Português

Dinâmico distúrbio de condução intraventricular em idosa coronariana

Reporte de caso

Paciente do sexo feminino, caucasiana, 67 anos, do lar, conhecida diabética tipo 2, hipertensa, dislipêmica e portadora de severa doença arterial coronariana (DAC) com acometimento triarterial (revascularizada em fevereiro/2000: uma ponte de artéria mamaria para DA e duas safenas). Admitida na sala de emergência no dia 29/12/2015 por episódios sequenciais de pré-síncope (4 episódios).

Vem fazendo uso regular de ácido acetilsalicílico 100mg/dia, atorvastatina 40mg/dia, carvedilol 25mg 2x/dia, espironolactona 25mg 1x/dia, e losartana potássica 50mg 2x/dia.

Nesta admissão, o laboratório mostrou níveis de troponina normais, assim como das enzimas cardíacas.

ECO: VE=47/34 AE=40 FE=54% acinesia infero-lateral e ínfero-apical. Sorologia para Chagas negativo.

Trazia consigo a seguinte sequência cronológica evolutiva de seis eletrocardiogramas.

Perguntas:

1. Quais distúrbios dromótropos podem ser diagnosticados em cada um dos traçados?
2. Qual é a abordagem adequada?

English

Dynamic intraventricular conduction disturbance: Tetrafascicular block a new proposal terminology in elderly woman with severe coronary artery disease:

Case report

Female patient, Caucasian, 67 years old, housewife, known type 2 diabetes mellitus, hypertension, hypercholesterolemia with severe coronary artery disease (CAD): triple vessel involvement (coronary artery bypass grafting (CABG in February / 2000: three coronary artery bypass grafts, one mammary artery to LAD and two saphenous vein).

Admitted to the emergency room on Dec 29, 2015 by complaint of repetitive episodes of pre-syncope (4 episodes). See ECG-6.

She has been using regularly aspirin 100 mg/day, atorvastatin 40 mg/day, carvedilol 25mg 2x/day, spironolactone 25 mg 1x/day, and losartan potassium 50mg 2x/day.

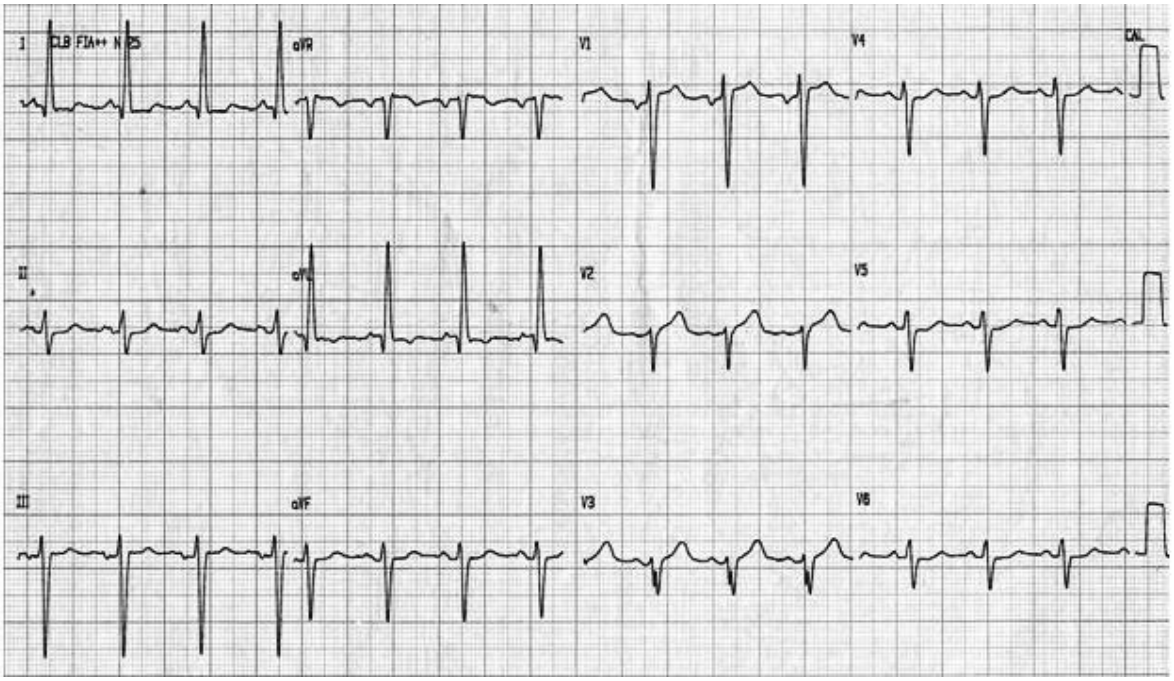
In this admission, lab showed normal Troponin type I and C levels, as well as cardiac enzymes. ECHO: LV=47/34 LA=40 LVEF=54% akinesis inferolateral and ínferoapical. Negative Chagas serology

She brought the next evolutionary chronological sequence of six electrocardiograms.

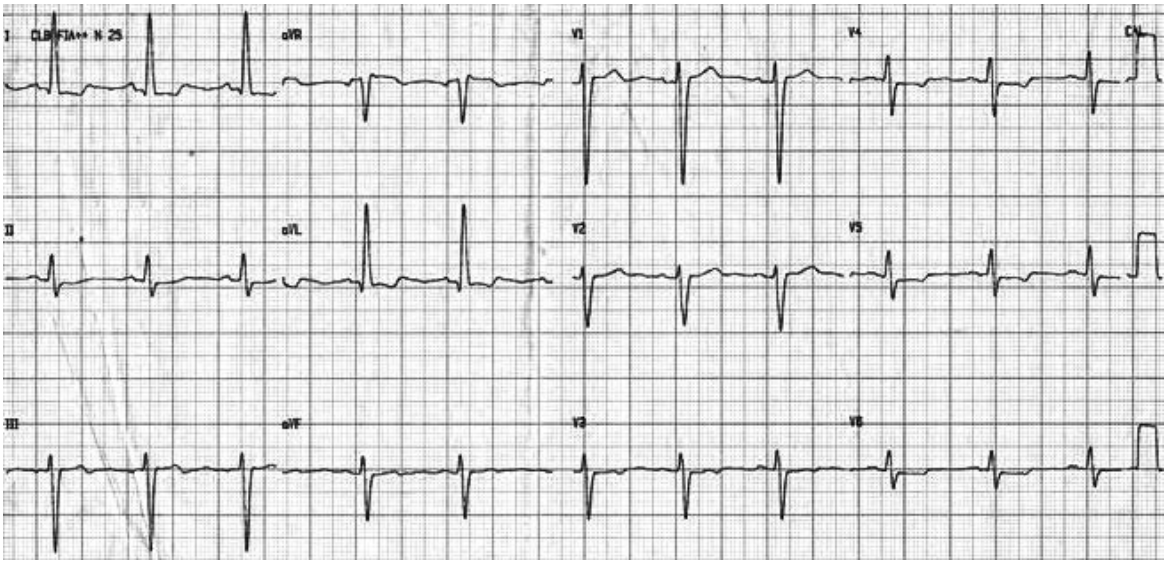
Questions:

1. Which intraventricular dromotropic disturbances can be diagnosed in each ECG?
2. Which is the appropriate approach?

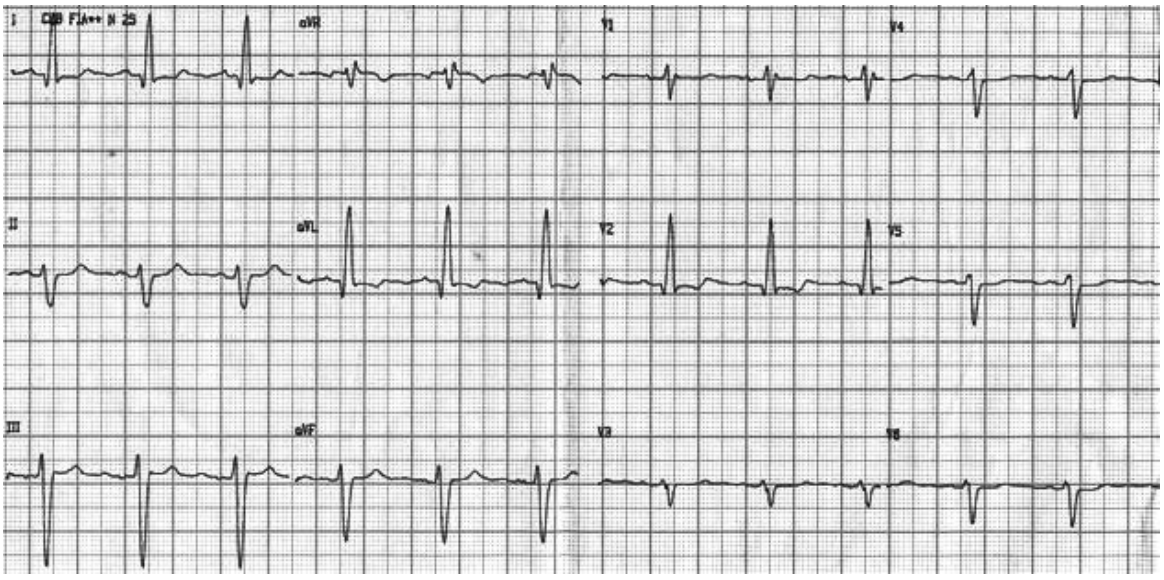
First ECG (Jan 24, 2011)



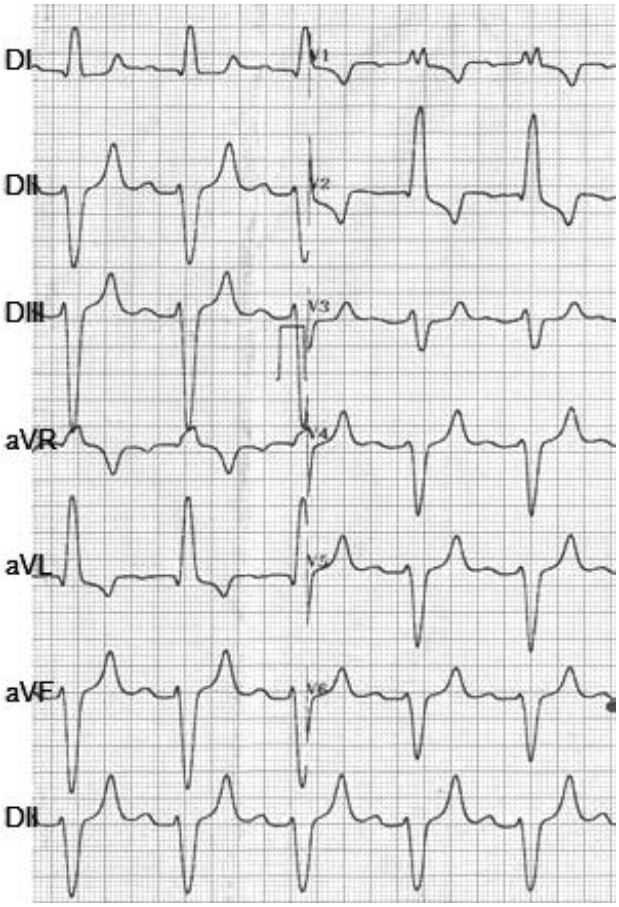
Second ECG (Dec 12, 2012)



