58-year-old man carrier of revascularized coronary artery disease + mitral valve repair and severe progressive heart failure associated with intraventricular conduction disturbances

Raed Abu Shama, M.D.
Consultant Cardiologist and Electrophysiologist at Saud Al Babtain Cardiac Center (SBCC), Saudi Arabia

Dearest Andrés
I hope this email finds you well.
This is Raed Abu Shama again. The Palestinian electrophysiologist.
It has been a long time since we chatted and discussed cases. . . .
I have an interesting ECG for you. I think you are the one who will be able to solve this mystery!
It is for a 58 yr old male with IHD, S/P CABG, MV Repair in 2016. Severe LV dysfunction, LVEF 20%. Presented with progressive HF symptoms. MPI was done and showed mild ischemia in the mid anterior wall and in the inferior wall. Echo showed severe MR despite excellent initial repair results. They showed me the ECG today and I said the following:
This is a rare form of true trifascicular block. There is RBBB + LAFB + LSFB (QRS duration 156 ms).
Do you agree or have other alternatives?
Thank you in advance for your help.
Questions

1. Which is the ECG diagnosis?
2. Which is the appropriate approach?

Respect

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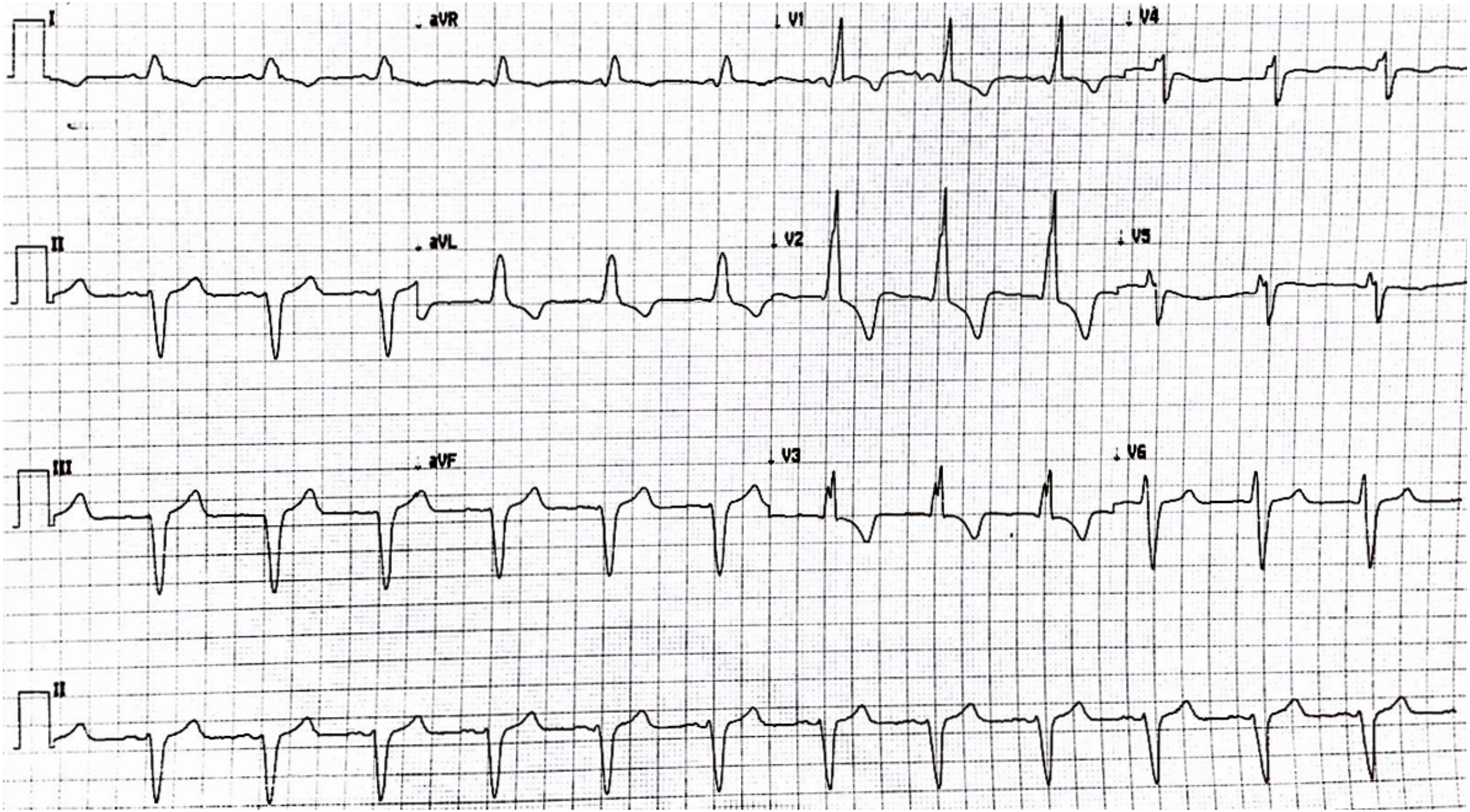
I
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Colleagues opinions

Spanish

Buen día estimado Andrés! Es un paciente con revascularización coronaria ("by pass" Ao Co) y plástica mitral. No menciona cuales arterias fueron revascularizadas ni si tuvo infarto previo. Tiene severo deterioro de la fracción de eyección del ventrículo izquierdo (FEVI= 20%) e IM, con alteración motilidad ínfero-anterior, con VI probablemente dilatado. **ECG:** ritmo sinusal 75 lpm, onda P bimodal con separación entre domos > 40 mseg, intervalo PR 180 mseg, duración del QRS 160 mseg,, fragmentación del fQRS de V3 a V5 conocido marcador electrocardiográfico de riesgo de MS, . eje eléctrico del QRS con extremo desvío a la izquierda (-60°), y QTc 450 mseg. **Conclusión** Crecimiento de aurícula izquierda, bloqueo divisional antero-superior izquierdo, bloqueo de la rama derecha, fuerzas anteriores prominentes(FAP). Estas últimas pueden corresponder tanto a bloqueo divisional antero-medial o de la división media de la rama izquierda por probable obstrucción proximal de la arteria coronaria descendente anterior izquierda(DA) antes de su primera rama perforante septal, o a necrosis ínfero-basal-lateral(antigua dorsal) del VI. Se trata de un paciente de alto riesgo por sus antecedentes, su bajísima fracción de eyección del ventrículo izquierdo y la mencionada fQRS. **Conducta:** Eventual nueva revascularización, optimizar tratamiento farmacológico e implante CDI y quizás resincronización. Cordialmente y a la espera de opiniones de expertos. Dr. Juan Carlos **Manzzardo Mendoza Argentina**

English

Good morning dear Andrés! This is a patient with aortocoronary bypass and as I understood it, mitral valve repair. It doesn't say which arteries were bypassed or if there was previous myocardial infarction. He has severe deterioration in his left ventricular function, (LVEF 20%) and mitral valve insuficience MVI, with inferior-posterior hypomotility, and probably LV dilated. **ECG:** sinus rhythm, heart rate 75 bpm, biphasic P wave with separation between humps >40 ms, PR interal duration 180 ms, QRS duration 160 ms, QRS axis -60°, QTc 450 ms. **Conclusion:** LA enlargement, LAFB, RBBB and proeminentn QRS anterior forces (PAF), fragmented QRS (fQRS) from V3 through V5; PAF could correspond to LSFB by ADA obstruction before the 1st septal perforator branch, or inferobasal-lateral necrosis(ancient dorsal) of the LV. This is a patient in high risk due to his history and current state. I would consider the possibility of a new revascularization, optimizing the pharmacological treatment and ICD implant, maybe with CRT.

Cordially, and waiting for the opinions by the experts,

Dr Juan Carlos **Manzzardo MD** Mendoza Argentina

Mi opinión del ECG: ritmo sinusal, agrandamiento de auricula izquierda bloqueo del fascículo antero-superior izquierdo bloqueo de rama derecho. Evoca también bloqueo del fascículo medio septal (diferenciar con un VCG una desviación en el plano horizontal cuadrante anteriorderecho de fuerzas prominentes por falta de oposición de fuerzas con necrosis lateral en ese contexto clinico relatado)
En síntesis
Bloqueo de tres divisions : RBBB + LAFB + LSFB.
Si el realmanente, Posterior izquierdo está comprometido (El HV con un EEF lo revelsaria) el paciente tiene riesgo potencial de bloqueo atrioventricular completo (BAVC),con riesgo de vida; por lo que recomendaría un dispositivo eléctrico acorde al la situación clínica y hemodinámica del paciente
Saludos
Juan José Sirena MD Santiago del Estero. Argentina

English

My opinion on the ECG. Sinus rhythm, left atrial enlargement(LAE), left anterior fascicular block(LSFB) , right bundle branch block(RBBB) There seems to be also LSFB (differentiate with VCG with horizontal plane shift, right anterior quadrant of PAF by lack of opposition of forces with lateral necrosis in the reported clinical scenario)
In summary
Trifascicular block: RBBB + LAFB+ LSFB If the remaining left posterior fascicle is compromised (an EPS would reveal it), the patient has a potential risk for complete atrioventricular block, with risk of life; so I would recommend an electric device implantation according to the clinical and hemodynamic scenario of the patient.

Regards,
Juan José Sirena MD
Santiago del Estero
Argentina

Spanish

Estimado Potro. El ECG referido presenta signos de crecimiento biauricular con un PR normal un QRS de 160 msg que evidencia un trastorno de la conducción no solo de la rama derecha y del fascículo anterior izquierdo e intramiocardico esto evidente por la presencia de rsR en AVR sin correlación son V1 y V2 que comienzan con onda q y fuerzas anteriores prominentes con empastamiento de la onda R. Podría ser la expresión de un bloqueo del fascículo medial por isquemia sumado al BRD y presenta un QRS fragmentado de V4 a V6.

Estos cambios los interpretó por isquemia en territorio de la DA proximal sumado a necrosis en territorio de la Cx, si esta es dominante. El compromiso de la DA y CX explica perfectamente la disfunción de ambos músculos papilares y la presencia de IM severa y miocardiopatia dilatada con baja FEY. Se realizó una plástica mitral y CRM aunque no refrieron los puentes realizados, creo que revascularizaron una CX ocluida y probablemte una DA, debería descartarse el fenómeno de robo coronario y que el puente en la DA no tenga un adecuado funcionamiento por esto presenta isquemia en dicho territorio a pesar de la revascularizacion. Realizaría una nueva CCG a fin de evaluar flujo de los bypass realizados. Un marcapasos no va a solucionar la disfunción de los músculos papilares y no es candidato a TRC a pesar del QRS de 160 msg.

Un abrazo. Martin Ibarrola MD Provincia de Buenos Aires Argentina.

English Dear Potro: The submitted ECG presents signs of biatrial enlargement with normal PR, QRS of 160 ms showing a conduction disorder not just of the right bundle branch and the left anterior fascicle and intramyocardial; this is evident by the presence of rsR in aVR with no correlation in V1 and V2, that starts with q wave and prominent anterior forces with R wave slurring. It could be the expression of left septal fascicular block by ischemia, added to the RBBB and presenting fragmented QRS from V4 through V6. I interpret these changes as ischemia in the proximal ADA territory, added to necrosis in the Cx territory, if this is dominant. ADA and Cx involvement perfectly explains the dysfunction in both papillary muscles and the presence of severe MVI and dilated cardiomyopathy with low LVEF (20%). Mitral valve repair was conducted, and revascularization, although the bypasses made are not stated, but I think they revascularized occluded Cx and probably ADA. The phenomenon of coronary steal should be ruled out, as well as the ADA bypass not having a proper function leading to ischemia in this territory in spite of the revascularization. I would perform another coronary angiography to evaluate the flow in the bypasses made. A pacemaker will not solve the dysfunction of the papillary muscles, and he is not a candidate to CRT in spite of the QRS of 160 ms.

Warm regards,

Martin Ibarrola MD Buenos Aires Argentina

Dear professor, what do you think about a quadrifascicular block in this case? I mean: apparently there is electrocardiographical partial left bundle branch block in D1 and aVL. And obvious signs of RBBB. Also, as stated by your friend in Saudi Arabia, there is LSFB + LASB.

What I also suspect is that the left inferior posterior fascicle is also partially blocked: there is a bigger Initial activation delay in aVL than in V6, a sign that Medrano defines as a bifascicular block (Medrano, Arq Cardiol Mex, 2002:72.240-248).

What do you think about this idea?

Dr. José Nunes de Alencar Neto
Cardiologista - Eletrofisiologia

Portuguese

Caro Andrés, Trata-se de um grave acometimento do sistema His-Purkinge pois além do bloqueio trifascicular já mencionado (BRD+BDASE+BDAM), apresenta-se no plano frontal padrão de bloqueio de ramo esquerdo (vide D1 e aVL), contrastando com as precordiais de BRD. Trata-se portanto de BRE mascarado. O fato do intervalo PR ser normal não exclui um intervalo HV aumentado, pois tal intervalo pode ser compensado pelo intervalo AH no limite inferior da normalidade.

Comprovando isso, poderá se indicar um marcapasso definitivo.

Marcelo Garcia Leal MD Ribeirão Preto São Paulo Brasil.

English

Dear Andrés, This is a serious affection of the His-Purkinge system because in addition to the aforementioned trifascicular block (BRD + BDASE + BDAM), the frontal plane presents a pattern of left bundle branch block (see D1 and aVL), contrasting with the precordial BRD. It is therefore masked BRE. The fact that the PR interval is normal does not exclude an increased HV interval, since such an interval can be compensated for by the AH interval at the lower limit of normality.

If this is proven, a definitive pacemaker may be indicated.

Marcelo **Garcia Leal MD** Ribeirão Preto São Paulo Brazil.

Coordenador do serviço de arritmia clínica da divisão de cardiologia do Hospital das Clínicas de Ribeirão Preto.

Coordenador da enfermaria de cardiologia do Hospital das Clínicas de Ribeirão Preto

Chefe do serviço de cardiologia da Santa Casa de Ribeirão Preto.

