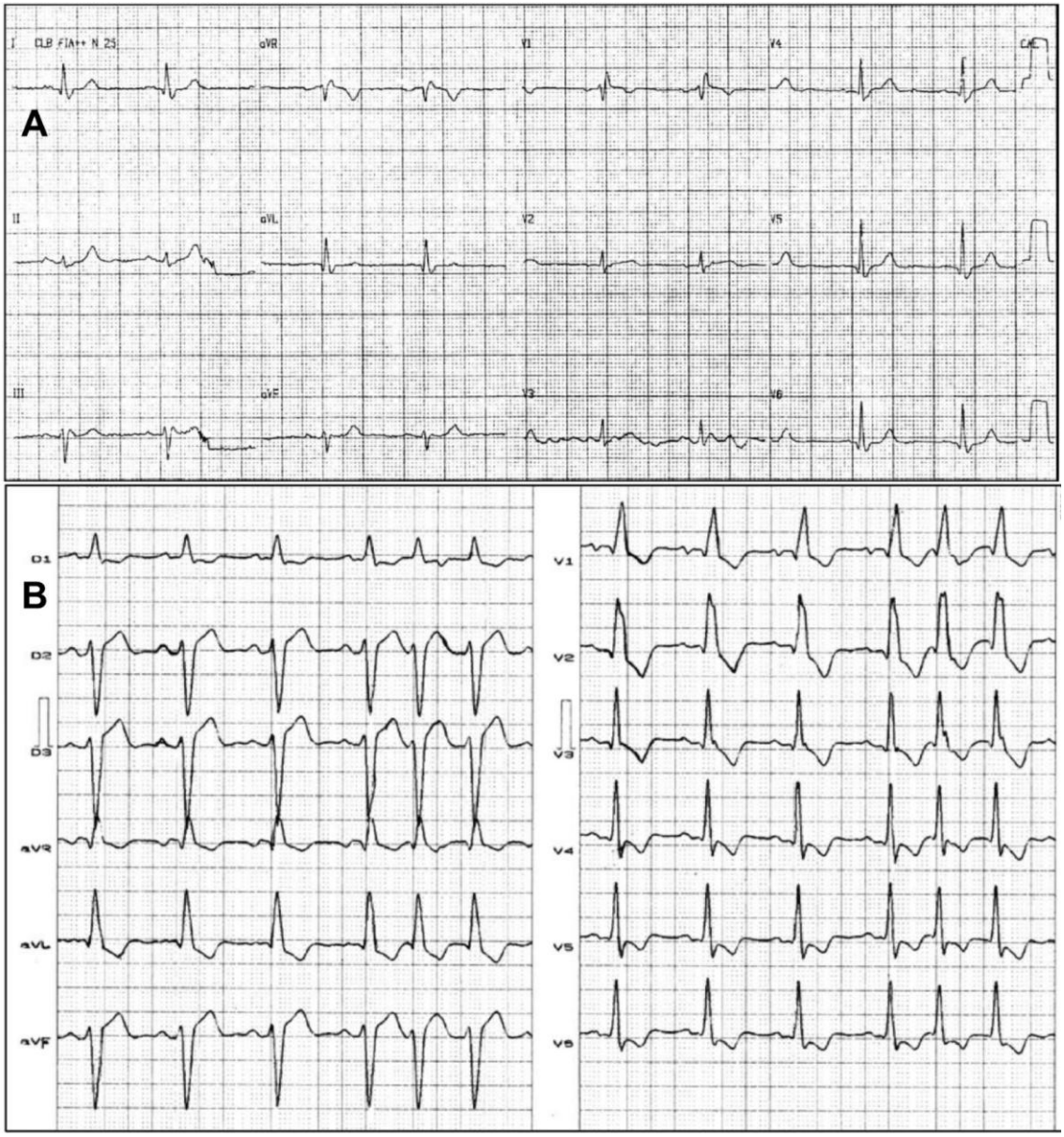

Figure 14: A) Preoperative Electrocardiogram (ECG) before appendicectomy at the age of nine. Conclusion: Incomplete Right Bundle Branch Block (IRBBB). B) ECG performed 14 years later. Complete RBBB (CRBBB): QRS duration >120 msec, S in V5-V6 and final broad R in aVR; atypical left anterior fascicular block (LAFB): extreme left axis deviation in the frontal plane, SIII > SII, rS pattern in the inferior leads and qR pattern in aVL. Why atypical? Because of the absence of initial q wave in I, V5-V6, characteristic of LAFB; Left Septal Fascicular Block (LSFB): this diagnosis is based on the presence of prominent anterior QRS forces not explained by right ventricular enlargement or lateral MI (previously named "posterior" MI); Trifascicular block: RBBB and LAFB and LSFB; Sudden increase of heart rate from the fifth beat onward: supraventricular tachycardia with a rate close to 150 beat/min, which maintains a morphology similar to the baseline complex.