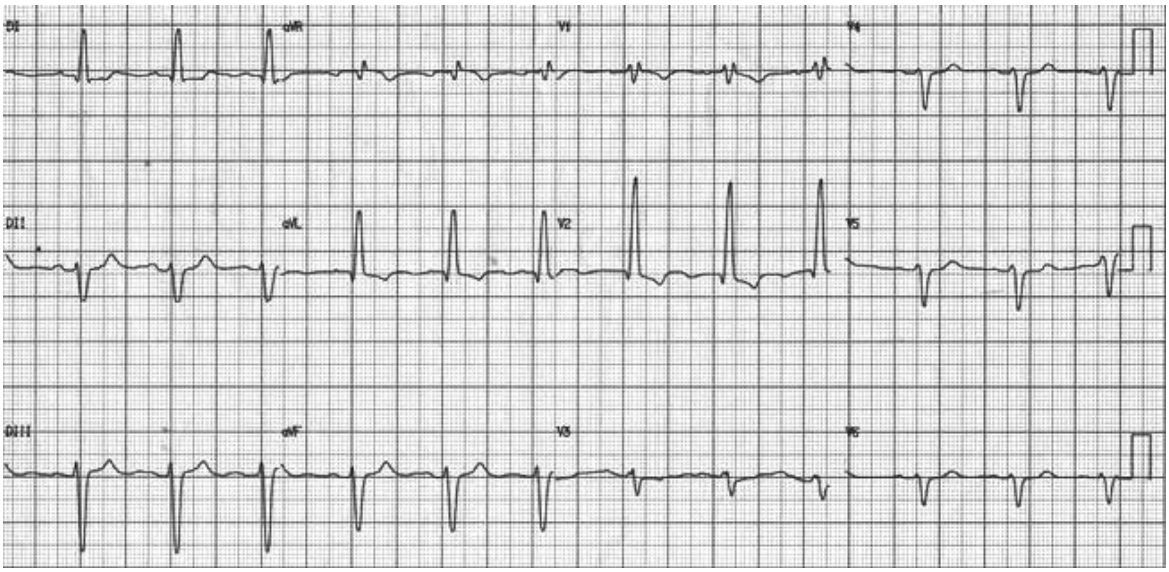
Third ECG (Apr 02, 2014)



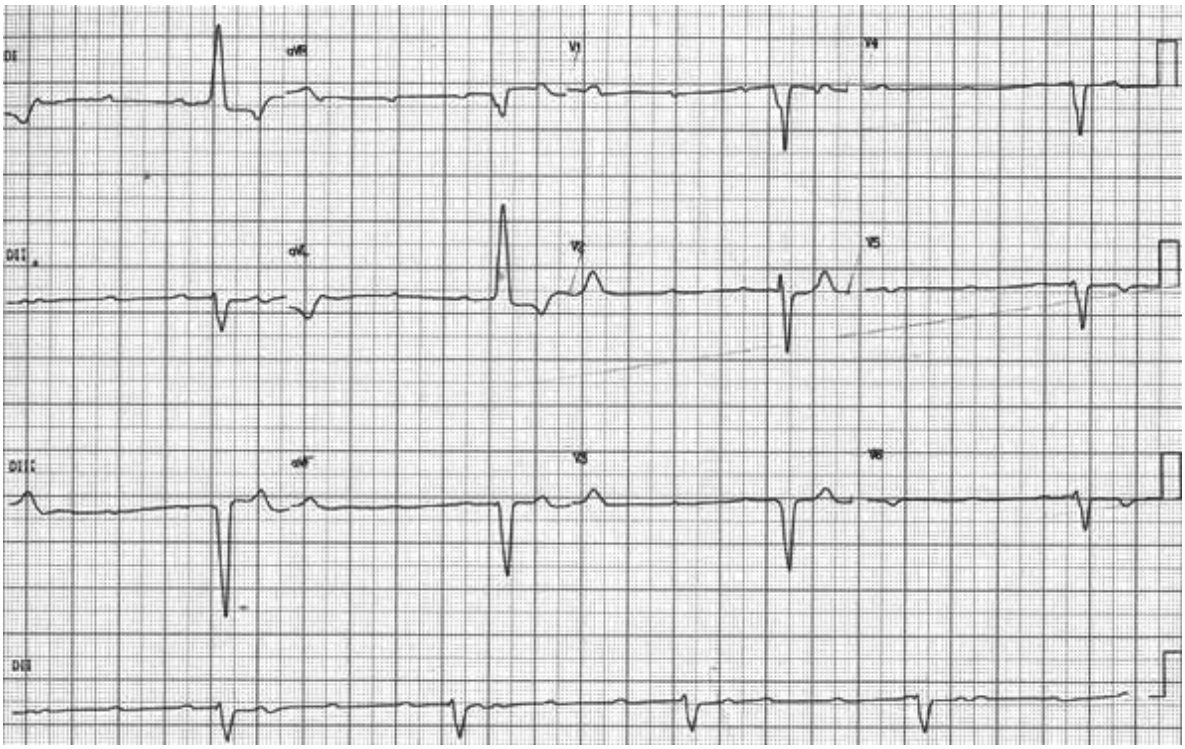
Fourth ECG (Dec 20, 2015)



Fifth ECG (Dec 23, 2015)



Sixth ECG (Dec. 29, 2015)



Colleagues opinions

Raimundo & Andrés,

Very nice tracings. Thank you!

These ECGs show a progressive deterioration of intraventricular conduction. Initially left anterior fascicular block. Then incomplete RBBB followed by high degree RBBB.

Then the left septal fascicle blocks. Finally, high degree or complete A-V block, probably due to the fact that conduction over the posterior fascicle also deteriorated.

Muchas gracias por enviarnos casos tan interesantes!

Un abrazo,

Mario González MD

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Spanish

Hola amigos. Mi opinión: Todos los ECG muestran ritmo sinusal. En referencia a los disturbios conducción tenemos

- **ECG1.** Bloqueo del fascículo anterior izquierdo (q 1 SIII)
- **ECG 2.** Íden agregada isquemia anterolateral
- **ECG 3.** Bloqueo del fascículo o fibras medioseptales (observar las fuerzas anteriores prominentes en plano horizontal) y necrosis de cara lateral
- **ECG 4.** QRS se prolonga a 140 ms con rsr en V1 y S profunda y empastada en V6. Es decir, se agrega bloqueo de la rama derecha. Además, aparece intervalo PR prolongado 300 ms (BAV de primer grado. ¿Puede existir defecto velocidad papel?
- **ECG 5**. Desaparece BAV 1 grado. Resto igual
- **ECG 6.** ¡Aparece BAV COMPLETO! con ritmo ventricular 30- 35 lpm

Síntesis:
Evolución de distintos grados de trastornos de conducción BFA-BFMS-BRD-BAVC .

Conducta: Por ser un paciente sintomático está indicado implante de marcapaso DDD

Juan José Sirena M.D. Santiago del Estero Argentina.

English. Hello friends. My opinion All ECG shows sinus rhythm. In reference to dromotropic disturbances we have

- **ECG1. LAFB (q1-SIII)**
- **ECG 2. Idem added anterolateral ischemia**
- **ECG 3. LSFB or middle septal fibers block (see Prominent Anterior Forces (PAF) in the horizontal plane) and lateral wall necrosis**
- **ECG 4. Prolonged a PR interval (300 ms): first grade AV block) (There may be default speed paper), prolonged QRS duration (140 ms) with rsr´ pattern in V1, deep and wide final S wave in V6: RBBB**
- **ECG 5: Disappear the first degree AV block. Others aspects equal ECG4**
- **ECG 6. Complete AV block with ventricular heart rate between 30- 35bpm**

Synthesis:
Evolution of varying degrees of dromotropic disturbances: LAFB-LSFB-RBBB-Complete AV block Approach: In symptomatic patient DDD pacemaker is indicated.

The first and 3rd ECGs show LAFB pattern, less prominent for ECG
ECG#2, LAFB
ECG #3 also shows a pattern of Left septal fascicular block hence bifascicular block.
ECG# 4th shows RBBB with LAFB and likely Left septal fascicular block as well.
ECG # 5 is similar to #4 but with RV conduction delay.
ECG #6 shows atrial tachycardia with 3:1 AV block with escape focus from junctional tissue

Melvin Sheinman
Professor UCSF School of Medicine 500m Parnassus Avenue, MU San Francisco CA 94143
Melvin.Scheinman@ucsf.edu

Dear colleagues: Dr. Scheinman is best known as the first person to have performed catheter ablation in humans. This was done after extensive animal studies. Dr. Scheinman and his team used high-energy direct current shocks and were the first to ablate accessory pathways and used this technique to ablate the fast AV nodal pathway.
Dr Scheinman thank a lot for your constant support!! Raimundo and Andrés.

Final conclusions

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https://ekgvcg.wordpress.com/
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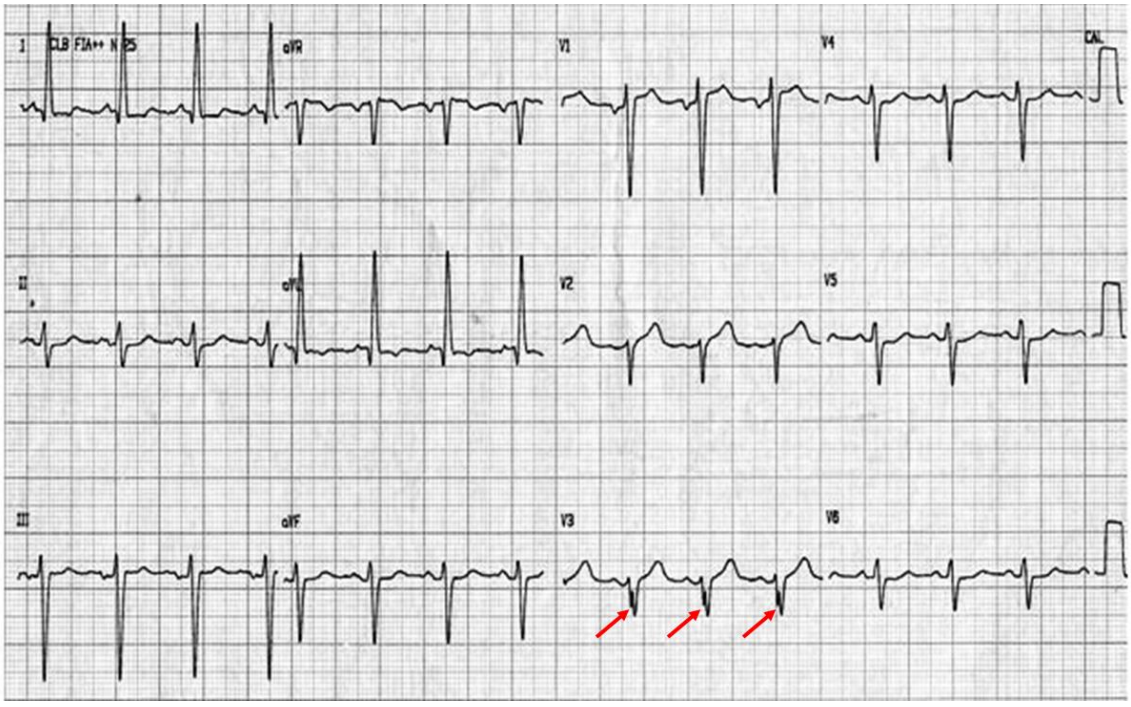
First ECG (Jan 24, 2011)