Final conclusions

By Andrés Ricardo Pérez-Riera, Raimundo Barbosa-Barros and Raed Abu Shama,

Dear colleagues: this is a case of "Standard Type" or Standard Masquerading Bundle-Branch Block: consisting of the pattern of left bundle-branch block (LBBB) in the limb leads (I and aVL) and right bundle-branch block (RBBB) in the unipolar precordial leads (**Richman1954; Ortega-Carnicer 1986**). Additionally, he has LSFB. Consequently, this is a new variant never published before!!! There are 3 types of Masquerading (or 4 Total).

I. The "standard type" or standard masquerading Bundle-Branch Block: consisting of the pattern of LBBB in the limb leads and right bundle-branch block (RBBB) in the unipolar precordial leads. In the present cases additionally we have LSFB. LAFB may obscure the diagnosis of RBBB, abolishing the S waves in lead I, aVL and the terminal R wave in V_1 (**Rosenbaum 1968; 1971; 1973; Sclarovsky 1979**).
II. The "Precordial Type" or Precordial Masquerading Bundle-Branch Block
III. The Standard and Precordial Masquerading Bundle-Branch Block in association.
IV. The Total Masquerading Bundle Branch Block (**Kukla 2015**).

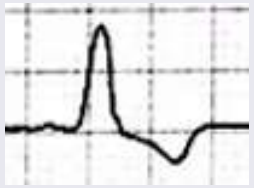
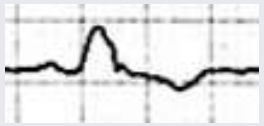
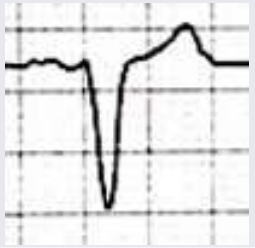
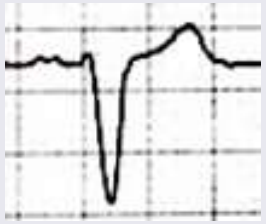
In "standard masquerading" RBBB the presence of a high degree of LAFB obscured totally or partially the diagnosis of RBBB only in the frontal plane by abolishing (or becomes very small) the final broad S wave in the leads I and aVL. Consequently, the limb leads resemble LBBB although the precordial ECG remain typical for CRBBB associated with LSFB. The precordial leads reflect the feature of RBBB in this case associated with LSFB because the R wave of V2 is very pointed, and additionally we have absence of initial q wave in left leads consequence of absence of first vector or septal vector of Penaloza e Tranchesi of ventricular depolarization (**Penaloza 1955**).

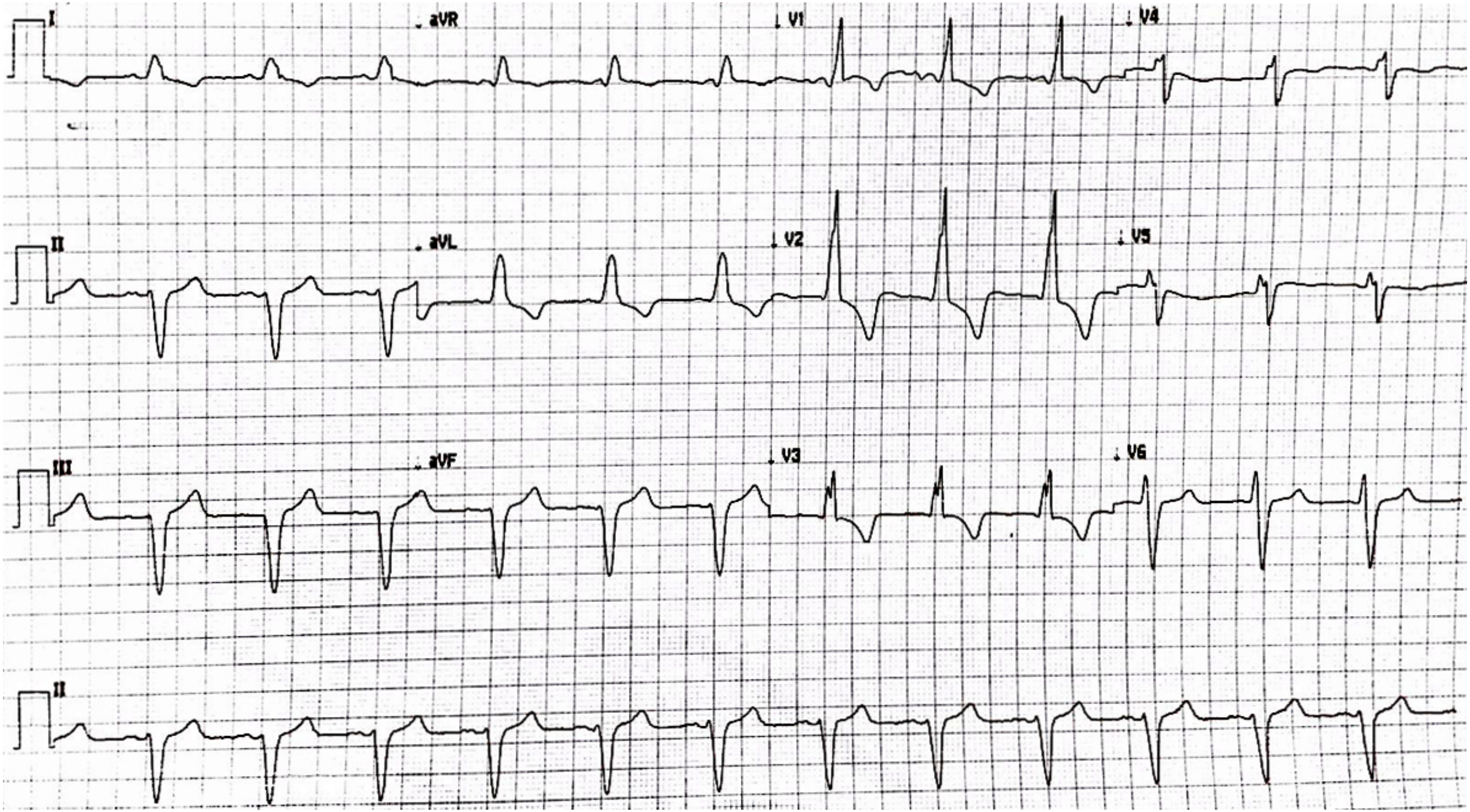
Necessary conditions for the presence of standard masquerading RBBB:

1. High degree of LAFB. When LAFB has high degree the QRS axis in limb leads is near −60°, the ECG becomes atypical. It can lead to very small or even absent of S wave in lead I. The ECG masquerading RBBB and it imitates LBBB is limb leads (**Kukla 2015**).
2. RBBB
3. Bilateral bundle-branch lesions of considerable intensity, which do not completely disrupt the continuity of the branches
4. Left Ventricular Enlargement or Hypertrophy (LVE/LVH) and marked biventricular hypertrophy
5. Localized block in the LV.
6. Severe fibrosis, or truly massive myocardial infarction mainly in anterior wall.

Possible etiologies: Coronary heart disease, long standing systemic hypertension, cardiomyopathies (Ex. Chronic Chagasic myocarditis in Latin America) (**Dubner 2008**), Lev´s disease (progressive idiopathic fibrosis and calcification of the intraventricular conduction system of the heart), Lenegre´s disease (genetic SCN5A mutation allelic with Brugada syndrome) and association of the previous one. Konopka et al presented a case of masquerading bundle branch block obscuring the diagnosis of Brugada syndrome (**Konopka 2017**). **Prognosis:** poor (**Dhanse 2016**). Nevertheless, Acunzo et al. presented cases with aberrant conduction in apparent healthy individuals (**Acunzo 2013**).

The four main developmental ECG patters of standard type (modified from Schamroth 1975)

		aVL	I	II	III
1.	Uncomplicated isolated 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 or absence of the final QRS vectors and diminution or absence of the initial QRS vectors (the present case)	R 	R 	QS or rS 	QS or rS 


I
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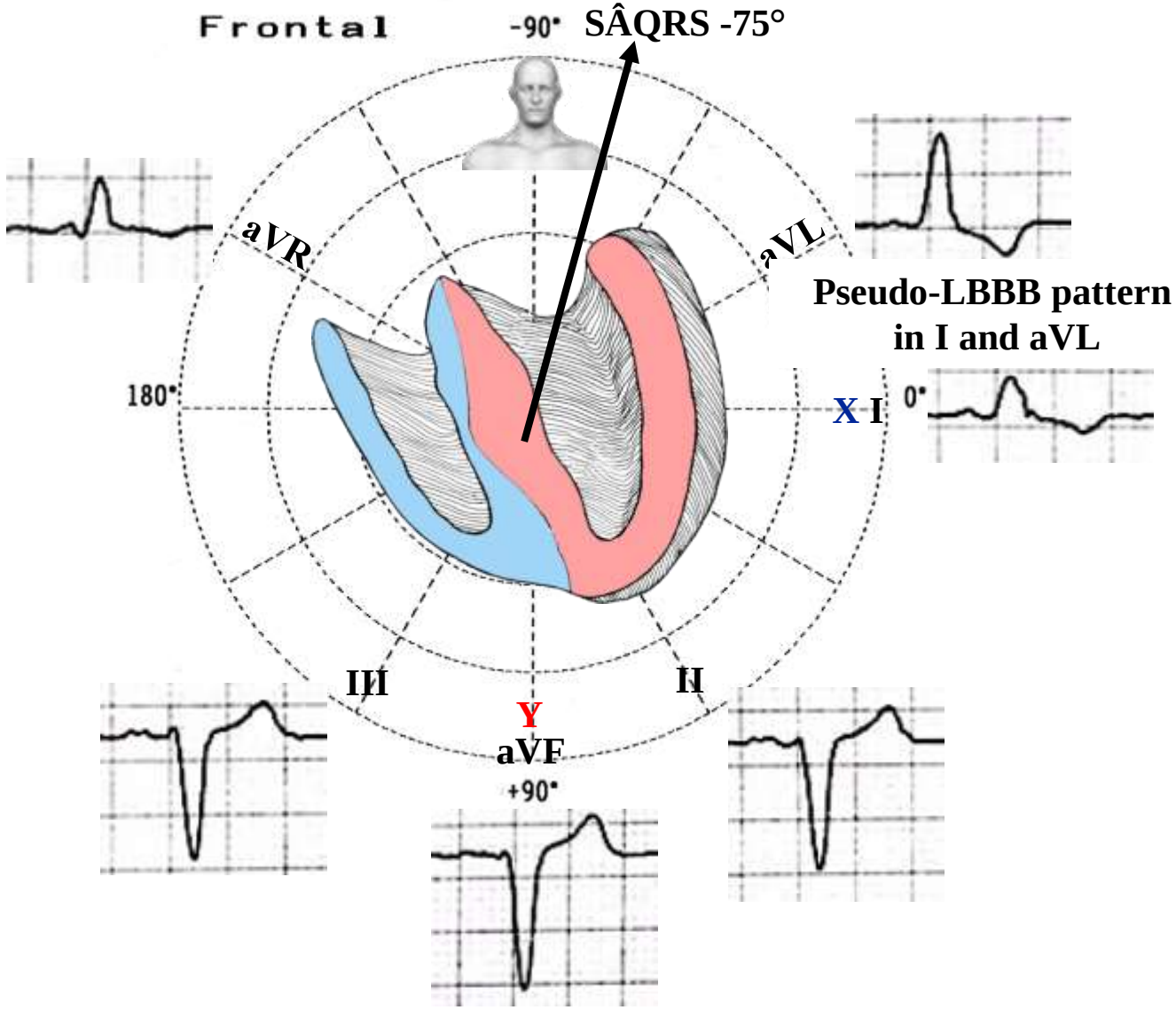
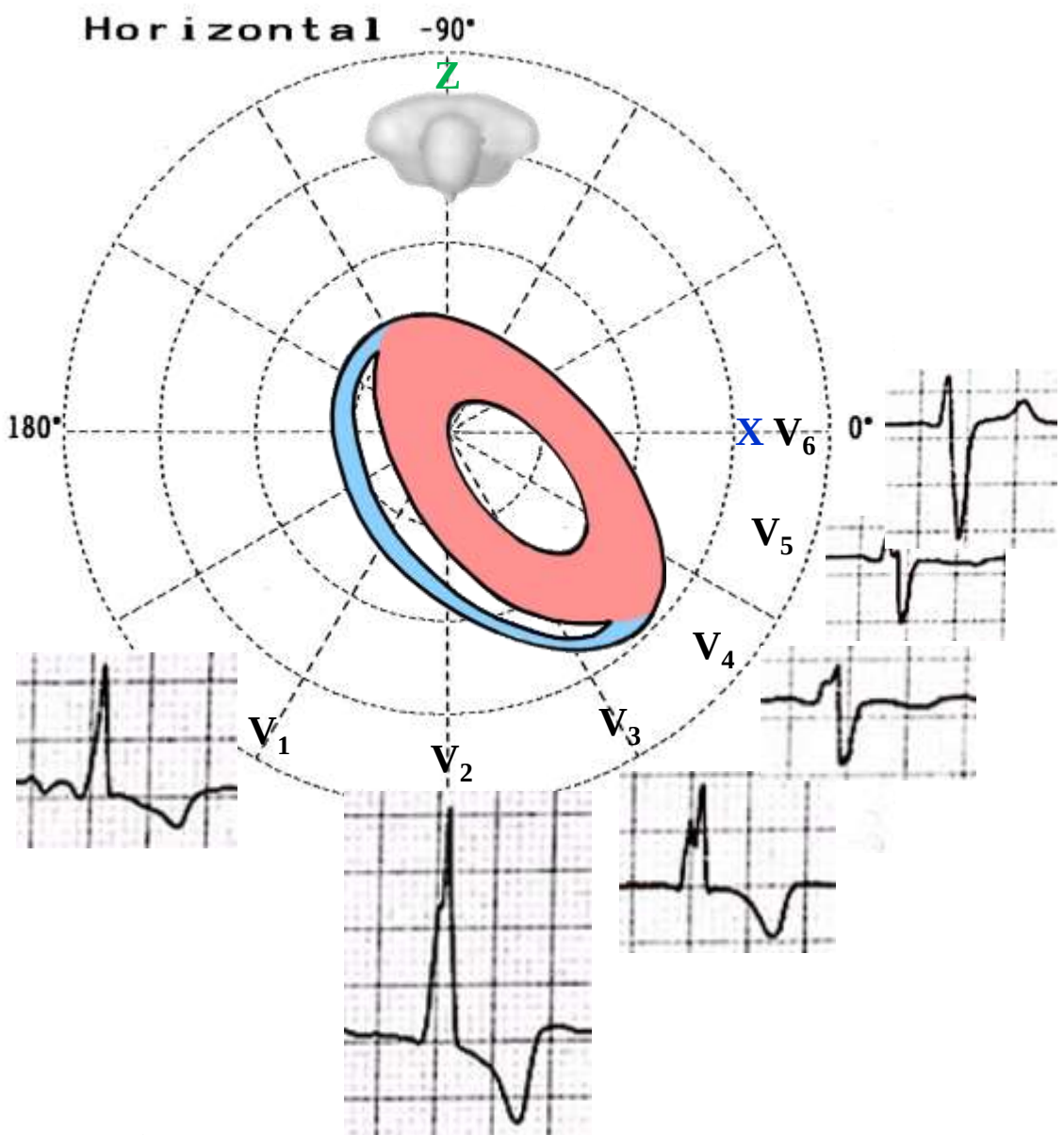
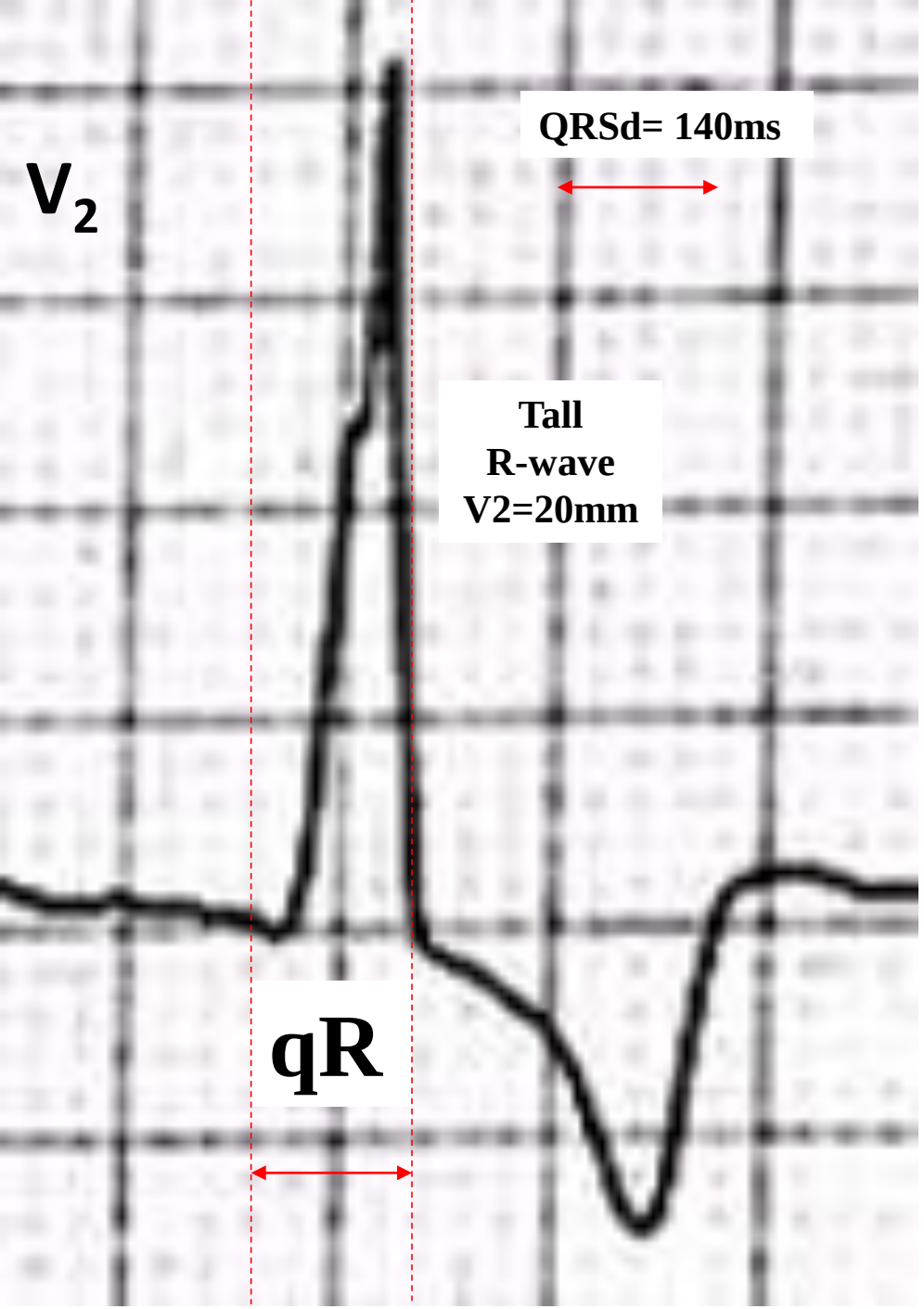
II