Prominent Anterior QRS Forces: Concept/Values

Prominent anterior QRS forces in the ECG occur when the voltage of the R wave in any precordial lead of the anterior or anteroseptal wall from V1 (+115°) to V4 (+47°) is greater than the normal maximal limit for gender and age. In the presence of prominent anterior QRS forces in the anterior wall (tall R waves) in the right and/or

middle precordial leads V1 through V3 or V4, certain entities need to be considered in the differential diagnosis **[64] (Figure 15).**

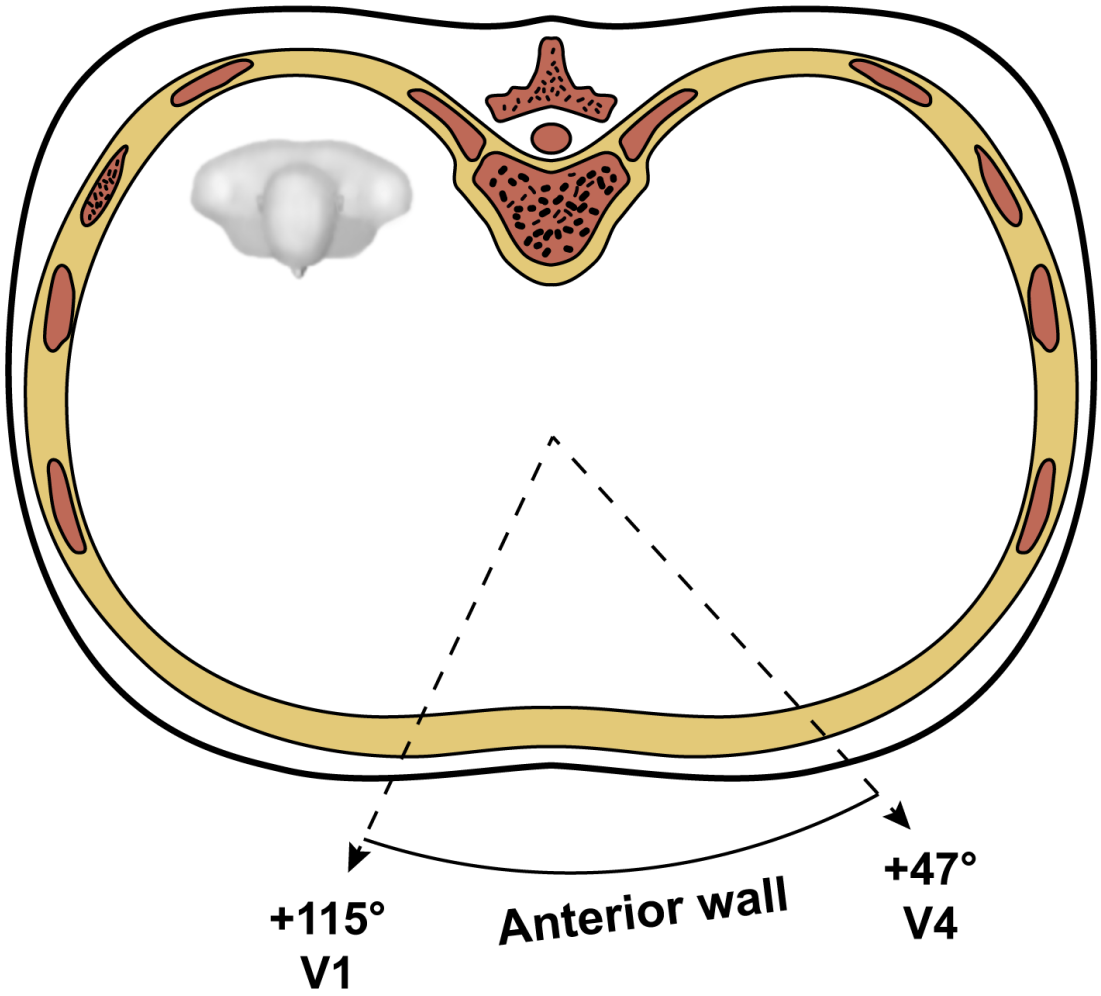

Figure 15: The mean amplitude and the lower and upper range of the R wave in lead V1 in three different age categories is shown in **Table 1.** We consider values exceeding the upper range as one of the diagnostic criteria for PAF (for example, in lead V_1 in 20-30 years old, R wave voltage > 8.9 mm in women and > 5.3 mm in men) **[65-67].**

Table 1: Maximal normal R wave voltage (mm) from V1 to V4 related gender and age.