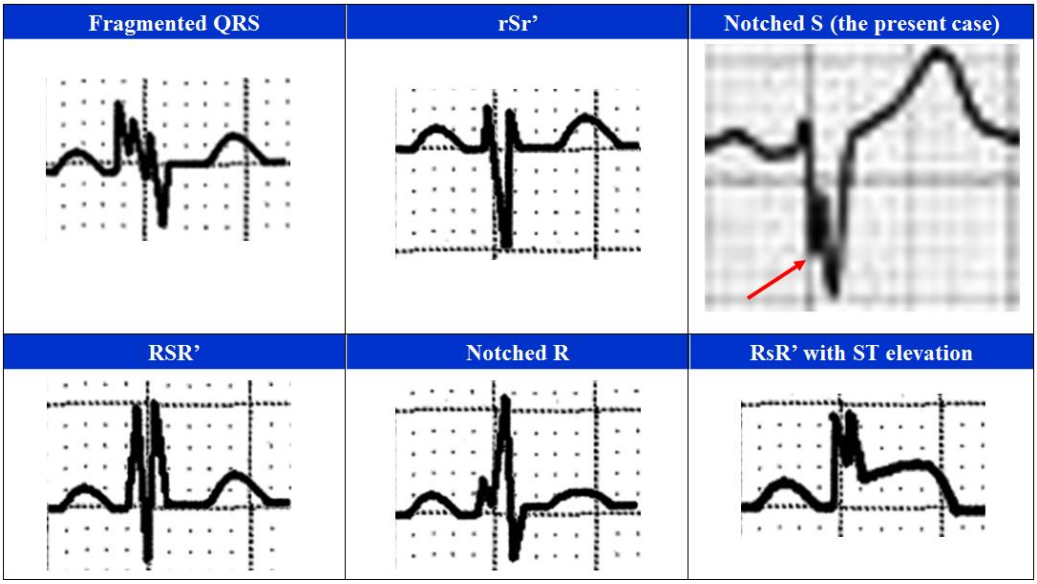
ECG diagnoses:

1) **Left atrial enlargement (LAE):** P-terminal force (PTF-V1) exceeding 0.04 mm/s. This is the terminal, negative part of the P wave in lead V1 expressed as the multiplication of its depth in millimeters and width in seconds (mm/s). The normal PTF-V1 does not exceed 0.04 s wide and 1mm deep, i.e., 0.04 mm/s.
2) **Rosembaum type IV minimal degree of Left anterior fascicular block**: (SIII > 15mm) This pattern is indicative of LAFB+ LVH. The ECG LAFB types of Rosenbaum are:
   - *LAFB type I or "Standard":* SÂQRS near –60°; q wave without s wave in I and no r' complex in II. S wave of III <15 mm. Tendency to isodiphasism in aVR. This variety of LAFB is the most frequent (50% of the cases).
   - *LAFB Type II:* horizontal heart with clockwise rotation: SÂQRS: around –60º; Voltage of QRS in the FP reduced by the posterior orientation of the final vectors; III: rSr'; I: Rs of low voltage. RS from V2 to V6.
   - *LAFB Type III:* ectomorph, vertical heart: Possible presence of "P pulmonale" with SÂP to the right of +75º, SÂQRS: beyond –60º near –90º; I: R wave of low voltage; aVR predominantly positive (axis of QRS to the right of – 60º).
   - *LAFB Type IV:* association of LAFB + LVE: Frequent presence of LAE, S III > 15 mm, inverted T wave in one or more of the left leads: I, aVL, V_5 and V_6 (in the present case in aVL). Voltage of R wave in I and increased S wave of II and S of III.
3) **LAE+ LVH**
4) **Reversal progression of R wave from V_1 to V_3:** In normal conditions, there should be a gradual increase in the amplitude of the R-wave from V_1 to V_3-V_4. This is referred to

as R-wave progression. Lead V_1 may or may not have an R wave, but one should show up by lead V_2 and get a little taller in lead V3 and reach its maximum height in lead V4 or V5. Unfortunately, this is highly dependent on lead placement.

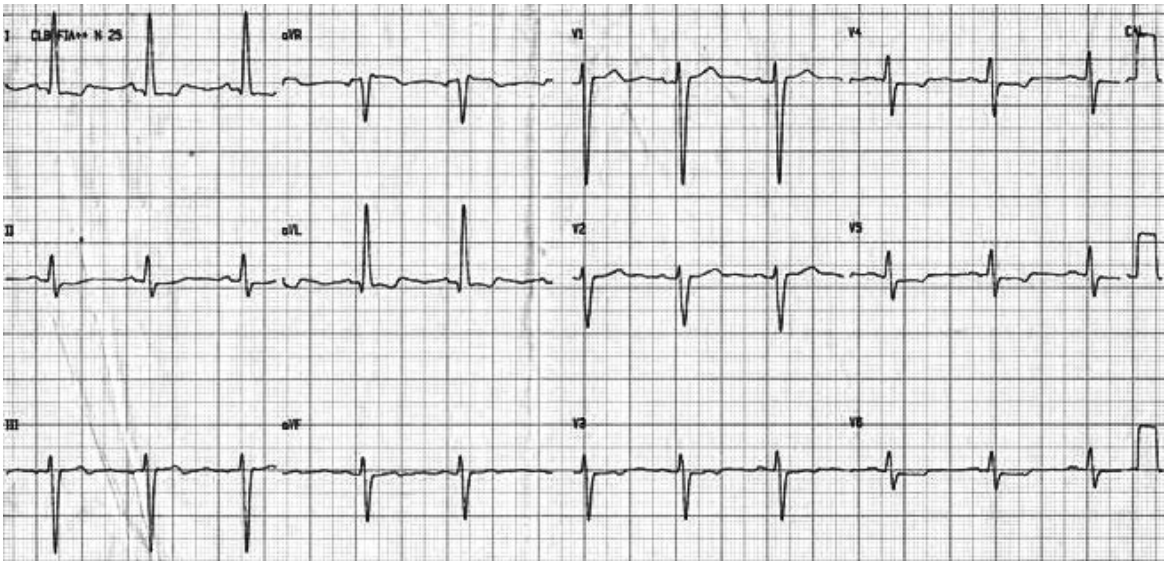
5) **Notch in the descendent ramp of V_3:** is this a fragmented QRS (fQRS)? fQRS is a marker of myocardial scar evaluated by ECG. It is defined as additional spikes within the QRS complex. In patients with CAD, fQRS is associated with myocardial scar detected by single photon emission tomography and is a predictor of cardiac events. fQRS is also a predictor of mortality and arrhythmic events in patients with reduced LVEF.

Patterns of fragmented QRS



The fQRS has QRS duration is < 120ms and several patterns which included an additional R wave (R') or notching in the nadir of the S wave, or >1 R' (fragmentation) in at least 2 contiguous leads, corresponding to a major coronary artery territory. The presence of an fQRS in 2 contiguous anterior leads (V_1 toV_5) was assigned to myocardial scar in anterior segments or in the LAD territory. The presence of an fQRS in 2 contiguous lateral leads (I, aVL, and V6) was assigned to lateral segments or LCX territory myocardial scar. Similarly, the presence of an fQRS in 2 contiguous inferior leads (II, III, and aVF) is assigned to myocardial scar in the inferior segments or in the RCA territory. In the present case does not meet the fragmented QRS criterion because the notch is observed only in one precordial lead. There is considerable overlap in the regional myocardial scar distribution, in particular, the coronary artery territory because of considerable variation in coronary anatomy and collateral circulation, the aforementioned assignments were thought to be most appropriate for clinical correlation and myocardial scar location. The fQRS) without typical BBB pattern represents remote MI in patients with known or suspected CAD. The fQRS has a substantially higher sensitivity than the Q wave. The specificity of fQRS is also comparable to that of Q wave in anterior ECG leads but is lower than that in lateral and inferior leads. Additionally, the fQRS has a higher negative predictive value for myocardial scar than does the Q wave (**Das 2006**).

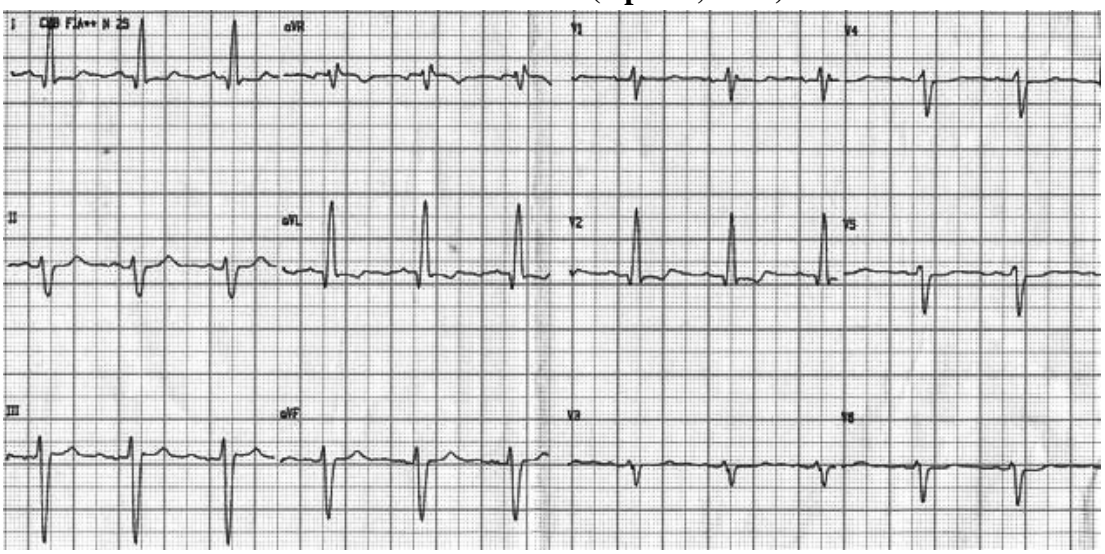
Second ECG-2 (Dec 12, 2012)



ECG diagnoses:

1. **Left ventricular hypertrophy**
2. **QRS axis** -25° II is predominantly positive: R>s: The QRS pattern in I and aVL(qR) + rS pattern in inferior leads II, III and aVL with SIII>SII would suggest a minimum degree of LAFB.
3. **Possible loss of viable myocardium (amplitudes of the QRS complexes in lateral leads are < 10mm) and ischemic or mix ST and T changes in anterolateral** leads (anterior V3 to V4 and lateral I+ aVL + V5-V6). Minimal ST depression followed by negative T waves. This lateral loss of R height suggests lateral myocardial loss from a prior lateral or anterolateral MI.

Third ECG-3 (Apr 02, 2014)



ECG diagnoses:

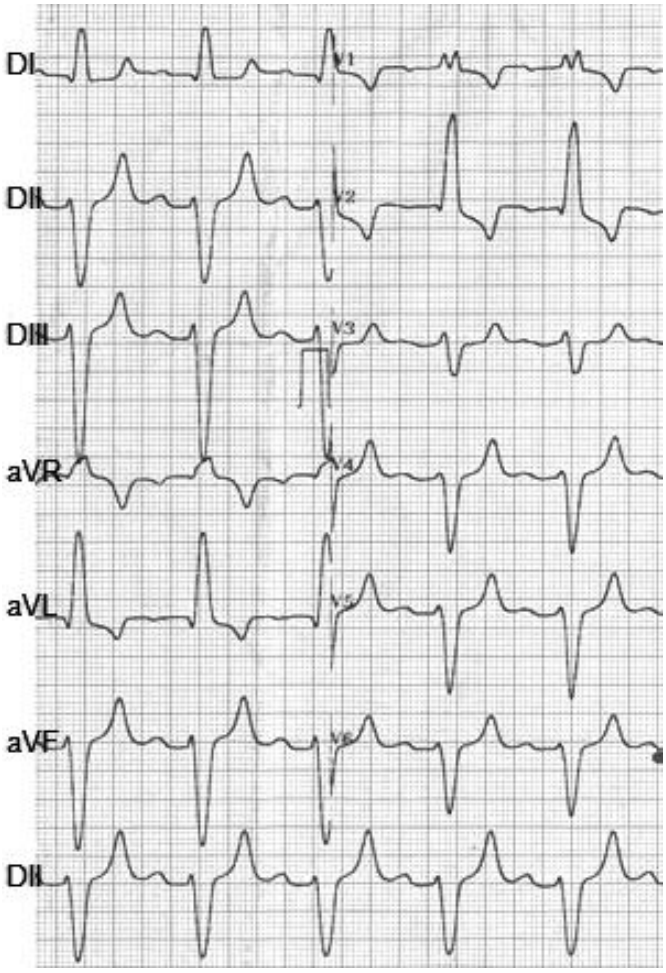
Left Anterior Fascicular Block: higher degree of LAFB: QRS axis -60°, rS pattern in inferior leads, SIII > SII, SIII > 15 mm and qR in I and aVL. Deeper S waves are recorded in leads V5 and V6 as a result of the superiorly directed forces in presence of LAFB (**Elizari 2007**). These deep S waves disappear in higher V leads, attributed to downward displacement of the electrical center of ventricular depolarization. Such us the present case frequent absence of the Q wave in leads V_5-V_6 is observed in presence of LAFB.