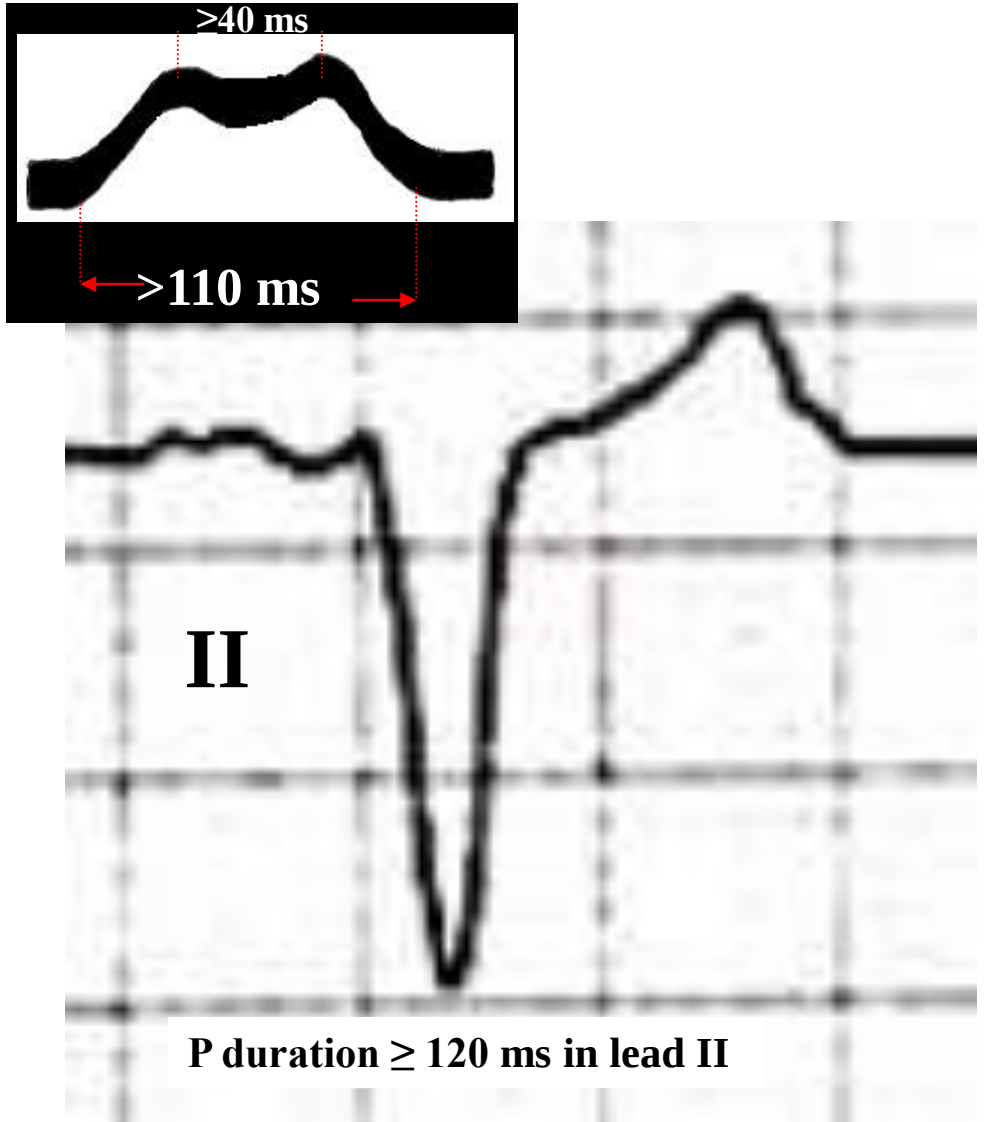
Pseudo-LBBB pattern in I and aVL

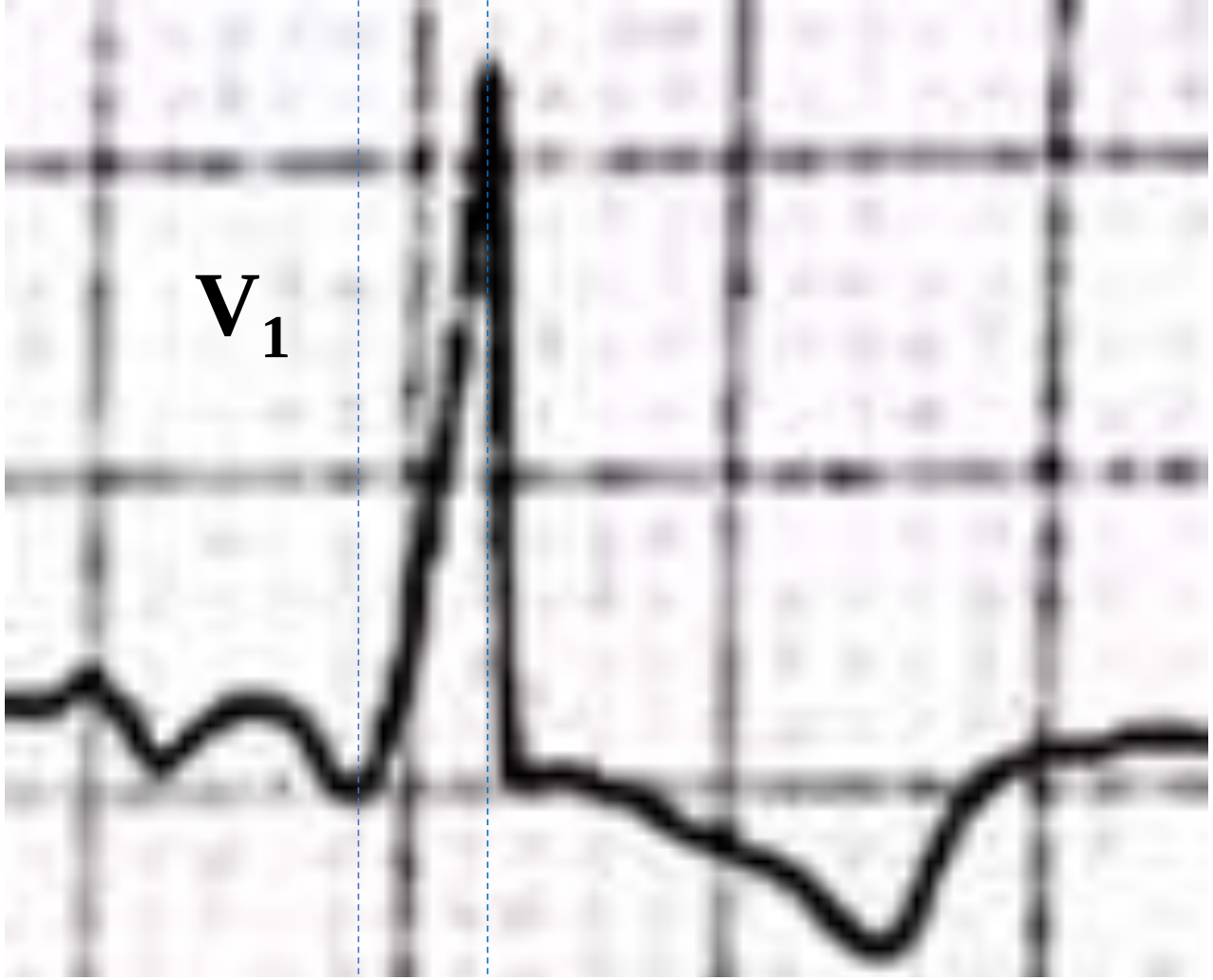


Prominent anterior QRS forces, embryonic initial q wave in V1-V2, tall R wave in V2(20mm), R “in crescendo” V1-V2 and decrescent in lateral leads, prolonged R-wave peak time in V1(all criteria of LSFB) (**Pérez-Riera 2011; Pastore 1016**)



Abnormal broad and bifid or bimodal P-wave: Left Atrial Enlargement (LAE)