Age	Mean in women	Mean in men	Range in women	Range in men
V1				
20-30	3.3	1.6	0.3-8.9	0-5.3
30-40	2.2	1.6	0.2-5.4	0-5.8
40-60	1.7	1.4	0.1-4.9	0.1-4.0
V2				
20-30	7.4	4.6	1.7-13.9	1.1-9.2
30-40	5.4	3.7	0.6-12.1	0-4.10.1

40-60	4.6	3.6	0.6-12.0	0.2-9.1
V3				
20-30	11.6	8.2	2.2- 26.6	2.3 -17.5
30-40	9.4	7.1	2.2-22.5	0-8.23.3
40-60	8.4	7.1	1.4-11.6	1.0-17.7
V4				
20-30	16.6	11.5	6.1- 27.7	5.0 -19.6
30-40	14.8	11.8	5.2-29.2	4.1-25.9
40-60	14.2	12.4	5.2-25.6	3.7-23.6

Another criterion used by some authors to consider the presence of PAF uses the R/S ratio in V1. Thus, an R/S ratio in V1 ≥1 is considered abnormal in adults. Tall lead V1 (tall R in V1) is defined as an R/S ratio ≥ 1. From our point of view, these values cannot be considered as valid for the diagnosis of PAF, since in 1% of normal individuals this ratio is considered as a normal variant. In lead V2, ≈ in 25% of men and 12% of women the R/S ratio is 1. **Table 1** shows normal amplitudes of R wave in lead V1 to V4 (mm).

Differential Diagnosis of LSFB with Other Causes of Prominent Anterior QRS Forces

- Normal variant with marked counterclockwise rotation of the heart around the longitudinal axis **[8].** PAF is observed in ~1% of normal subjects; **[68]**
- Athlete's heart; **[69]**
- Misplaced precordial leads; **[68,70]**
- Lateral myocardial infarction (previously known as strictly posterior)
- Vectorcardiographic right ventricular hypertrophy; **[71]**
- Diastolic LVH; **[71,72]**
- RBBB; **[73,74]**
- Ventricular pre-excitation with left-sided accessory pathways may give rise to prominent R-waves in the right precordial leads simulating right ventricular hypertrophy **[75] (Figure 16).**

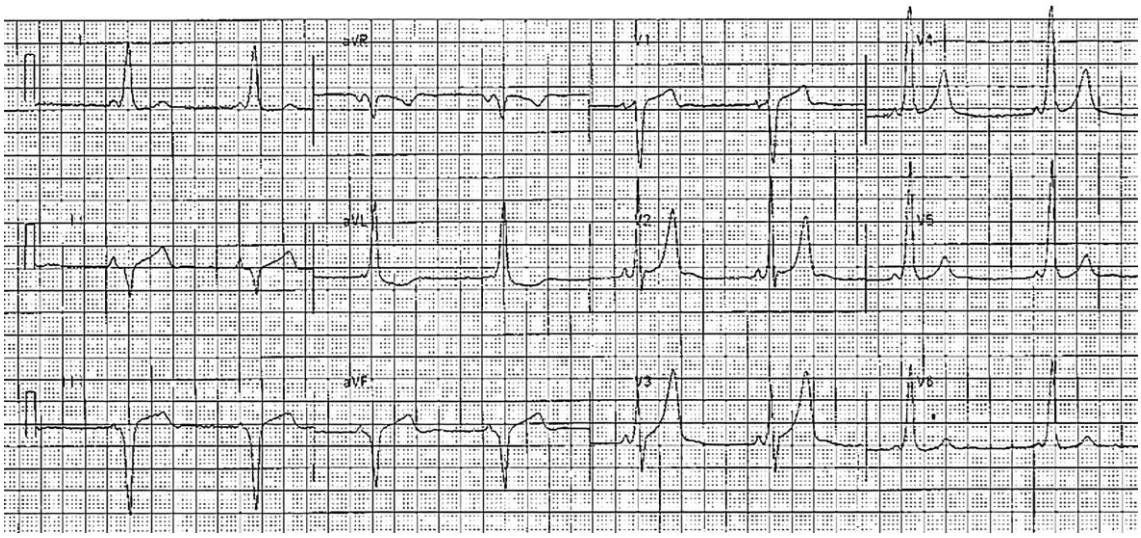

Figure 16: The 12-lead Electrocardiogram (ECG) shows a short PR interval, delta waves, pseudo-LVH pattern in left-sided limb leads (I, aVL), and pseudo inferior myocardial infarction and prominent anterior QRS forces (PAF) in V2.