V1: An rSr' pattern with QRS duration <120ms in the right precordial leads can be due to incomplete right bundle branch block (which may progress to complete right bundle branch block) or can be a normal electrophysiological variant. QRS duration < 120ms with triphasic QRS pattern type rSr' in V_1 with the wave r' of lower voltage than the initial R. This pattern is possible to observe as normal variant in 2,4% in healthy individuals (**His 1962**), in wrong placement of electrode or decrease of anteroposterior diameter of chest (pectus excavatum and "straight back" syndrome). The final small r' wave have been attributed to physiologic late activation of the crista supraventricular of the right ventricular outflow tract (**Surawicz 2008**). An R' deflection in the right precordial leads V_2-V_3 is possible in presence of LAFB, especially in those recorded at a higher level, if the terminal QRS vector is directed anteriorly as well as superiorly. In these cases, it is useful to map in right precordial leads V_3R and V_4R and V_2H (high V_2). In the true LAFB or final R' wave or r' wave of V_2 is greater than the one of V_3R and V_4R, indicating that the final forces are heading predominantly to the left.

V2: Sudden **P**rominent **A**nterior QRS **F**orces(**PAF**) with small (embryonic) q wave in V2 and qRq pattern (≥15 mm voltage R waves in V_2), ventricular activation time in V2 ≥ 35 ms, followed by rS from V3 to V6 and absence of initial q wave in left precordial leads V_5, V_6: Possible LSFB associated with anterolateral inactive electrical area.

Conclusion: LAFB+ LSFB? possible **left bifascicular block**. Loss of QRS voltage from V_3 to V_6 mainly in V_3 suggesting anterolateral inactive electrical area.

Fourth ECG-4 (Dec 20, 2015)



ECG diagnoses:

I. **First degree AV block:** Prolonged PR interval (PRi = 270 ms) Incomplete Left Posterior Fascicular Block? (ILPFB)

II. **Left Anterior Fascicular Block (LAFB):** Extreme left axis deviation (QRS axis -80°), qR in I and aVL, rS pattern in III, III and aVF, SIII>SII, SIII> 15mm: Type IV Rosembaum LAFB+ LVH.

III. **Complete Right Bundle Branch Block(CRBBB)**

IV. **Left septal fascicular block(LSFB) in association?**

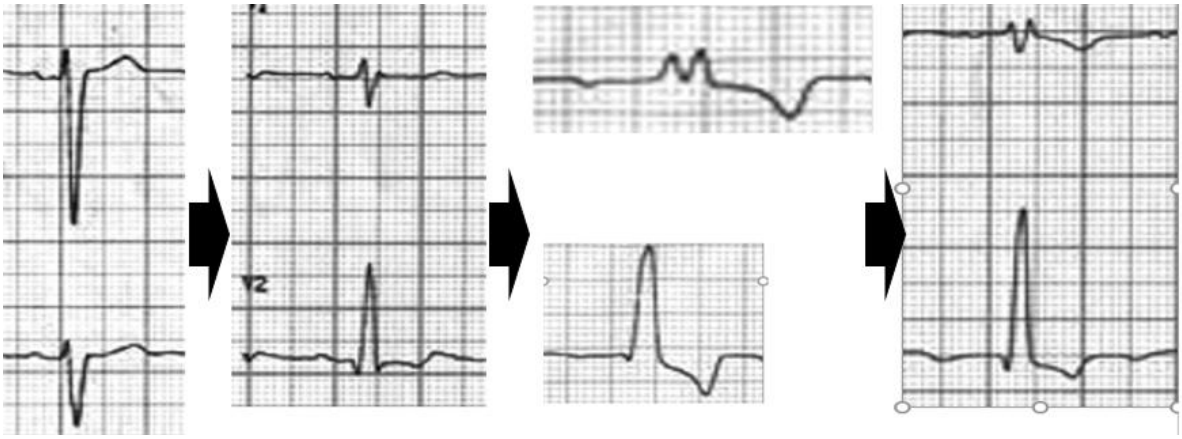
V. **Possible inactive area in lateral wall or fibrotic**

VI. **Standard Masquerading RBBB:** simultaneous presence of a high-degree LAFB, right bundle branch block often accompanied with severe left ventricular enlargement and/or fibrotic block in the anterolateral wall of the left ventricle (**Elizari 2013**).

VII. **Possible tetrafascicular block?**

Conclusion: first degree AV block (**incomplete LPFB?) + LAFB + CRBBB + LSFB+ masquerading RBBB: Tetrafascicular block** (This is new hypothetical terminology that we are proposing).

V1-V2 patterns in sequential ECG from ECG-2 to ECG-5



ECG-2 V1-V2	ECG-3 V1-V2	ECG-4 V1-V2	ECG-5 V1-V2
Normal PR Normal QRS duration and normal pattern.	Normal PR V1: rsr': normal variant? IRBBB? V2: qRs Prominent Anterior QRS Forces (PAF): LSFB?	First degree AV block: PR interval = 270ms Wide QRS: >120ms V1: RsR' V2: qR. R wave ≥ 15mm Incomplete LPFB?+ RBBB + LSFB?	First degree AV block: PR interval = 320ms V1: rsr' V2: qR Incomplete LPFB?+ IRBBB?+ LSFB?

Standard Masquerading Right Bundle-Branch Block: The ECG complex coined by the first time in 1954 by Richman (**Richman 1954**) **"masquerading bundle-branch block"** to day we know that is essentially a complete right bundle branch block(CRBBB) associated with left anterior fascicular block(LAFB), with further modifications of the initial and final QRS vectors, so that standard leads, and at times the left precordial leads, resemble left bundle branch block(LBBB). The limb leads I and aVL show a LBBB-like pattern without final S wave, but the precordial leads show a RBBB. In the "standard masquerading type" the LAFB obscured totally or partially the diagnosis of CRBBB only on frontal plane leads by abolishing (or becomes very small) the final broad S wave in the left leads I and aVL (**Ortega-Carnicer 1986**) and the precordial leads remain the typical CRBBB pattern. (**Schamroth 1975**). In most cases of RBBB and LAFB, the QRS axis deviation is located between - 80° to -120°, when predominant LV forces, the QRS axis deviation is near about -90°, turning the pattern into an atypical one (**Kukla 2014**).

In the first word masquerading BBB is seen most commonly with chronic coronary artery disease and hypertension (**Choudhary 2014**). Also in the setting of acute myocardial infarction (**Sclarovsky 1979; Dwivedi 1999**).

Since the pioneer Rosembaum´s et al studies (**Rosembaum 1968; Rosembaum 1973**) we know two masquerading ECG types:

I) The "standard type" masquerading bundle-branch block

II) The "precordial type". masquerading bundle-branch block

Several conditions are capable of producing the finding:

1) **Severe left ventricular hypertrophy**
2) **Focal blocks (infarction or fibrosis of the anterolateral wall of the left ventricle)**
3) **Peri-infarction or intra-infarction block.**
4) **Metabolic blocks when in the setting of acute MI (Sclarovsky 1979):** consequence of increased extracellular K^+ released by necrotic myocardial cells on viable conducting fibers. The distal His bundle and proximal bundle branches are more susceptible to the depolarizing effect of extracellular K^+ than the more proximal conducting tissue. The K^+ release from necrotic cells is greatest 24 hours after the AMI and the necrotic area gradually loses K^+ until the fourth day after the AMI (**Hackel 1972**).

In standard masquerading RBBB, in frontal plane there are four main developmental phases that do not necessarily occur in the chronological sequence. Table 1 shows the four main developmental ECG patters of standard masquerading RBBB in left leads and inferior leads.

Table 1

	aVL	I	II	III
1. Uncomplicated LAFB: QRS duration < 120ms	qR	qR	rS	rS SIII>SII
2. LAFB with CRBBB: QRS duration ≥120ms	qRS	qRS	rS with notch on ascending ramp of S	rS with notch on ascending ramp of S
3. LAFB with CRBBB and diminution of the final QRS vectors. QRS duration≥120ms	qR	qR	rS	rS
4. LAFB with CRBBB and diminution of the final QRS vectors and diminution the initial QRS vectors	R	R	QS	QS

The precordial type (*"precordial masquerading right bundle-branch block"*) shows the pattern of CRBBB in the right precordial leads and complete left branch block pattern (CLBBB) in the left-side precordial leads. This result from CRBBB associated with severe

left ventricular hypertrophy (LVH), a localized block in the anterolateral wall of the left ventricle often due to myocardial infarction, and usually LAFB. Presumably, the intramural left ventricular block, together with the LVH or the LAFB, or both, produce predominant leftward forces which tend to cancel out the late rightward forces of the RBBB in the left precordial leads.

Another masquerading variant in cases of with severe *chronic* fibrotic heart disease c*oncomitant standard and precordial masquerade CRBBB is possible*. In these cases, the standard left limbs I and aVL shows qR pattern with low voltage and wide QRS duration (pseudo-atypical complicated LBBB pattern) and in the precordial leads from V1 to V4 QS or Qr pattern consequences of severe anterior fibrosis that masquerade the existent CRBBB.

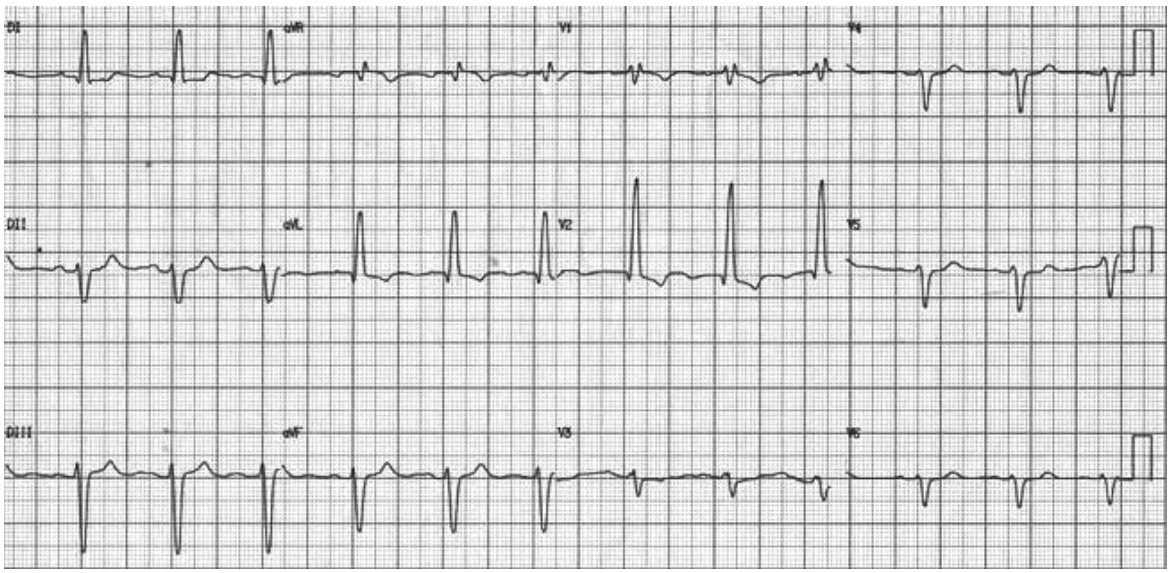
Masquerading bundle-branch block can be associated with severe and diffuse conduction system disease, and that patients with this finding may require permanent pacemaker implantation, especially if they are symptomatic (**Kowey 1989**).