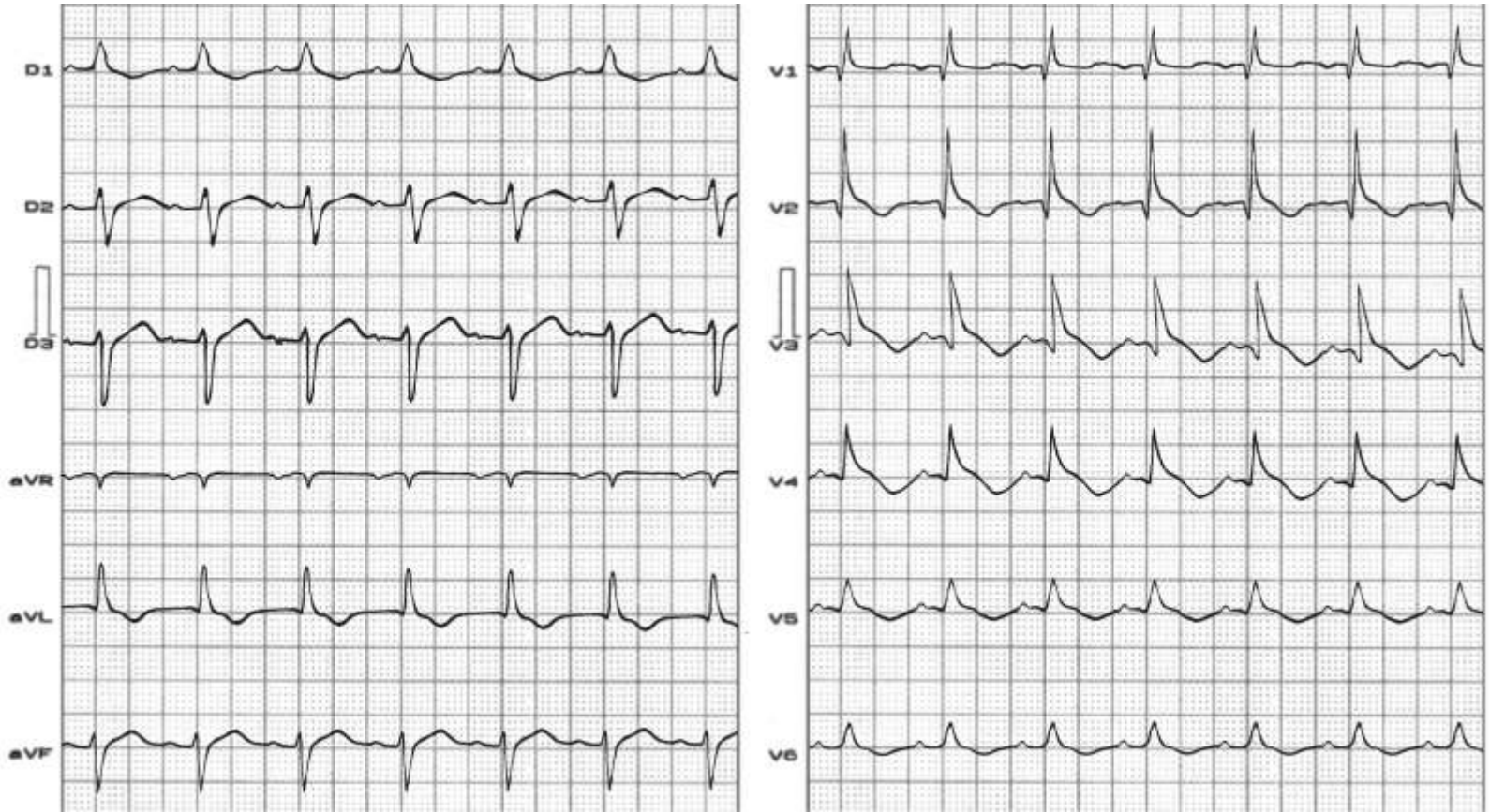
Ventricular activation time or R-wave peak time = 80ms

II. The precordial type (*"precordial masquerading right bundle-branch block"*

This type 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/enlargement (LVH/LVE), 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. Finally, 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**).

III. The Standard and Precordial masquerading bundle-branch block in association

In this case the limb leads show an apparent pattern Left bundle-branch block with extreme left axis deviation (LAFB) and the precordial leads exhibit the pattern of CRBBB in the right precordial leads and LBBB pattern in left precordial leads V5-V6. Additionally, an abnormal Q waves are frequently present on right precordial leads.



ECG diagnosis in the present case

1. Left atrial enlargement; 2) Standard Masquerading Bundle-Branch Block; 3) Advanced LAFB; 4) Complete RBBB; 5) LSFB (**Pérez-Riera 2011; Pastore 1016**) Prominent QRS anterior forces (**Nakaya 1978**), prolonged R-wave peak time in V1, R-wave "in crescendo" de V1 to V2 and decreasing in lateral leads, R-V_2 wave tall > 15mm, absence of initial q wave in lateral leads V5-V6, I and aVL (consequence of absence of the first vector, middle septal vector or Peñaloza and Tranchesi vector (**Penaloza 1955**). The present case is a new variant of trifascicular block. 6) Probable lateral myocardial infarction in association (anterior dorsal) The posterior or dorsal wall does not exist !!) (**Bayés de Luna 2006**)

The term trifascicular block refers to a combination of RBBB with intermittent LAFB and LPFB, and trifascicular block is a possibility only when RBBB is associated with either LAFB or LPFB and incomplete AV-block (because the AV block may be sited in the AV node, the bundle of His, or the LBB proximally, rather than in the remaining fascicle.

1. Probable lateral myocardial infarction. Rosembaum et al. described 8 possible types of trifascicular blocks (**Rosenbaum 1969a;b**).

- Block is present in the RBB as well as in the two main divisions of the LBB, resulting in complete AV block.
- Block is permanent in the RBB and the LPF or the LBB and intermittent in LAF. The ECG will show RBBB+ LPFB and varying degree of incomplete AV block.
- Block is permanent in the RBB and the LAF or in the LBB and intermittent in LPF. The ECG will shows RBBB+LAFB+ and varying degrees of incomplete AV block.
- Block is permanent in both main division of LBB and intermittent in RBB. The ECG will show LBBB and incomplete AV block.
- Block is permanent in the RBB and incomplete in both main divisions of the LBB. The ECG may show BRBB with either LAFB or LPFB and incomplete AV block.
- Block is permanent in the LPF and in LAF and RBB. The EGG usually shows RBBB with LPFB or LBBB and incomplete AV block. Less likely possibility are incomplete LBBB with LPFB and pure LPFB
- Block is permanent in the LAF and intermittent in LPF and RBB. The ECG will usually show RBBB with LAFB or LBBB, and incomplete AV block. Less likely possibilities are incomplete RBBB with LAFB, and pure LAFB.
- Block is unstable or intermittent in all three fascicle except LSF. The ECG most likely will show RBBB with either LAFB or LPFB or LPFB; or LBBB, LAFB, or LPFB; incomplete RBBB or LBBB with either LAFB or LPFB or a normal QRS complexes

In the present case we have LAFB+ RBBB+ LSFB another trifascicular block yet not described in the literature This ECG pattern show the tretrafascicular nature of the intraventricular conduction system.

Approach

Optimize drugs for the treatment of heart failure and ICD implantation (LV dysfunction due to prior MI who are at least 40 days post-MI, have an LVEF ≤ 30%, and are in NYHA Functional Class I)

Previous CDI implant perform EPS to check the HV interval. If New York Heart Association Class II-IV HF is present, and LVEF ≤35%, ECG QRS width ≥ 120 ms in the presence of LBBB, cardiac resynchronization therapy (CRT) is indicated. In the present case, CRT is not indicated because the ECG pattern does not present the so called "the responder pattern". Accurate selection of patients with LBBB helps increasing response to CRT. There is no agreement on LBBB definition: the classical or traditional LBBB according to American Heart Association (**Surawicz 2009**) or LBBB with stricter Strauss criteria or true-LBBB for complete LBBB that include a QRS duration ≥ 140 ms for men and ≥ 130 ms for women, along with mid-QRS notching or slurring in ≥ 2 contiguous leads **(Strauss 2011).** In a universe of LBBB applying stricter criteria, only 37% of patients undergoing CRT showed a true-LBBB according to Strauss. Accurate identification of true-LBBB has a potential additional value in better selecting patients. Patients with an LBBB according to Strauss show a significantly more prolonged QRS duration, greater baseline end-systolic and end-diastolic volumes compared with those with IVCD or classical LBBB (**Migliore 2016**).

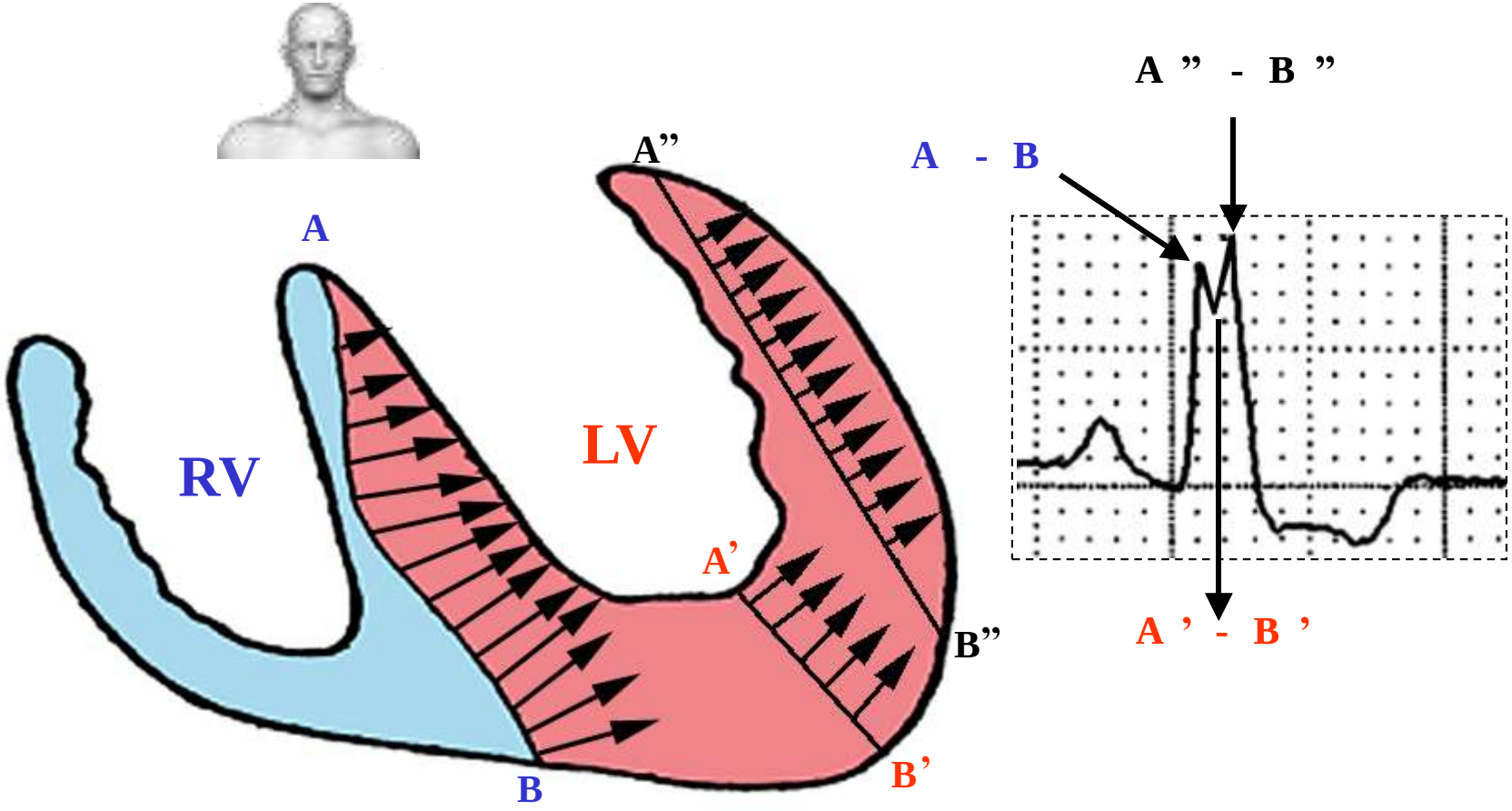
In patients with conventional wider LBBB morphology, the presence of mid-QRS notching or slurring is a strong predictor of better response to CRT (**Tian 2013**).

Reevaluation of the data of CRT trials and EPS findings in LBBB provided evidence that stricter or "true" LBBB. In "true" LBBB, after the 40th ms of the QRS notched/slurred R waves are characteristic in minimum two of I, aVL, V1, V2, V5 and V6 leads, in addition to a ≥40 ms increase of the QRS complex, as compared to the traditional QRS complex. In contrast, slowly and continuously widened "LBBB like" QRS patterns are mostly occur in LVH or in a metabolic/infiltrative disease (**Préda 2013**).

Response to CRT seems to increase as the QRSd becomes longer, with greatest benefit in QRSd ≥150 ms. Data have placed more emphasis on QRS morphology, demonstrating variability in clinical response between patients with left bundle branch block, non-left bundle branch block, and RBBB morphology. Notably, myocardial scarring and cardiac dimensions, among other variables, may alter heterogeneity in ventricular activation. Understanding the electrophysiological underpinnings of the QRS complex has become important not only to predict response but also to facilitate the patient-specific delivery of CRT. Response in patients with RBBB morphology or IVCD pattern are neutral (non-responders) (**Poole 2016**).

Additionally, electrical remodeling (ER) and mechanical remodeling after CRT of native conduction precedes detectable LV structural changes after CRT. ER of native conduction is associated with better clinical outcome following CRT (**Cvijić 2017**).