- Hypertrophic cardiomyopathy of the basal portion of the interventricular septum, isolated apical hypertrophic cardiomyopathy (associated with giant negative T waves and a "spade-like" remodeling of the left ventricle), and diffuse concentric LVH. Tall right precordial R waves may constitute a marker of hypertrophic cardiomyopathy in asymptomatic young adults. Echocardiography is useful for diagnostic purposes; **[76,77]**
- Cardiomyopathy associated with Duchenne muscular dystrophy; **[78,79]**
- Endomyocardial fibrosis; **[80]**
- Acquired dextroposition of the heart: Normally about one third of the heart lies to the right and two thirds to the left of the midsternal line. Acquired dextrocardia, therefore, may be defined as a displacement to such an extent that the whole heart lies to the right of the left sternal border. Acquired displacement of the heart to the right varies from a minor discernible amount to an extreme condition in which the heart lies in contact with the lateral or posterolateral thoracic wall. As a rule, the long axis remains unchanged with the apex farthest to the left. Right-sided pneumonectomy causes dextroposition; **[81]**
- LSFB; and a combination of the above.

Proposed ECG/Vectrocardiograhic VCG) Criteria for LSFB

- QRS duration (QRSd): Normal or with a minor increase when isolated (up to 110 ms), in general close to 100 ms. Isolated LSFB does not increase the QRSd by more than 25 ms, due to multiple interconnections between the divisions/fascicles of the LBB in the endocardium of the left ventricle (Rosenbaum's "passage way zone").When associated with LAFB, LPFB or BBBs, the QRSd is ≥120 ms. Thus, a LSFB pattern with a prolonged QRSd indicates the presence of an additional conduction disturbances such as other fascicular block, BBB, myocardial fibrosis/infarction, focal block, or a combination of these.

- QRS axis in the Frontal Plane (FP): Normal when isolated. When associated with LAFB (very frequent), there is left axis deviation between -45° and -90°. When LSFB is associated with LPFB (only one case reported in the literature), **[82]** the QRS axis is deviated to the right.
- Increased VAT/RWPT in V1-V2: ≥ 35 ms. Note: the terminology intrinsicoid deflection is not recommended anymore **[83].**
- R wave voltage of V1 ≥5 mm, R/S ratio >2; S wave depth <5 mm, and characteristic r/R wave voltage "in crescendo" from V1 through V3 and decreasing from V5 to V6.
- R wave amplitude in V2 >15 mm: prominent anterior QRS forces (PAFs). Note: PAFs in the ECG occur when the voltage of R wave in any precordial lead of the anterior or anteroseptal wall from V1 (+115°) through V4 (+47°) **(Figure 2)** is greater than the normal maximal limit for gender and age **[84-86] (Table 1).**
- R/S ratio in V2 >2;
- Possible small (embryonic) initial q wave in V2 and V3, or in V1 and V2 as a consequence of predominance of the first posteroinferior (1_{PI}) over the first anterosuperior (1_{AS}) vector in the initial 20 ms of ventricular activation. Consequently, the resulting vector will have a backward and leftward direction, giving rise to the eventual small initial q wave in V2 or from V1 to V3.
- Absence of q wave in the left precordial leads V5, V6 and I (by absence of the first septal vector, 1_{AM}).
- Appearance of intermittent or transient PAF during the acute phase of acute coronary syndrome/myocardial ischemia/infarction involving the anterior wall before **(Figure 8)** or after Percutaneous Coronary Intervention (PCI) **[12,55,87].** In this scenario, we consider that intermittent PAF has the same clinical significance as of two established ECG patterns: the Wellens' syndrome **[88]** and the de Winter pattern **[89,90].** Our previous publication has reinforced the concept of the Wellens' syndrome associated with intermittent LSFB **[53].** We believe that these three patterns have similar clinical significance, representing different ECG manifestations of critical LAD coronary artery obstruction in acute coronary syndrome. The de Winter pattern has been proposed as representing an ST segment elevation myocardial infarction equivalent **[91].** We think that growing evidence supports the need for urgent or emergent coronary angiography in these clinical scenarios.
- Intermittent LSFB has also been described in patients with stable effort angina due to critical proximal stenosis of the LAD **[52,92]** during treadmill stress testing in patients with severe anterior wall myocardial ischemia,[14] during early atrial extrastimuli with some degree of aberrant conduction, and also in cases characterized by anterior displacement of the QRS-loop in the Horizontal Plane (HP) in VCG **[93,94],** and in association with pause-dependent paroxysmal phase 4 atrioventricular block **[95].** Note: for the LSFB diagnosis it is necessary to rule out the other known causes of PAFs in the right precordial leads (from V1 to V4). In normal subjects, PAFs are observed in ~1%[68] as a consequence of counterclockwise rotation around the longitudinal axis of the heart, resulting in an early QRS transition (V2) to the right in the precordial leads **[8,72,96-98].** PAFS may also be caused by athlete's heart **[64,69],** misplaced precordial leads **[68,70],** lateral or laterobasal myocardial infarction **[99]** (vectorcardiographic types A **[100-102]** and B **[103,104])** right ventricular hypertrophy, diastolic, eccentric or volumetric LVH or septal hypertrophy (increased magnitude of the first anteromedial septal vector or 1_{AM} vector), biventricular hypertrophy **[105]** CRBBB **[73,74],** ventricular pre-