Sclarovsky et al (**Sclarovsky 1979**) described 30 cases of acute myocardial infarction (AMI) in which LAFB obscured the diagnosis of RBBB, and present the clinical and electrocardiographic characteristics, evolution and immediate prognosis.

Unger et al (**Unger 1958**) studied two cases having the features of masquerading BBB were subjected to careful histologic study of the entire heart, including the conduction system. In each instance, bilateral BB lesions of considerable intensity, which did not completely disrupt the continuity of the branches, were demonstrated. In both cases revealed extensive destruction of the interventricular septum, the free walls of the LV, severe bilateral ventricular hypertrophy and diffuse arteriolosclerosis. It is suggested that this ECG complex is the result of partial bilateral BBB. It is further suggested that the concept of masquerading BBB be discarded.

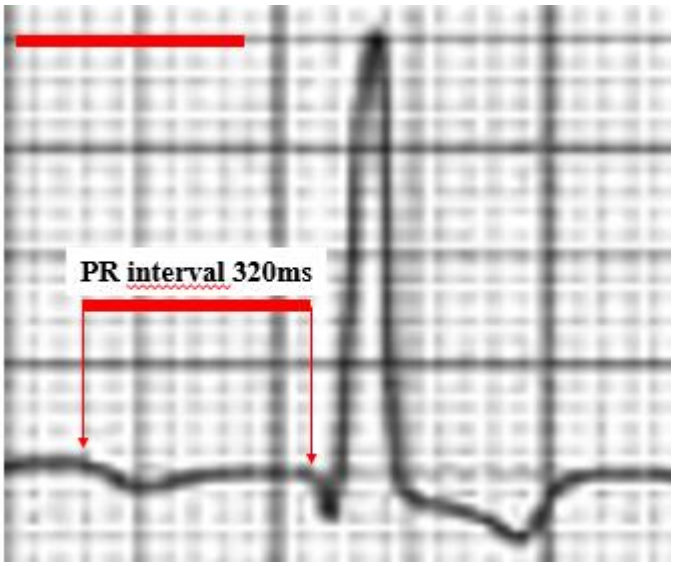
Progression to high degree atrioventricular block is common in the presence of masquerading BBB. Additionally, it is frequently associated to advanced heart failure, with consequent poor prognosis (**Gómez Barrado 1997**).

Fifth ECG-5 (Dec 23, 2015)



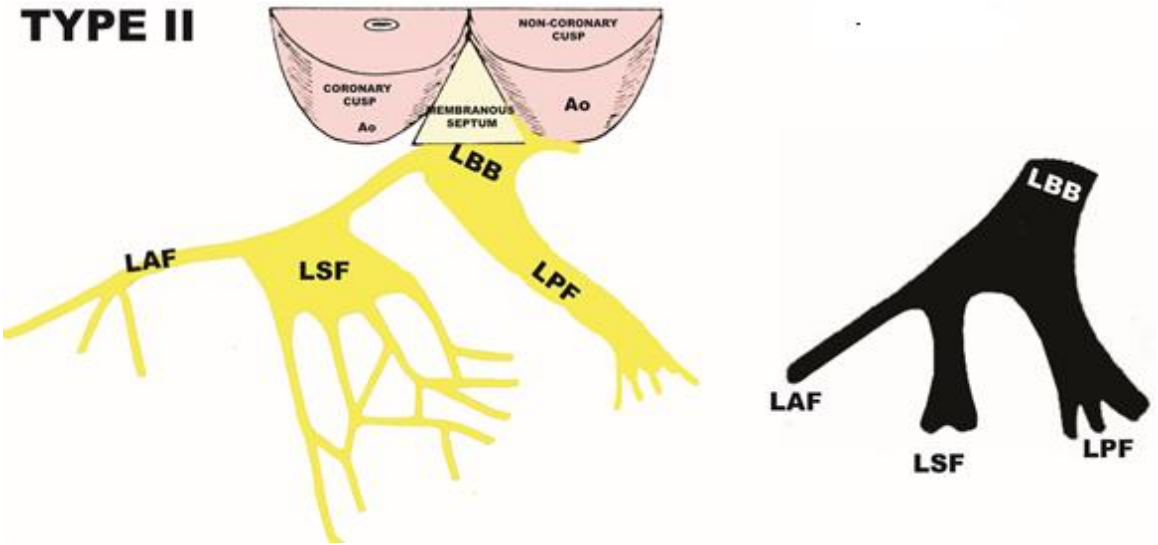
ECG diagnosis:

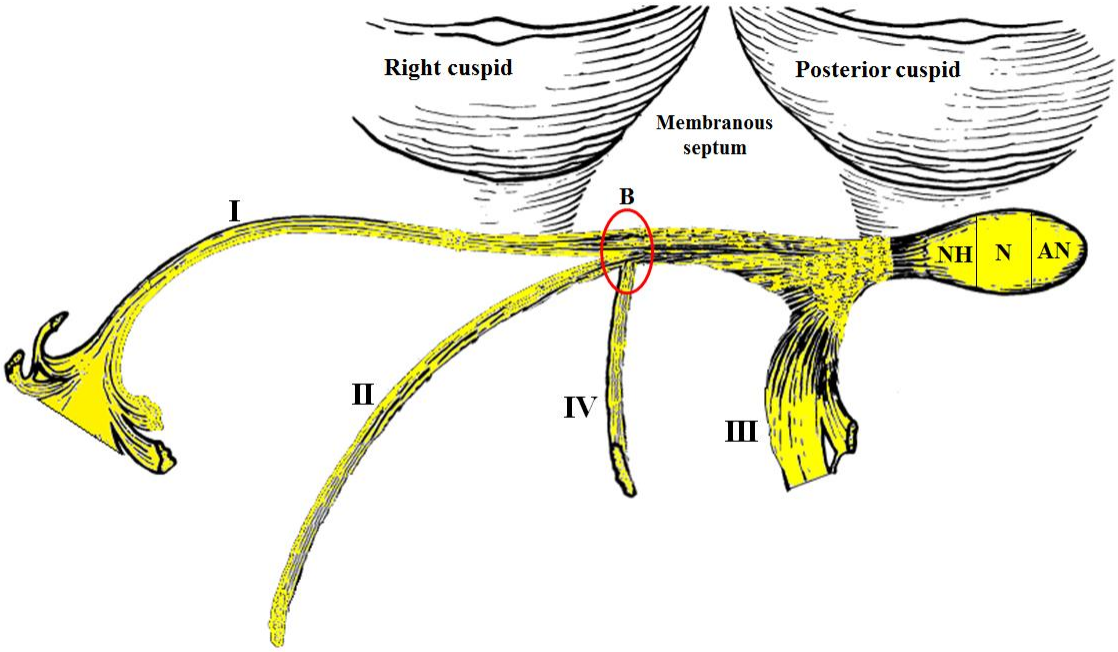
1) **First degree AV block: PR interval 320ms: incomplete LPFB?**
2) **LAFB**
3) **Incomplete RBBB? V1: rsr'**
4) **LSFB? V2: qR. R ≥ 15mm 20mm**
5) **Tetrafascicular block? Incomplete LPFB+ LAFB+ RBBB+ LSFB**



Acunzo et al (**Acunzo 2013**) presented the ECG/VCG manifestations of RBBB with LSFB with or without LAFB in premature atrial beats of patients without apparent structural heart disease. The combination of RBBB and LSFB in absence of apparent structural heart disease may be related to the simultaneous occurrence of block of conduction through these components of the Purkinje network. The left anterior fascicle of the left bundle may also be involved.

We speculate that when RBBB, LSFB and LAFB are concomitantly present with premature atrial beats, the patient probably has the type II variant





Modified from (Merideth 1973)

I. Right Bundle Branch: Length ≈ 50 mm; diameter 1.5 to 2 mm

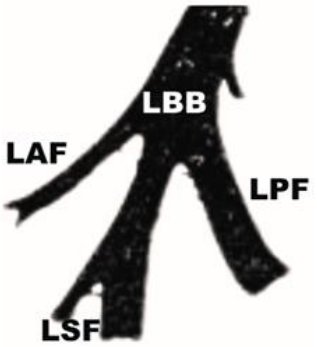
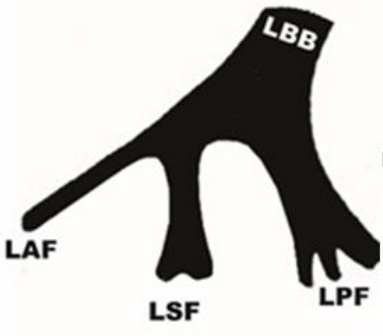
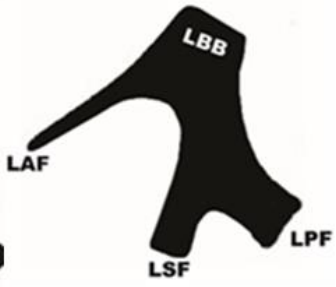
II. Left Anterior Fascicle: Length ≈ 70 mm; diameter 1 mm

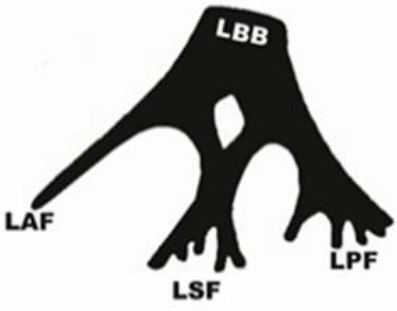
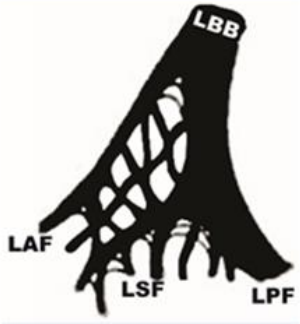
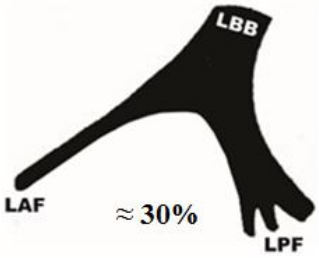
III. Left Posterior Fascicle: Length ≈ 30 mm; diameter 8 to 9 mm

IV. Left Septal Fascicle type II: origin near Left Anterior Fascicle and Right Bundle Branch

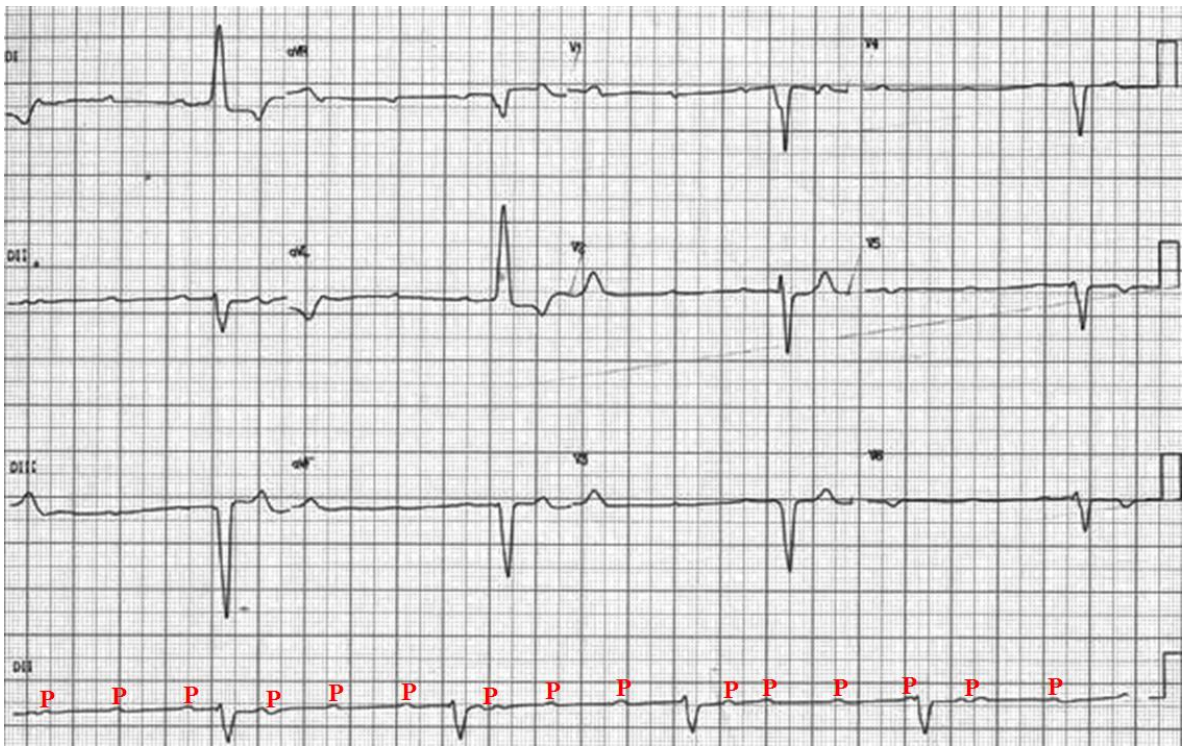
AN-N-NH: Atrial ventricular node (AVN) componentes. AVN has 7.5 mm length.