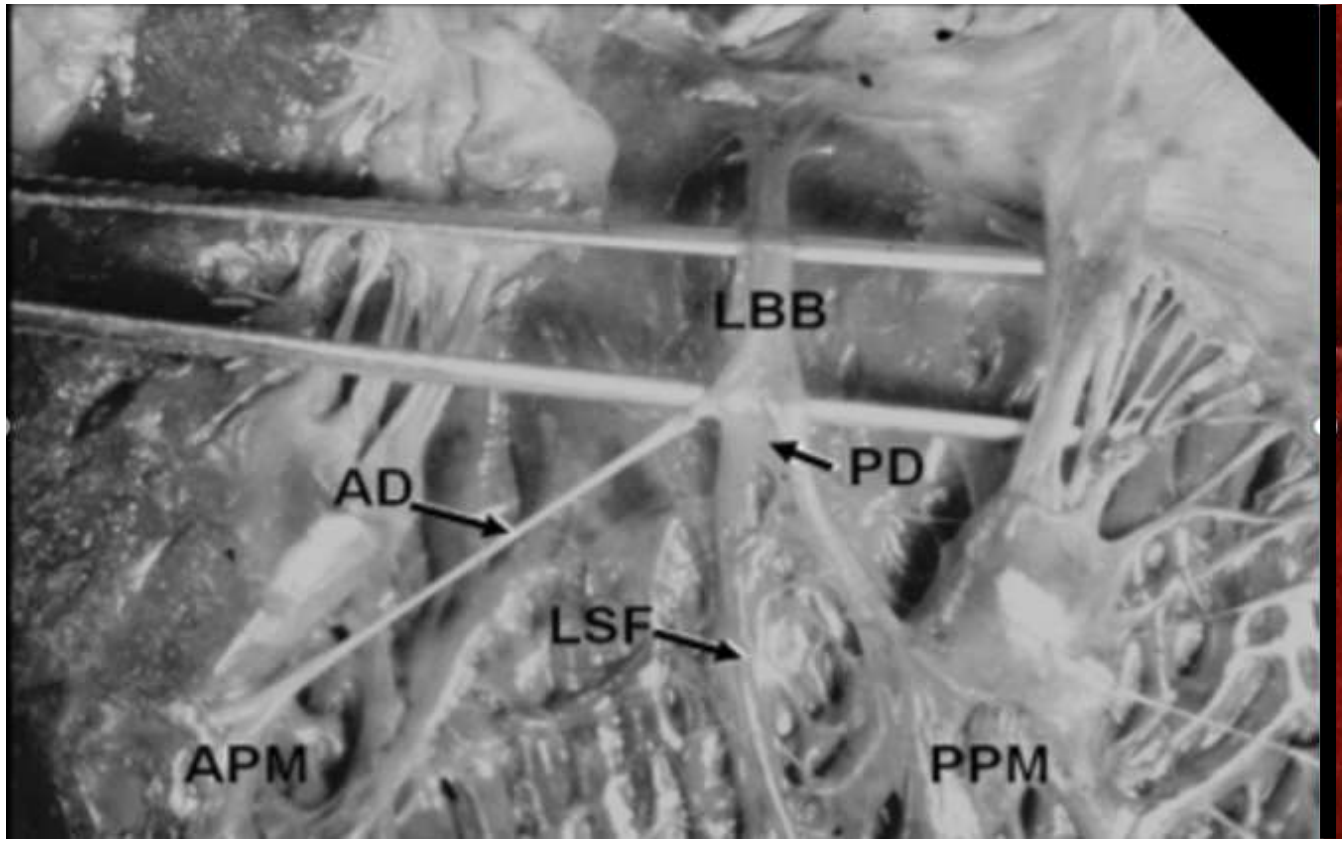
Monophasic R wave of slow recording in left leads I, aVL, V5 and V6 and electrophysiological explanation

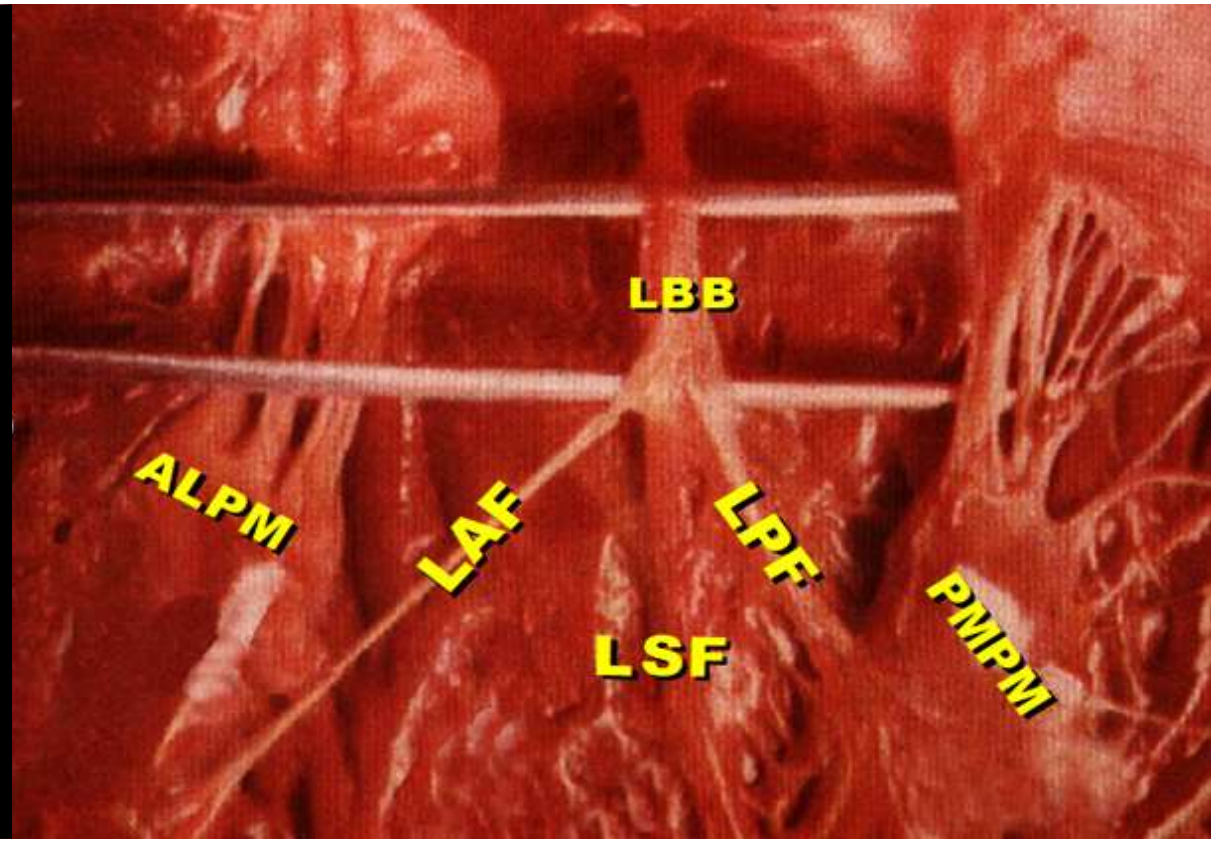


Septal depolarization from right to left makes a wide A-B wave front; however, when the stimulus reaches the central portion of the LV (cavity), it suffers a marked decrease in wavefront width (A’-B’) responsible for the notch in the apex of R wave (stricter Strauss criteria). Next, the wavefront reaches the LV free wall increasing again the width of the wavefront (**A”-B”**), responsible for the second apex of R wave. In the severe hypertrophies of the free wall, this second apex presents a higher voltage related to the first one.

Left Fascicular Blocks: It is time to replace the nomenclature "hemiblocks"

We have read with great interest the excellent review paper by Dr. Marcelo Víctor Elizari,(**Elizari 2017**) titled "The normal variants in the left bundle branch system". Dr. Elizari is the main living disciple of the school of Mauricio B. Rosenbaum. This legendary school of electrocardiography consolidated the concept of "hemiblocks" in the 60s and 70s of the 20th Century. With satisfaction, we have verified that the author shows the existence of the left septal fascicle (LSF) identified in Figure 10B of this review (see bellow).

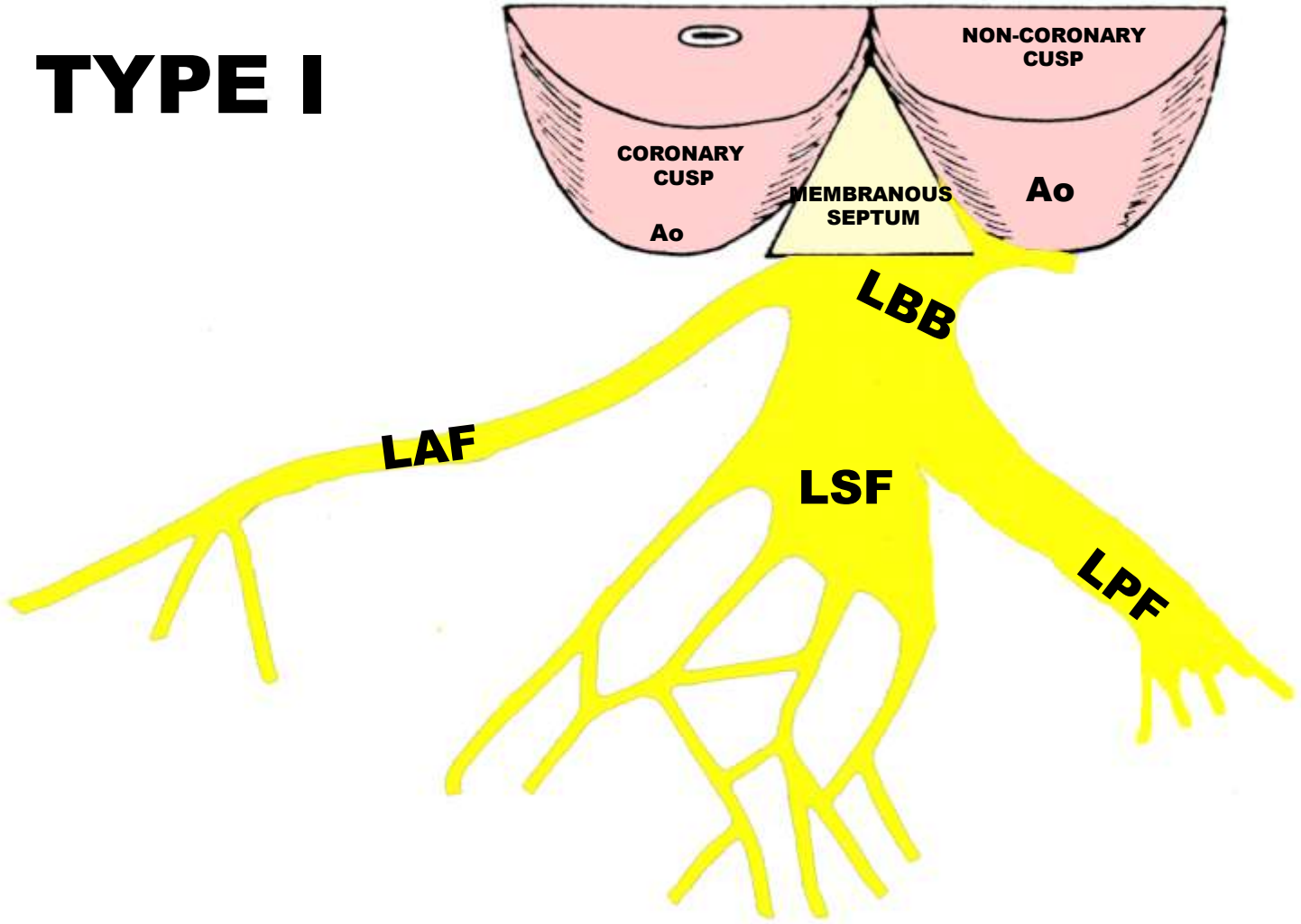




The endocardium has been removed and the main LBB (LBB) and its posterior division or left posterior fascicle (PD or LPF) have been dissected in their entirety. The anterior division or left anterior fascicle (AD or LAF) still surrounded by endocardium cuts freely across the left ventricular outflow tract as a false tendon. The difference between the extremely thin LAF and the much thicker posterior division (PD or LPF) is remarkable. After a short distance the latter gives off a gross strand going to the posterior third of the septum. LSF: left septal fibers or left septal fascicle ; APM or ALPM: anterolateral papillary muscle; PPM or PMPM: posterior papillary muscle or posteromedial papillary muscle.

Despite of this clear anatomical corroboration, the author seems to ascribe a minor category to the LSF, since in the conclusions he literally wrote: "**In some cases a well-defined left septal fascicle can be identified, usually arising from the posterior division**" This sentence does not clarify the percentage of cases in which this would happen. The classical histological study of the left bundle branch (LBB) system carried out by Demoulin & Kullbertus (**Demoulin 1972**), on 20 hearts from patients devoid of conduction defects confirmed the consistent presence of a thin (diameter 3 mm) and elongated (extension 35 mm) left anterior fascicle (LAF) and a wider (diameter 6.2 mm) and shorter (extension 15 mm) left posterior fascicle (LPF). However, in addition to these two well-known fascicles, the LBB was observed to frequently give off a third one (LSF) designated to cover the midseptal surface in 55% of the cases. This easily identified structure emerged either from the common LBB (25%) or from the LAF (15%). In the remaining 45% of the cases, LPF or by a complicated plexus of ramifications given off by both the LAF and LPF (30%). The anatomical features were such that, in most cases, it seemed reasonable to describe the left ventricular Purkinje arborizations as composed of three main, widely interconnected peripheral portion depending upon the LAF, LPF and LSF (central radiation or plexus of ramification coursing to the midseptal area). These observations are concordant to the Rossi ones in 1971. The use of the term hemiblocks leads to think in the existence of only two fascicles in the LBB system. Additionally, we consider the functional demonstration in the human heart by the Dutch investigator Dick Durrer in 1970 (**Durrer 1970**) to be conclusive; in it the initial ventricular activation is shown to occur in 3 points and not two. The LSF is responsible for the first vector of ventricular depolarization, middle third vector of the left septal surface, vector 1, antero-medial (1_{AM} vector), corresponding to the first vector described empirically in 1955 by Peñaloza and Tranchesi, and for this reason in Brazil it is known by the eponymous term first vector of Penaloza-Tranchesi (**Penaloza 1955**). The resulting vectors of the anterior paraseptal and posterior paraseptal area by presenting opposite directions, nullify each other, thus expressing only the vector of the mid-septal region depending from the LSF. This vector is responsible for the small initial r wave of V1 or V1-V2 and the concomitant q wave of the left leads V5-V6. Then, considering the trifascicularity of the left His system, it would be appropriate to drop the term hemiblocks. Because of this, we think it is more appropriate to call these blocks: left anterior fascicular block (LAFB), left posterior fascicular block (LPFB) and left septal fascicular block (LSFB). The assumptions which formed the basis of left hemiblock concept were extremely useful for didactic purposes. Nevertheless, currently, the trifascicular concept of the LBB deserve attention, particularly from the anatomical, histopathological, electro-vectorcardiographical, electrophysiological, and mainly clinical points of view, because in the developed countries, the transient (**Athanassopoulos1979**) or permanent LSFB is caused mainly by significant proximal obstruction of left anterior descending coronary artery, before its first septal perforator branch (**Uchida 2006; Riera 2008**). On the other hand, in Latin America the main cause of the LSFB is South American trypanosomiasis cardiomyopathy (Chagas' disease) (**Pérez-Riera 2015; Pérez-Riera 2016**). We described the electro-vectorcaardiographic evolution of LSFB in a case of Kearns-Sayre syndrome (**Riera 2008**).