excitation with left lateral (negative delta waves in leads I or aVL, a normal QRS axis and early precordial R-wave transition) or left posterior (manifested negative delta waves in II, III and aVF and a prominent R wave in V1) **[106]** accessory pathway location, obstructive and non-obstructive hypertrophic cardiomyopathy **[107],** Duchenne muscular dystrophy (increased R/S ratio in the right precordial leads (deep Q waves in the lateral leads, conduction abnormalities, and arrhythmias) **[108],** right endomyocardial fibrosis (characteristic peaked P waves in lead II and QR pattern with diminutive R wave in lead V1, dominant R wave in lead V2 in the absence of QR pattern is found in some patients) **[109],** dextroversion and dextroposition of the heart. Dextroposition is a condition in which the heart is on the right side because of factors that either reduce the volume of the right lung or occupy space in the left thoracic cavity. In this condition, the apex lies on the left side, and cardiac anomalies are rare. Reasons for dextroposition include collapse of the right lung, where the heart moves toward the sparsely occupied space on the right and left pneumonectomy (5.6 to 11.2% of cases) associated with Poland syndrome with partial agenesis of two or more ribs and displacement of the heart toward the right side **[110],** and a combination of the previous ones. Out of a total of 25 of our publications on LSFB (indexed to PubMed), the vast majority revealed transient or intermittent PAF [46,47,52,53,59,95], six were review papers and only two papers dealt with permanent PAF. In contrast to LSFB, almost all other causes of PAF result in permanent or fixed ECG changes with the possible exception of other dromotropic disturbances such as ventricular pre-excitation and CRBBB, which are electrocardiographically easily distinguishable from LSFB. Consequently, we considered transient PAFs as the major criterion for LSFB.

Vectorcardiographic Characterization (All in the Horizontal Plane [HP]) [9,10,29,111]

- QRS loop in the HP with an area predominantly located in the left anterior quadrant ($\geq {}^{2}/_{3}$ of the loop area facing the orthogonal X lead: 0º to ±180º);
- Absence of normal convexity to the right of the initial 20 ms of the QRS loop;
- Discrete dextro- or rightward-orientation with moderate delay of the vector from 20 ms to 30 ms;
- Anterior location of the 40 to 50 ms vector;
- Posterior location with a reduced magnitude of the vector from 60 to 70 ms;
- Maximal vector of the QRS loop located to the right of +30º;
- Intermittent anterior displacement of the QRS loop **[44];**
- T loop with posterior orientation tendency (useful for the differential diagnosis with lateral (previously named posterior) MI);
- The QRS loop rotation may be counterclockwise (incomplete LSFB) or clockwise (advanced or complete LSFB or in association with CRBBB, LAFB, or LPFB).

Conclusions

We have comprehensively listed all the ECG/VCG criteria for LSFB, as well as the hitherto published main causes of the conduction disorder. Despite the fact that LSFB is considered a relevant dromotropic disturbance, it is still an ignored/neglected entity. We consider that knowledge about the intermittent form of LSFB observed in the clinical setting of myocardial ischemia has extremely high clinical importance, since it contains the same

clinical information as the Wellens' syndrome and the de Winter pattern (junctional ST depression followed by tall symmetrical T-waves). The development of LSFB in the clinical context of acute coronary syndrome should be considered an ST segment elevation myocardial infarction equivalent, similar to the de Winter pattern, and highly suggestive of critical proximal obstruction of the LAD coronary artery. We have demonstrated a case of Wellens' syndrome associated with an intermittent form of LSFB, which reinforces their common etiology/significance. Proper ECG diagnosis of the intermittent form of LSFB would allow for speedy, appropriate handling in case of acute coronary syndrome.

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