The red ellipse (B) suggests the size and location of the lesion which produces RBBB + LAFB + LPFB in type II variant.

Type I	Type II	Type III
		

Type IV	Type V	Type VI
		

Sixth ECG-6 (Dec. 29, 2015)



ECG diagnoses: Advanced degree of heart block or high grade AV block. Atrial tachycardia with 3:1 AV block (P rate ≈100 bpm) and narrow escape rhythm with minimal variable conduction from junction area (heart rate 24 bpm). High grade AV block (a.k.a. advanced heart block) is a form of third degree heart block. This occurs when AV dissociation is present, however intermittently some sinus node action potentials (P waves) are randomly conducted to the ventricles. This is different than "complete heart block" or third-degree AV block (another form of third degree heart block) where no sinus node activity conducts through the AV node to the ventricles at all. In most cases of complete AV block, an escape rhythm originates from the ventricles, with wide QRS complexes at a low regular rate of 30-40 beats/min. A higher anatomic location of the block results in a higher location of the escape rhythm pacemaker, a faster escape rhythm (40-60 beats/min in the region of NH or His bundle), and a narrower QRS duration such as the present case. But the HR is very low.
The AVN is supplied by the RCA (90%) or by the LCX (10%) and is innervated by both sympathetic and parasympathetic fibers. It receives impulses anteriorly via the intra-atrial fibers in the septum and posteriorly via the crista terminalis. Impulses arriving at the AVN are transmitted to the ventricle in a 1:1 ratio. As faster impulses arrive, the conduction to the ventricles slows; this is called decremental conduction.
The His-Purkinje system is composed of 2 bundles of Purkinje fibers (the LBB and RBB) that conduct electrical impulses to allow rapid ventricular activation. The His-Purkinje system is yet another location where AV block may occur.
AV block may be caused by acute myocardial ischemia or infarction. Inferior myocardial infarction may lead to third-degree block, usually at the AVN level; this may occur through

other mechanisms via the Bezold-Jarisch reflex. Anterior MI usually is associated with third-degree block resulting from ischemia or infarction of bundle branches.

First-degree AV block and second-degree Mobitz I AV block usually involve a delay at the level of the AVN, whereas second-degree Mobitz II AV block generally involves blockage in the His bundle or lower regions of the conduction system. Third-degree AV block involves conduction disturbances in the AV node or the His-Purkinje system.

In CAD patients it occurs most frequently in CAD when the infarction is of the inferior wall and results from interference to the blood supply of the AV node or upper part of the bundle of His. The blood supply to this region is derived as a rule from the RCA, and it is occlusion of this artery that is responsible for 90% of inferior MIs. In the remaining 10%, it is occlusion of the LCX that is responsible. The artery to the AV node (sometimes more than one), named the ramus septi fibrosi by Gross (**Gross 1921**), is given off at the posterior junction of the interatrial and interventricular septa at the crux of the heart. The AV nodal artery originates from the artery that crosses the crux of the heart, whether it be the right or left coronary. The AV nodal artery passes anteriorly from its origin, travels deep to the coronary sinus and rises deep to the base of the interatrial septum. James and associates (**James 1965; 1968**) in their studies of the atrial coronary arteries, demonstrated that the AV nodal artery arises from the RCA in 83 % of cases, from the LCX artery in 7%, and from both left and right coronary arteries in 10%. Gross (**Gross 1921**) demonstrated anastomoses between the AV nodal artery and other atrial arteries. The primary blood supply to the region of the AV node is the RCA in about 92 % of cases. This region is likely to be compromised with RCA occlusion. It may be that diseases of the other atrial arteries may contribute to or account for some of the disorders in function of the AV node or upper part of the bundle of His. That conduction disturbances are seen in a minority of cases of inferior MI is surprising. The site of thrombosis in the coronary arteries is, in 84% of cases, in the proximal portion of the artery (**Wartman 1948**) and proximal to origin of the artery to the AV node. More frequent disorders in function of the AV node might be anticipated. However, in the anatomic study by Blondeau and associates (**Blondeau 1961; 1961**) of 17 cases of MI with heart block the region of the node and upper part of the bundle of His was found to be the site of massive necrosis only when there was a thrombosis of the artery to the node itself. This was present in two of 17 cases. In the other 15 cases there were spotty degenerative lesions in this region. The infarction of the postero (infero) septal region constantly present in inferior infarctions did not extend beyond the AV fibrous ring to the AV node. Pre-his develops during AMI is not a threat to life. Master reported (**Master 1938**) that simple prolongation of the PR interval exerted no adverse effect on the clinical course or prognosis of AMI. More advanced degrees of AV block carry a graver prognosis. Prognosis for Recovery from Block. In this series no patient who recovered had a residual AV conduction disturbance. That the conduction disturbance is usually transient may be explained by the resolution of inflammatory and congestive changes in the region of the AV node when anastomotic channels become effective. Anoxemia is another factor that may account for the transient nature of the block.

In clinical studies, Greene and Gilbert (**Greene 1921**) demonstrated that the AV node is very sensitive to oxygen lack.

Notion of coronary circulation in the heart and the intraventricular conduction system

There are many factors which influence the clinical course in a patient with acute myocardial infarction (AMI), but one of the most important ones is anatomic location. To understand the consequences of an occlusion of a major branch of the coronary tree, one must visualize exactly what is downstream from the point of occlusion. The following discussion will stress the concept of considering all possible anatomic structures of critical functional importance which may be located downstream from a point of coronary occlusion. However, since most cardiologists think of an infarct in terms of its general location rather than in terms of the coronary artery occluded, the discussion will be oriented to topography of the MI, and the pertinent anatomy of the coronary arteries and conduction system introduced on that basis.

Left anterior descending artery (LAD) (also ***LAD***, anterior interventricular branch of the left coronary ***artery***, anterior descending branch ***LAD***, anterior interventricular branch of the left coronary ***artery***, or anterior descending branch),

After originating from the left main coronary artery (LMCA), the left anterior descending artery (LAD) runs along the anterior interventricular sulcus and supplies the apical portion of both ventricles. The LAD artery is mostly epicardial but can be intramuscular in places. The LAD gives off two types of branches: *septal perforating* and *diagonal arteries*.

- Septal perforating branches originate from the LAD at 90 degrees to the surface of the heart, perforating and supplying the anterior $^{2}/_{3}$ of the IVS. An important identifying characteristic of the LAD artery is the identification of 4-6 perpendicular septal perforating branches (S_1 to S_4- S_6). These branches are approximately 7.5 cm in length. The septal branches of the LAD supply two-thirds of the superior portion of the interventricular septum (IVS), while the inferior portion of the septum is supplied by septal branches of the posterior descending coronary artery (PDA), which usually arises from the right coronary artery (RCA) and infrequently from the left circumflex artery (LCX). The IVS, which constitutes about one-third of the mass of the left ventricle, is a common wall for both the left and right ventricles and is a vital component to left and right ventricular function. The IVS is the most densely vascularized portion of the heart (**Levin 1988**), and is perfused mainly by anterior and posterior IVS branches arising from the coronary arteries (**Topaz 1996**). IVS plays an essential role in ventricular function since it contains important elements of the cardiac conduction system and comprises a large portion of the myocardium (**James 1958; Vemuri 1993**). Anterior interventricular septal perforating branches emerging from the proximal segments of the LAD also supply blood to the septomarginal trabecula, which bears the distal portion of the right branch of the atrioventricular bundle. Septal perforating arteries are an important source of collateral blood supply between the RCA and the left system (**Cohen 1999**). The first septal perforating branch (S_1) also supplies a significant portion of the conduction

system, including the His bundle and the AV node in 50% of patients (**Ozdemir 2001**). The size and anatomy of septal perforating branches vary widely. While patients usually have several small-caliber septal branches that are equal in size, a large S_1 may be found in approximately 15–30% of patients. A large S_1 may have obstructive disease or even coronary artery vasospasm, resulting in angina and significant clinical ischemia. Infarction of the IVS is usually due to LAD obstruction, but it can rarely be a product of a discrete stenosis in a large S_1. Complete occlusion of a large S_1 may present with angina and ST-segment elevation in the anteroseptal wall. It can also present with a RBBB and LAFB. In a normal person, the septal perforating branches of the LAD are located closer to the right ventricular endocardium. This is the reason why septal infarction following alcohol injection in severe O-HCM caused a predominance of complete RBBB, different from surgery of myotomy/myectomy wich causes LBBB (**Riera 2002**). During the surgical procedure, the surgeon removes a small portion of the hypertrophic basal left septum, where the trunk of the LBB, and the onset of its fascicles or divisions run. This explains the pattern of CLBBB observed in a high percentage of cases after this procedure. On the contrary, the percutaneous procedure causes necrosis with a basal transmural location, and somewhat lower, that extends to the right septal surface where the RBB is located, which explains why CRBBB is the rule after this procedure (**Pérez-Riera 2013**).

- These branches are frequently ignored as potential targets for revascularization secondary to the fact that they are surgically inaccessible, usually small in caliber (1.5–2.0 mm in diameter), and have an acutely angulated vessel origin. Septal perforating arteries are also associated with high restenosis rates after angioplasty due to their increased elasticity, and septal branch disease usually involves the ostium and functions as a branch ostial stenosis, which limits the role for stenting and prevents the delivery of bulky devices (**Ozdemir 2001**). We could not define the exact location of the septal perforating arteries in each patient from the study of the coronary arteriograms. A decrease in the intraluminal pressure with respect to the intramyocardial pressure and increased septal thickness, facilitate the appearance of septal perforating compression. This theory explains the septal perforating compression in obstructive hypertrophic cardiomyopathy (O-HCM), aortic stenosis, severe stenosis of LAD, cardiomyopathy and myocardial bridge and may predict the occurrence of this phenomenon in other conditions with thickened septum or leading to a decrease of the intraluminal pressure of the septal perforating arteries with respect to the intramyocardial pressure. It is interesting to speculate on the effects of the apparent reduction of coronary flow due to septal perforating compression. In the absence of a clinicopathologic correlation, we do not know the relationship of the angiographic finding to the fibrosis of the IVS that has been described in O-HCM. However, we did not observe an association of septal perforating compression and angina in patients with aortic stenosis or O-HCM and normal coronary arteries. Carroll and Falsetti did not observe a correlation between angina and retrograde

coronary flow in patients with aortic valve disease. These investigators attributed the retrograde flow to compression of the intramyocardial coronary arteries (**Carroll 1976**).