TYPE I



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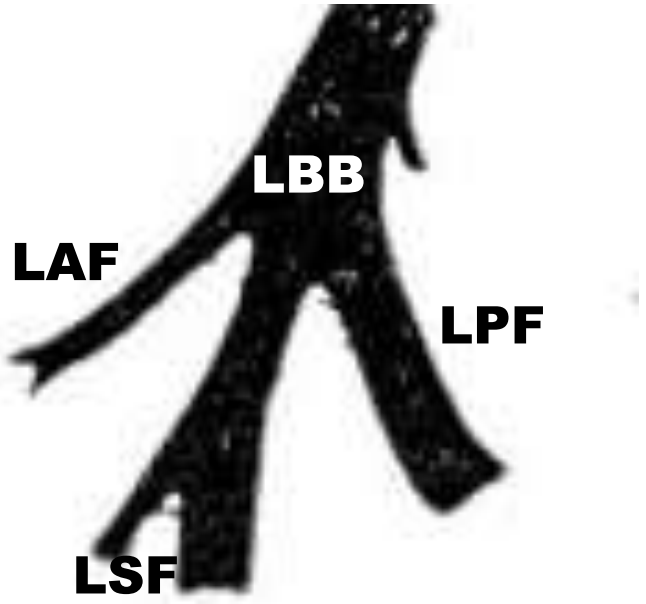
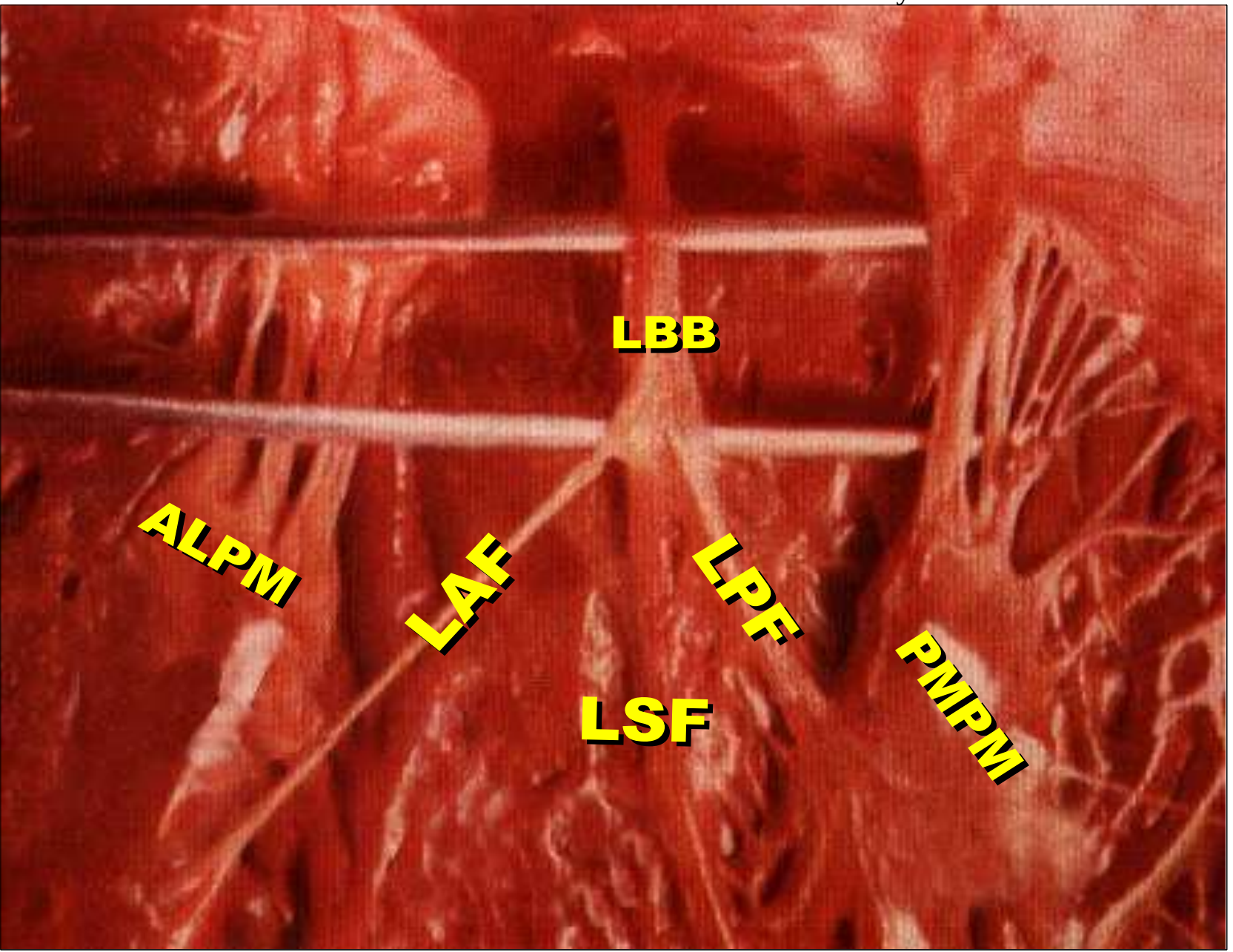


From Demoulin JC and Kulbertus HE. Histopathological examination of concept of left hemiblock. Br Heart J 1972;34:807-814.

The figure below shows a lateral view of the endocardium of the IVS in the human heart. In this case, unquestionably, the LSF originates from the main LBB. Additionally, the LAF conducts to the ALPM of the mitral valve and the LPF straight to the PMPM muscle of the mitral valve. Type I anatomic variation and the trifascicular nature of the human left His system.



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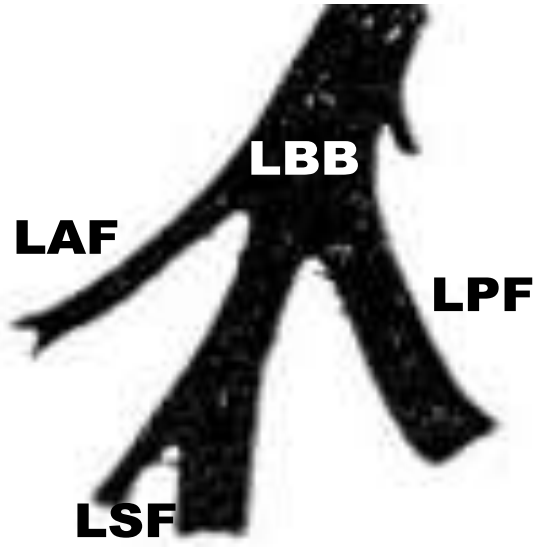
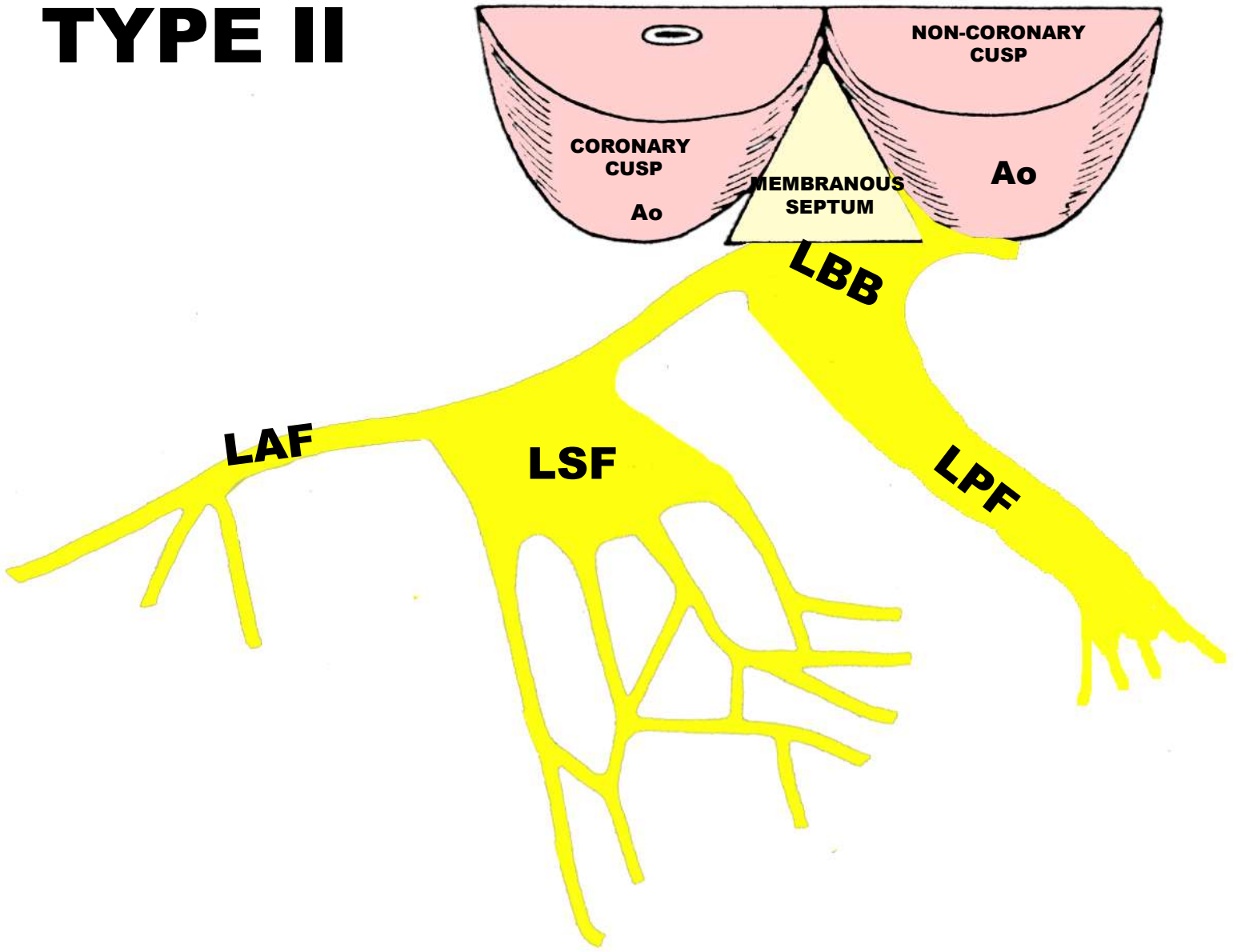
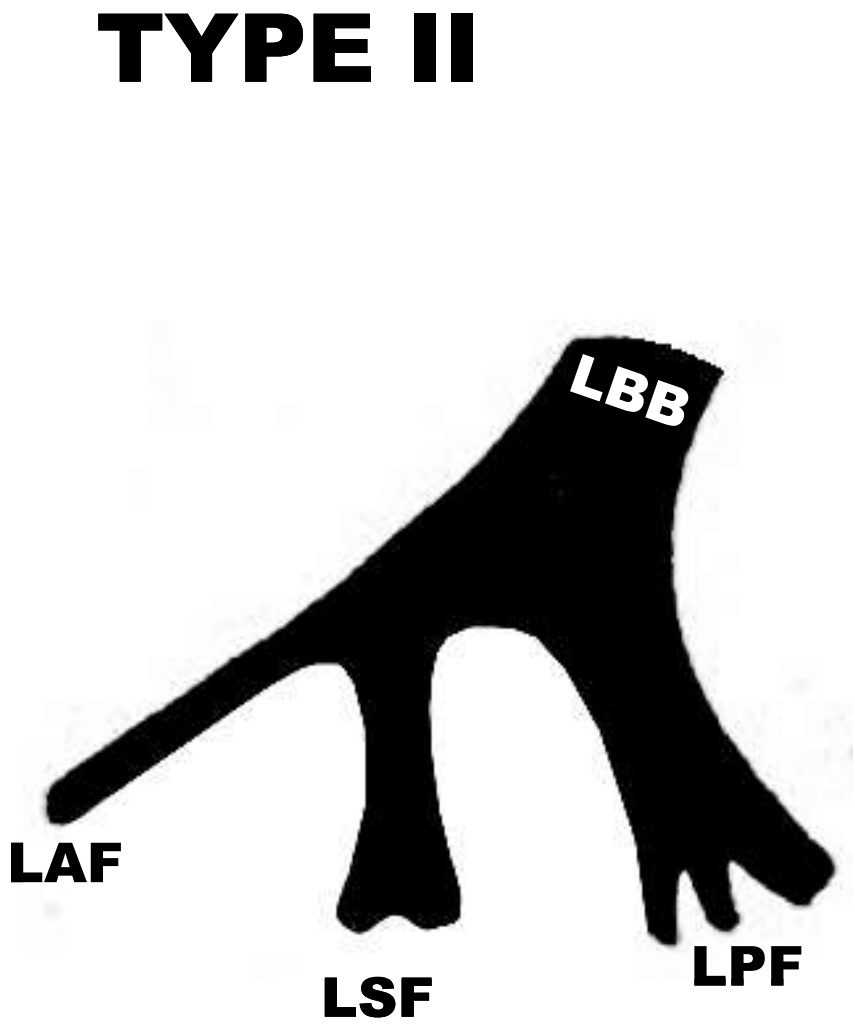


From Demoulin JC and Kulbertus HE. Histopathological examination of concept of left hemiblock. Br Heart J 1972;34:807-814.

PMPM POSTERO-MEDIAL PAPILLARY MUSCLE

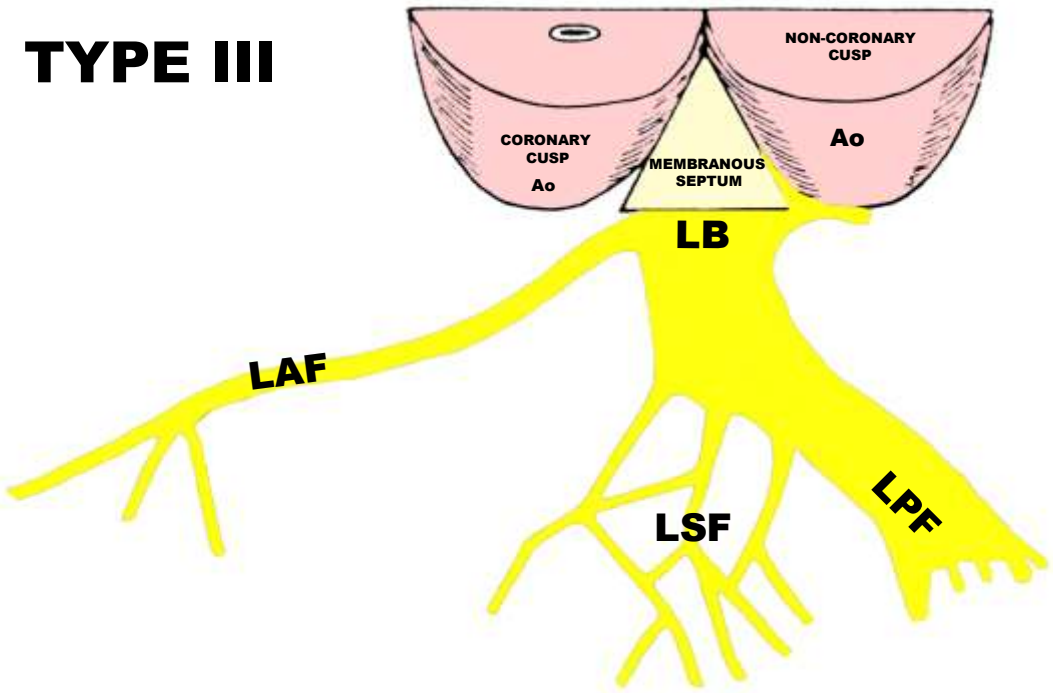
ALPM ANTERO-LATERAL PAPILLARY MUSCLE

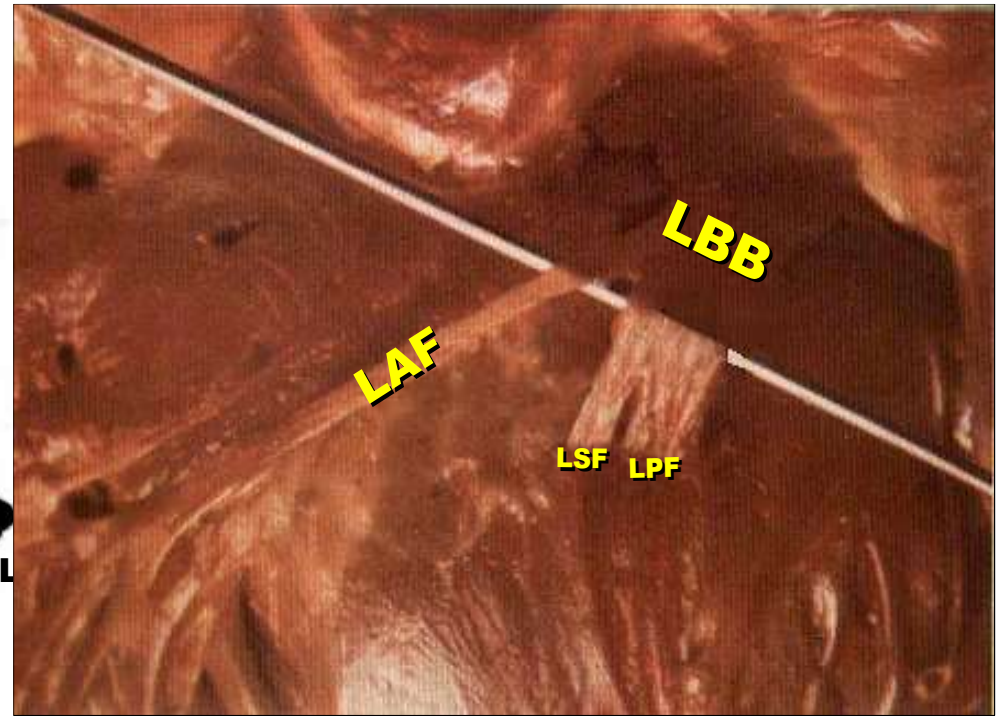


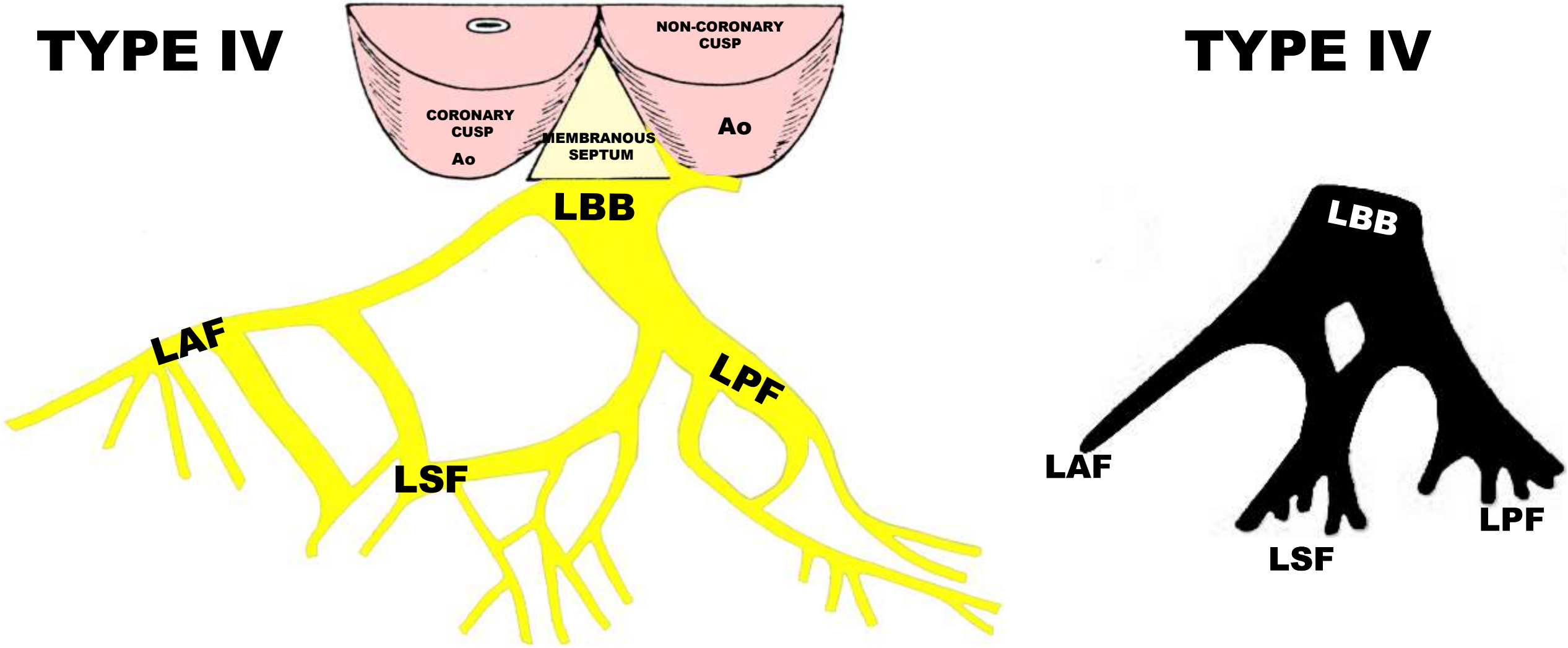


Type II. In this case, the LSF originates from the LAF of the LBB.

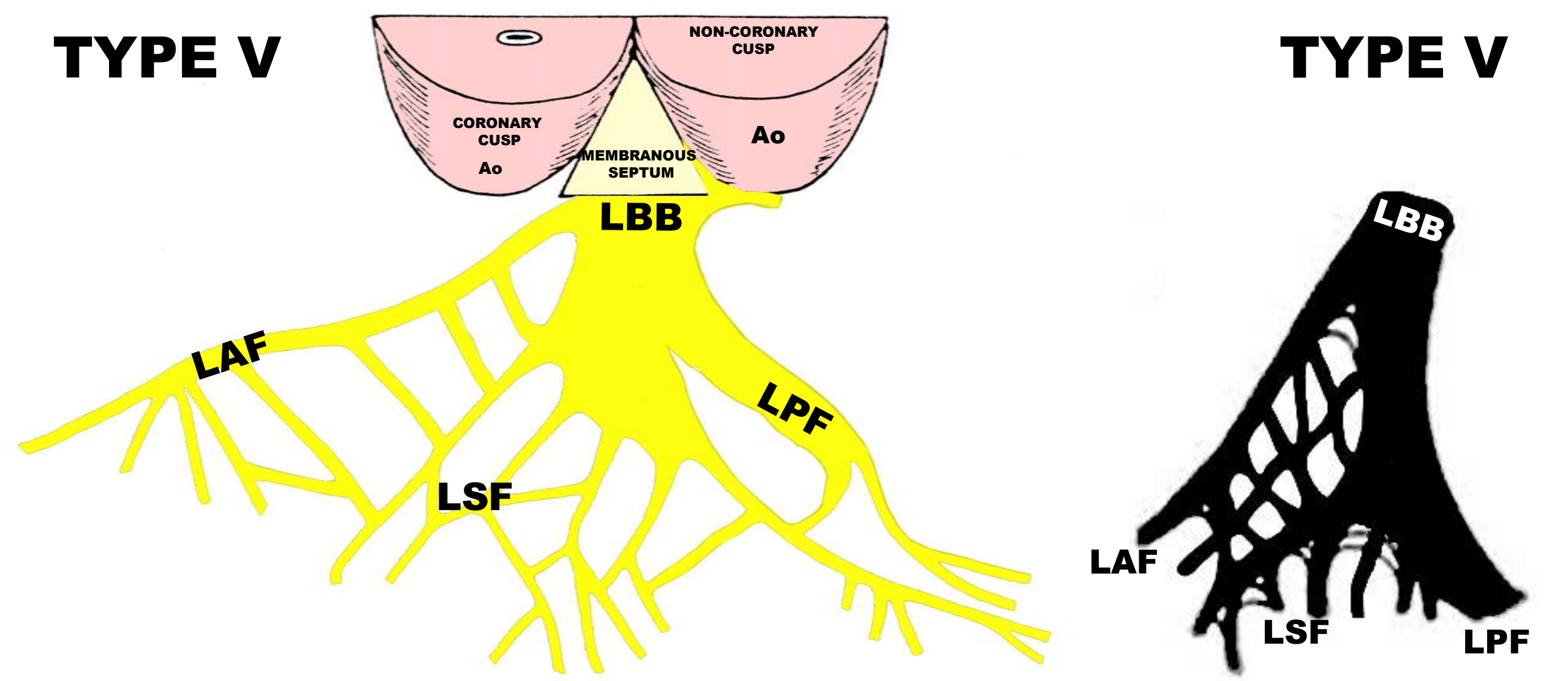
Type III. The left septal fascicle originates from the left posterior fascicle. This type represents 2.4% of all cases. On the other hand Elizari refers that this is the most common type (**Elizari 2017**).



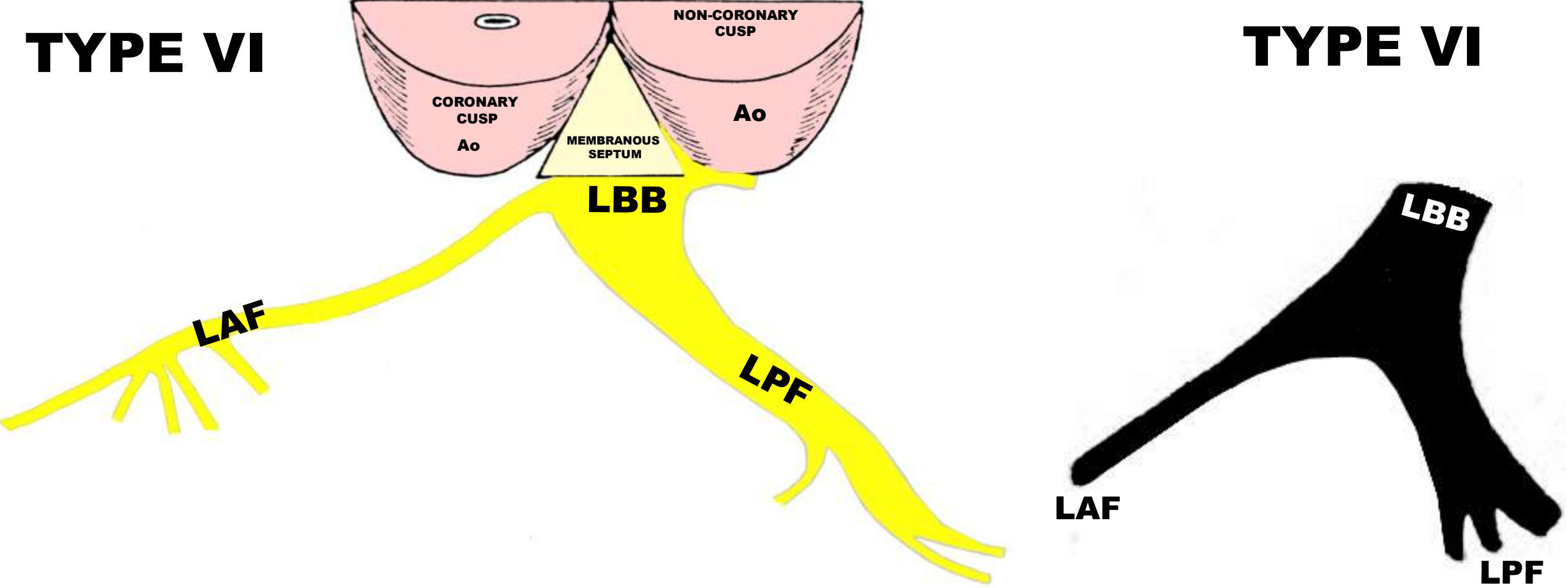




Type IV. In this case, the LSF originates concomitantly with the other two fascicles (LAF and LPF).