- Diagonals run along the surface of the heart and supply the lateral wall of the LV and the ALPM.

In the IVS there are two areas with different blood supplies:

I) *The upper portion, which includes the AV node, the atrioventricular bundle and the proximal segments of the two main bundle branches, that are supplied by a branch of the RCA. The S_1 also supplies a significant portion of the conduction system, including the His bundle, RBB and the AV node in 50% of patients (**Ozdemir 2001**).*

II) *The lower area, which comprises the greater mass of the septum, including most of the two main bundle branches, left fascicles, and the Purkinje arborization of the IVS, supplied mainly by the anterior septal perforating branches.*

Based on these findings, James and Burch stated that occlusion of the LAD may produce disturbance in heart conduction, depending upon the efficiency and extent of collateral circulation. The S_1 of the LAD artery is termed the ramus intermedius. As the LAD artery passes along the anterior interventricular groove toward the apex, it turns sharply to anastomose with the posterior interventricular branch of the RCA. As the LAD artery courses anteriorly along the ventricular septum, it sends off diagonal branches to the lateral wall of the LV. Azuma et al, (**Azuma 1994**) demonstrated that complete occlusion of the S_1 was associated with RBBB on ECG. Sigwart (**Sigwart 1995**) developed a catheter-based technique as a therapeutic option for severe obstructive hypertrophic cardiomyopathy (O-HCM) that is non-responsive to drug by injecting absolute ethanol into the first major S_1 to induce an artificial localized septal infarct. Airoldi et al (**Airoldi 2000**), reported that the treatment of O-HCM by catheter intervention was technically successful, but was also associated with a high incidence of RBBB, fascicular blocks and rarely complete AV block. These recent clinical findings and the possible complications stimulated our interest in a more detailed study of the anterior interventricular septal branch associated with the septomarginal trabecula, as part of an investigation to correlate the morphological and anatomical features with the images obtained from coronary catheterization.

Left circumflex artery

The LCA gives off the LCX artery at a right angle near the base of the left atrial appendage. The LCX artery courses in the coronary groove around the left border of the heart to the posterior surface of the heart to anastomose to the end of the RCA. In the AV groove, the LCX artery lies close to the annulus of the mitral valve. The atrial circumflex artery, the first branch off the LCX artery, supplies the left atrium. The LCX artery gives off an obtuse marginal (OM) branch at the left border of the heart near the base of the left atrial appendage to supply the posterolateral surface of the LV. In patients with a left-dominant heart, the LCX artery supplies the PDA. In fewer than 40% of patients, the sinus node artery may originate from the LCX artery.

Right coronary artery

The RCA is a single large artery that courses along the right AV groove. The RCA supplies the right atrium, right ventricle, IVS, and the SA and AV nodes. The RCA arises from the right aortic sinus and courses in the coronary (AV) groove between the right atrium and the right ventricle. In 60% of patients, the first branch of the RCA is the sinus node artery. As the RCA passes toward the inferior border of the heart, it gives off a right marginal branch that supplies the apex of the heart. After this branching, the RCA turns left to enter the posterior interventricular groove to give off the PDA, which supplies both ventricles. The AV node artery arises from the "U-turn" of the RCA at the crux (i.e., the junction of the AV septum with the AV groove). At this point, the PDA feeds the septal, right ventricular, and left ventricular branches. The PDA courses over the ventricular septum on the diaphragmatic surface of the heart. Unlike the septal branches off the LAD artery, the septal branches from the RCA typically are short (< 1.5 cm). Terminal branches of the RCA supply the PMPM of the mitral valve. (The LAD artery supplies the ALPM.) Near the apex, the PDA anastomoses with the anterior interventricular branch of the LCA.

His bundle and bundle branches

The AV node continues onto the His bundle via a course inferior to the commissure between the septal and anterior leaflets of the tricuspid valve. The bundle follows a course along the inferior border of the membranous septum and, near the aortic valve, gives off fibers that form the LBB. The bundle is located on the left side of the IVS in 80% of patients. In 20% of patients in whom the bundle is on the right side of the IVS, the His bundle is connected to the LBB by a narrow stem.

The bundle of His is a structure that connects with the distal part of the compact AV node, perforates the central fibrous body, and continues through the annulus fibrosus, where it is called the nonbranching portion as it penetrates the membranous septum. Connective tissue of the central fibrous body and membranous septum encloses the penetrating portion of the AV bundle, which may send out extensions into the central fibrous body. Proximal cells of the penetrating portion are heterogeneous and resemble those of the compact AV node; distal cells are similar to cells in the proximal bundle branches.

Branches from LAD supply the upper muscular IVS with blood, which makes the conduction system at this site more impervious to the ischemic damage, unless the ischemia is extensive (**Ellenbogen 2008**).

The LBB further subdivides into several smaller branches that begin at the ventricular septal surface and radiate around the LV. The RBB originates from the His bundle near the membranous septum and courses along the right ventricular septal surface, passing toward the base of the right anterior papillary muscle of the tricuspid valve.

Bundle branches

The bundle branches originate at the superior margin of the muscular interventricular septum, immediately below the membranous septum, with the cells of the LBB cascading downward as a continuous sheet onto the septum beneath the noncoronary aortic cusp. The RBB continues intramyocardially as an unbranched extension of the AV bundle down the right side of the IVS to the apex of the right ventricle and base of the anterior papillary muscle. The anatomy of the LBB system may be variable and may not conform to a constant bifascicular division. However, for clinical purposes and ECG, the concept of a trifascicular system is a simplification because there are strong arguments of the existence of the LSF. Irwin Hoffman from the Christus St Vincent Regional Medical Center, Santa Fe, USA (**Hoffman 2012**) in an editorial concluded that the true incidence of dysfunction in this middle division of the LBB, whether it arises separately as a true "third fascicle"—or from the inferior or posterior fascicle — remains unknown. Although infrequent, it may be the initial ECG evidence for serious and potentially lethal left anterior descending coronary disease. An appropriate name needed. Some possibilities are "middle fibers/septal fascicle block," "anterior conduction delay," or "left septal fascicular block".

The majority of blood supply to the human His bundle and its proximal branches is of dual origin, with anastomosis principally within the His bundle.

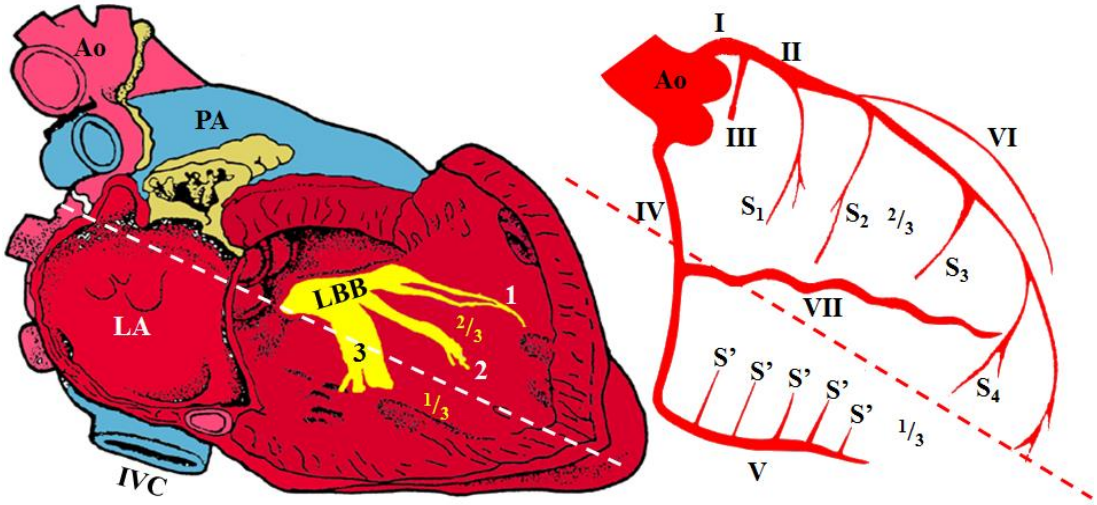
1) **His bundle blood supply** is supplied by the AV node artery from the right coronary artery (RCA) and the first septal branch of the left anterior descending artery (LAD) in 90% of cases, and entirely by the AV node artery in 10% of cases.
2) **Proximal right bundle branch (RBB) blood supply** comes from both the AV node artery and the septal branch in 50% of cases, from the septal branch alone in 40% of cases, and from the AV node artery alone in 10% of cases.
3) **Left bundle branch (LBB) blood supply** comes from the AV node artery (ramus septi fibrosi) from the right coronary artery (RCA) in 90% of the cases, and ramus septi ventriculorum superior and ramus critae along with branches from LAD (ramus limbi sinistri) in the remaining cases (**Rosenbaum 1968**).

Left fascicles blood supply

- **Left anterosuperior fascicle (LAF):** the blood supply to the LAF of the LBB originated in 50% of the cases not only from the anterior septal branch of the LAD, but also from the atrioventricular (AV) nodal artery, a branch of the RCA in 90% of the cases and of the LCX in 10% (**Frink 1973**). Thus, anatomic data support the observation that occlusion of the proximal segment of the LAD is not a prerequisite for the occurrence of LAFB. The appearance of LAFB during AMI is not a sign of a coexistent significant stenosis of the LAD or of more severe or extensive coronary artery disease. In these patients, other mechanisms such as the degree of the coronary collateral circulation may play a role in the occurrence of this conduction disturbance and supports the experimental and clinical reports that LAFB may be due to lesions

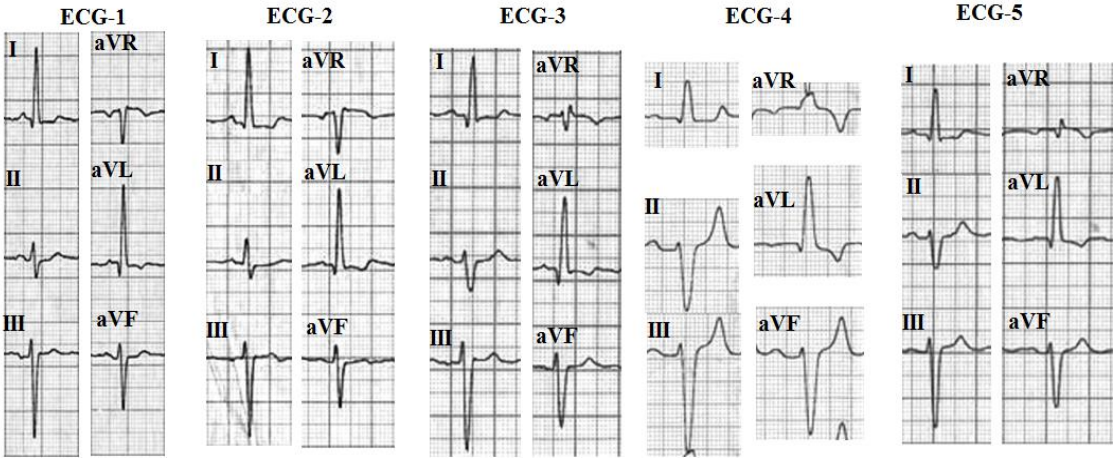
involving the His bundle by means of a longitudinal dissociation of this structure (**Bosch 1985**).

- **Left posteroinferior fascicle (LPF):** The broad nature of the LPI, its protected location in the left ventricular inflow tract as well as its dual blood supply (**James 1965**) makes isolated LPFB very rare (**Rokey 1984**). The posteromedial papillary muscle where LPF ends is supplied by those arteries that terminate on the diaphragmatic surface of the LV, and most commonly by a junction of terminal branches of the LCX and of the RCA. When the LCX supplies nearly all the diaphragmatic surface of the LV (10% of human hearts), its branches provide the entire blood supply for the posteromedial papillary muscle. The LPF is irrigated in 10% of cases by LAD only, in 40% of cases by LAD and RCA and in 50% of cases by RCA only.
- **Left septal fascicle (LSF):** It is irrigated exclusively by the septal perforating artery from the LAD. Septal perforating branches from the LAD supply the upper $^{2}/_{3}$ superior portion of IVS at this site. Most of the blood supply to the IVS is provided by the LAD. Branches into the septum from PDA rarely penetrate more than 10 mm from the epicardium (slightly more than the normal thickness of the free wall of the left ventricle), so that for practical purposes one may consider the entire blood supply of the IVS to be derived from four to six nearly equal size septal perforating branches of the LAD.



Comparative study of the first five ECGs in frontal and horizontal planes

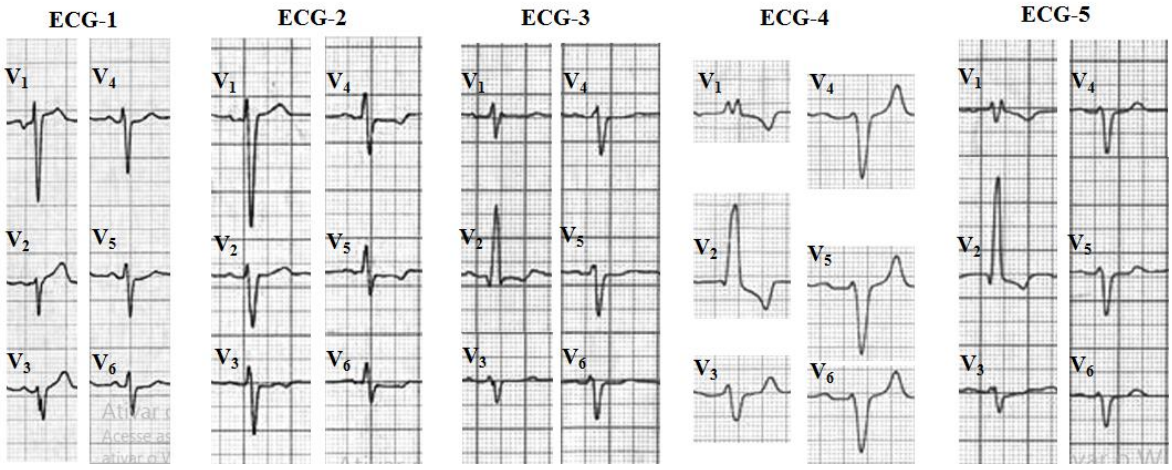
Frontal plane



	ECG1	ECG2	ECG3	ECG4	ECG5
PR interval	Normal	Normal	Normal	Prolonged	Very prolongued
QRS axis	-30° borderline	-25° Normal	- 60° Extreme LAD	-80° Extreme LAD	- 65° Extreme LAD
I-aVL	qR	qR	qR	qR	qR
II-III-aVF	rS SIII>SII	rS SIII>SII	rS SIII>SII	rS SIII>SII	rS SIII>SII

LAD: Left Axis Deviation.

Horizontal plane



	ECG1	ECG2	ECG3	ECG4	ECG5
V1	rS	rS	rSr’	rr’	rSr’
V2	rS	rS	qRs	qR	qR
V3	rS with notch	rS	rS	rS	rS
V4	rS	rS	rS	rS	rS
V5	rS	rs	rS	rS	rS
V6	rS	rs	rS	rS	rS
QRSd	<120ms	<120ms	<120ms	>120ms	<120ms

References

1. Acunzo RS, Konopka IV, Sanchéz RA, Pizzarelli N, Wells FC, Baranchuk A, Chiale PA. Right bundle branch block and middle septal fiber block with or without left anterior fascicular block manifested as aberrant conduction in apparent healthy individuals: Electro-vectorcardiographic characterization. J Electrocardiol. 2013;46(2):167-72.
2. Airoldi F, Di Mario C, Catanoso A, et al. Progressive decrease of outflow gradient and septum thickness after percutaneous alcoholization of the interventricular septum in hypertrophic obstructive cardiomyopathy. Ital. Heart J. 2000;1, 200-6.
3. Azuma T, Maeda K, Akagi H, et al. Rest angina induced by coronary artery spasm at the first septal artery: a case report. J. Cardiol. 1994; 24, 161-5.
4. Blondeau M, Rizzon P, Lenegre J. Les troubles de la conduction auriculo-ven triculaire dans 1'infaretus myocardique recent. I. Etude elinique. Arch. mal. coeur 54: 1092, 1961.
5. BlondeauM, Rizzon P, Lenegre J.: Les troubles de la conduction auriculo-ven triculaire dans 1'infaretus myocardique: Les troubles de la conduction auriculo-venitriculaire danis l 'infaretus myocardique recent. II. Etude anatomique. Arch. mal. coeur 54: 1104, 1961.
6. Bosch X, Théroux P, Roy D, et al. Coronary angiographic significance of left anterior fascicular block during acute myocardial infarction. J Am Coll Cardiol. 1985;5(1):9-15.
7. Carroll RJ, Falsetti HL: Retrograde coronary artery flow in aortic valve disease. Circulation. 1976;54(3):494-9.
8. Choudhary , Namboodiri N, Tharakan JA. A case of 'Masquerading' bundle branch block: a forgotten concept. Indian Heart J. 2014;66(1):139-40.
9. Cohen ID, Khosla S, Levin TN, et al. Rotational atherectomy for left anterior descending artery septal perforator stenosis. Catheter Cardiovasc Interv 1999;46:79–82.
10. Das MK, Khan B, Jacob S, Kumar A, Mahenthiran J. Significance of a fragmented QRS complex versus a Q wave in patients with coronary artery disease. Circulation. 2006;113(21):2495-501.
11. Dwivedi S, Suresh K. Acute myocardial infarction presenting as masquerading bundle branch block. J Assoc Physicians India. 1999;47(8):838-9.
12. Elizari MV, Baranchuk A, Chiale PA. Masquerading bundle branch block: a variety of right bundle branch block with left anterior fascicular block Expert Rev Cardiovasc Ther. 2013;11(1):69-75.
13. Ellenbogen KA, Wood MA, eds. *Cardiac Pacing and ICDs*. 5th ed. Hoboken, NJ: Blackwell Publishing; 2008.
14. Frink RJ, James TN. The Normal blood supply to the human His bundle and proximal branches Circulation 1973;47:8-18.

15. Greene CW, Gilbert, NC.: Studies of the responses of the circulation to low oxygen tension. III. Changes in the pacemaker and in conduction during extreme oxygen want as shown in the human electrocardiogram. Arch. Int. Med. 1921;27:517.
16. Gómez Barrado JJ, Turégano Albarrán S, García Rubira JC, Hidalgo Urbano R, Pavón García M, Berjillos Cordón M, Cruz Fernández JM. Clinical and electrocardiographic characteristics of masquerading bifascicular block Rev Esp Cardiol. 1997;50(2):92-7.
17. Gross L.: The Blood Supply to the Heart. New York, Paul B. Hoeber, 1921.
18. Hackel DB, Wagner G, Ratliff NB, Cies A, Estes EH Jr. Anatomic studies of the cardiac conducting system in acute myocardial infarction. Am Heart J. 1972;83(1):77-81.
19. His RG, Lamb LE, Electrocardiographic findings in 122,043 individuals. Circulation. 1962;25:947-61.
20. Hoffman I. Quadrifascicular? J Electrocardiol. 2012;45(5):536-8.
21. James TN, Burch GE. Blood supply of the human interventricular septum. Circulation 1958;17:391-6.
22. James TN. Anatomy of the coronary arteries in health and disease. Circulation. 1965;32(6):1020-33.
23. James TN. The coronary circulation and conduction system in acute myocardial infarction.Prog Cardiovasc Dis. 1968;10(5):410-49.
24. Kowey PR, Koslow M, Marinchak RA Masquerading Bundel-branch block – Electrophysiological correlation J Electrophysiol. 1989;3:156-9.
25. Kukla P1, Baranchuk A, Jastrzębski M, Bryniarski L. Masquerading bundle branch block. Kardiol Pol. 2014;72(1):67-9.
26. Levin DC, Gardiner Jr GA. Coronary arteriography In: Heart Disease: a Textbook of Cardiovascular Medicine. (Braunwald E, ed). W.B. Saunders: Philadelphia; 1988. p. 268- 310.
27. Master AM, Dack S, Jafpe HL.: Partial and complete heart block in acute coronary artery occlusion. Am. J. M. Se. 1938;196:513.
28. Merideth J, Pruitt RD. Cardiac arrhythmias. 5. Disturbances in cardiac conduction and their management. Circulation. 1973;47(5):1098-107.
29. Ortega-Carnicer J, Malillos M, Muñoz L, Rodriguez-Garcia J. Left anterior hemiblock masking the diagnosis of right bundle branch block. J Electrocardiol. 1986;19:97-8.
30. Ozdemir M, Timurkaynak T, Cemri M, et al. Stenting of the septal perforator coronary artery. J Invasive Cardiol 2001;13:694–7.
31. Pérez-Riera AR, de Lucca AA, Barbosa-Barros R, et al. Value of electro-vectorcardiogram in hypertrophic cardiomyopathy. Ann Noninvasive Electrocardiol. 2013;18(4):311-26.
32. Richman JL, Wolff L. Left bundle branch block masquerading as right bundle branch block. Am Heart J. 1954;47 383-93.

33. Riera AR, de Cano SJ, Cano MN. Vector electrocardiographic alterations after percutaneous septal ablation in obstructive hypertrophic cardiomyopathy. Possible anatomic causes. Arq Bras Cardiol. 2002;79(5):466-75.
34. Rokey R, Chahine RA. Isolated left posterior fascicular block associated with acquired ventricular septal Defect. Clinical Cardiology 1984;7(6):364–9.
35. Rosembaum MB, Elizari MV, Lazzari JO. Los hemibloqueos. Buenos Aires; Paidos 1968.
36. Rosenbaum MB, Yesuron J, Lazzari JO, Elizari MV. Left anterior hemiblock obscuring the diagnosis of right bundle branch block.Circulation. 1973;48:298-303.
37. Schamroth L, Dekock J. The concept of 'masquerading' bundle-branch block. S Afr Med J. 1975;49: 399-400.
38. Sclarovsky S, Lewin RF, Strasberg B, Agmon J. Left anterior hemiblock obscuring the diagnosis of right bundle branch block in acute myocardial infarction. Circulation. 1979;60:26-32.
39. Sigwart U. Non-surgical myocardial reduction for hypertrophic obstructive cardiomyopathy. Lancet 1995;346,211-4.
40. Surawicz B, Knilans T. Chou's Electrocardiography in Clinical Practice Adult and Pediatric sixth edition (Hardcover) by Borys Surawicz MD MACC, Timothy Knilans MD Saunders Elservier 2008.
41. Topaz O, Vetrovec GW. Anomalous first septal perforator artery: anatomic-clinical correlates. Clin. Anat. 1996;9,14-8.
42. Unger PN, Lesser ME, Kugel VH, Lev M. The concept of masquerading bundle-branch block; an electrocardiographic-pathologic correlation. Circulation. 1958;17(3):397-409.
43. Vemuri DN, Kochar GS, Maniet AR, et al. Angioplasty of the septal perforators: acute outcome and long-term clinical efficacy. Am. Heart J. 1993;125,682-6.
44. Wartman WB, Hellerstein HK.: The incidence of heart disease in 2000 consecutive autopsies. Ann. Int. Med. 1948;28:41.
45. White PD.: Heart Disease. New York, Macmillan Company, 1951.