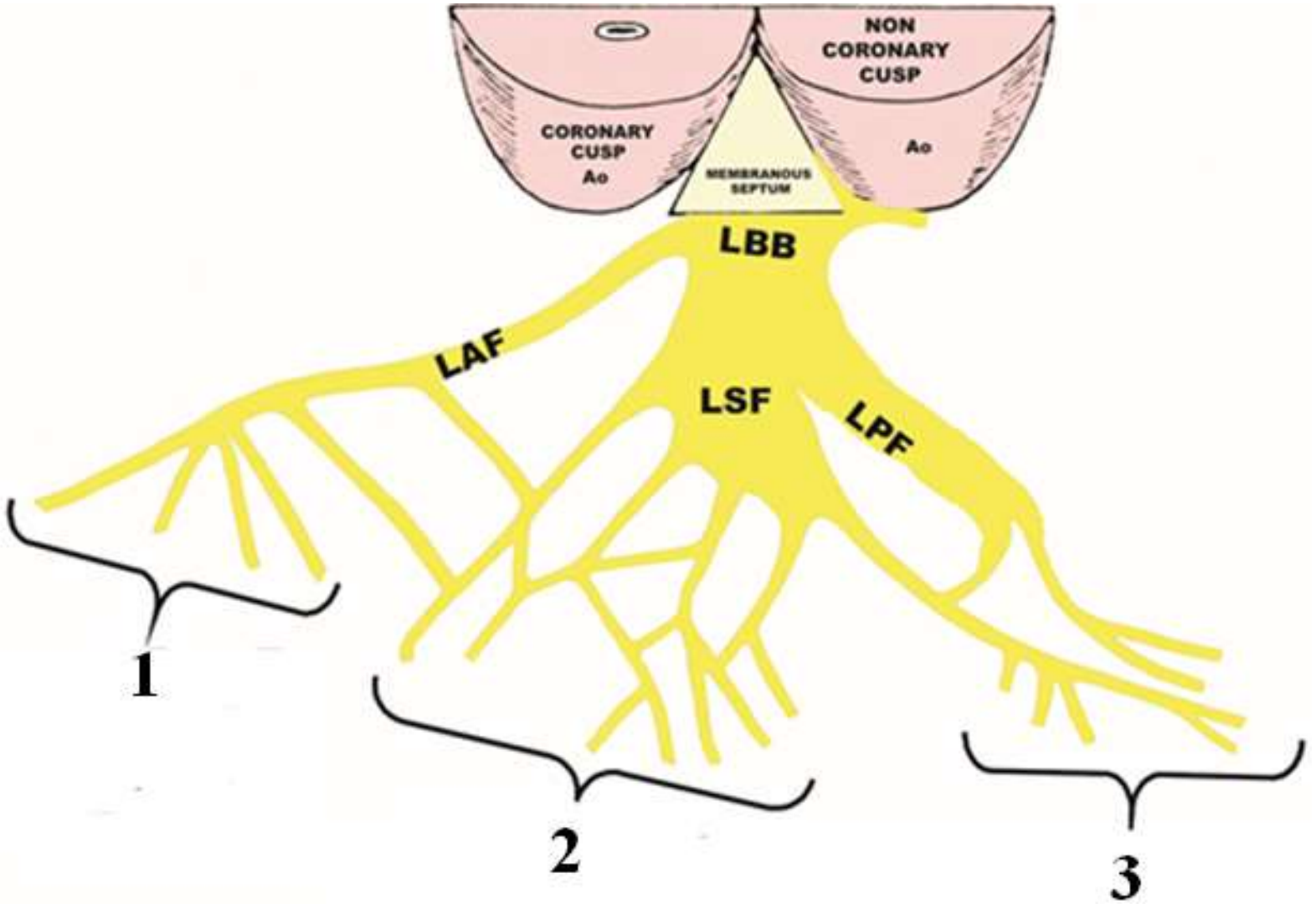


Type V: anatomic variation. The left septal fascicle is a interconnected network of Purkinje fibers that join the other two fascicles.



Type VI. Occurs in approximately 20% of cases. The left Hissian intraventricular system has only two fascicles: LAF and LPF

Ventricular distributions of the three fascicles (Modified from **Hecht 1973**)



1. **LAF:** it is distributed at the base of the anterolateral papillary muscle of the mitral valve, anterosuperior region of the septum and left ventricle anterolateral wall.
2. **LSF:** It is distributed in the apical, and centroseptal region and low septum
3. **LPF:** It is distributed at the base of the posteroinferior papillary muscle of the mitral valve, posteroinferior region of the septum and ínferobasal region of the left ventricle.

The Suano-Tawara concept

Anatomical, anatomopathological in humans and animals (**De Almeida 2015**), histological, histopathological (**Demoulin 1972;1973; Kulbertus 1976**), electrocardiographic, vectocardiographic (**Pérez-Riera 2008a, b; 2016**), ergometer test (**Uchida 2006**), and electrophysiologic (**Dhala 1996: Nogami 2011; Perrin 2012: Sung 2013**) studies have shown that the left bundle branch (LBB) divides into three fascicles or "fan-like interconnected network" in most human hearts. The LBB did not divide into two divisions without multiple interconnections (**Massing 1976**). The LBB originates at the crest of the muscular interventricular septum (IVS), just distal to the membranous septum. It arises in a fanlike fashion that descends inferiorly along the left ventricular septal surface beneath the noncoronary cusp of the aortic valve. The LBB usually branches into three major fascicles:

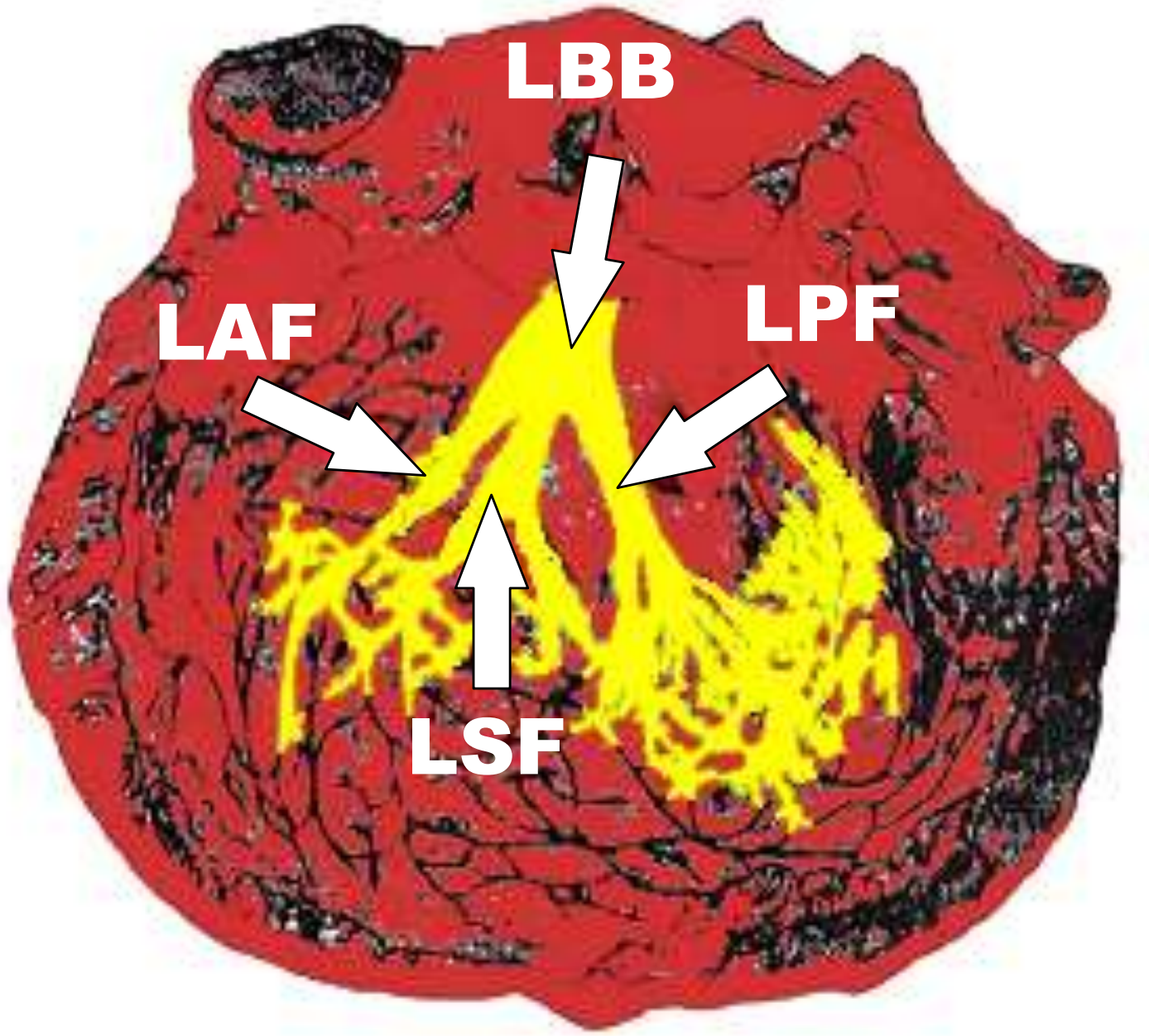
1) The left anterior fascicle (LAF) is directed to the base of the anterolateral papillary muscle of the mitral valve (ALPM);
2) Left posterior fascicle (LPF) is directed to the base of the posteromedial papillary muscle of mitral valve (PMPM);
3) Left septal fascicle (LSF): in approximately 85% of hearts, a central fascicle proceeds to the midseptal region with several morphologies. Approximately in 15% of cases, the left intraventricular system is bifascicular.

The LSF has been called with numerous names: left septal, third, left-middle, centro-septal fascicle, septal, medial division, left anterior-medial division, anterior-medial ramulus, anterior median branch of the LBB of His, and others. Since the end of the 19th Century, Dr. Whilhelm His Jr described the left Hissian intraventricular system as being trifascicular. He showed that a connective tissue sheet became a bundle connecting the upper and lower cardiac chambers, the bundle of His.

At the beginning of the 20th Century, Dr. Suano Tawara (**Tawara 1906**) clearly showed that anatomically, the trunk of the LBB splits into three fascicles. Tawara's pioneering work on the conduction system (figure in next slide): "The Conduction System of the Mammalian Heart" (1906), still serves as an invaluable reference for basic and clinical research .

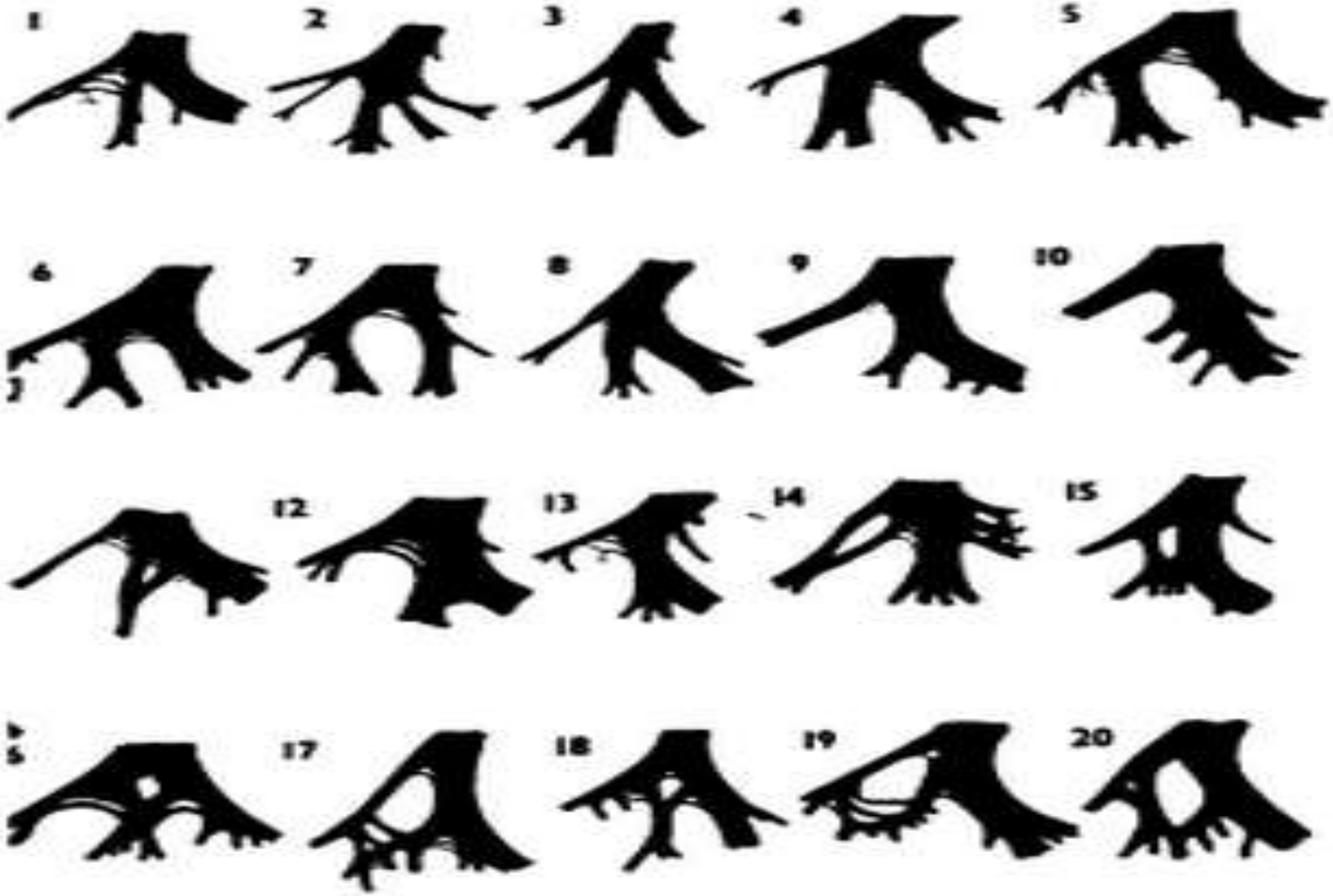
The trifascicular nature of the left His system

The trunk of the left bundle branch (LBB) of the His bundle split in three fascicles: LAF, LSF and LPF.

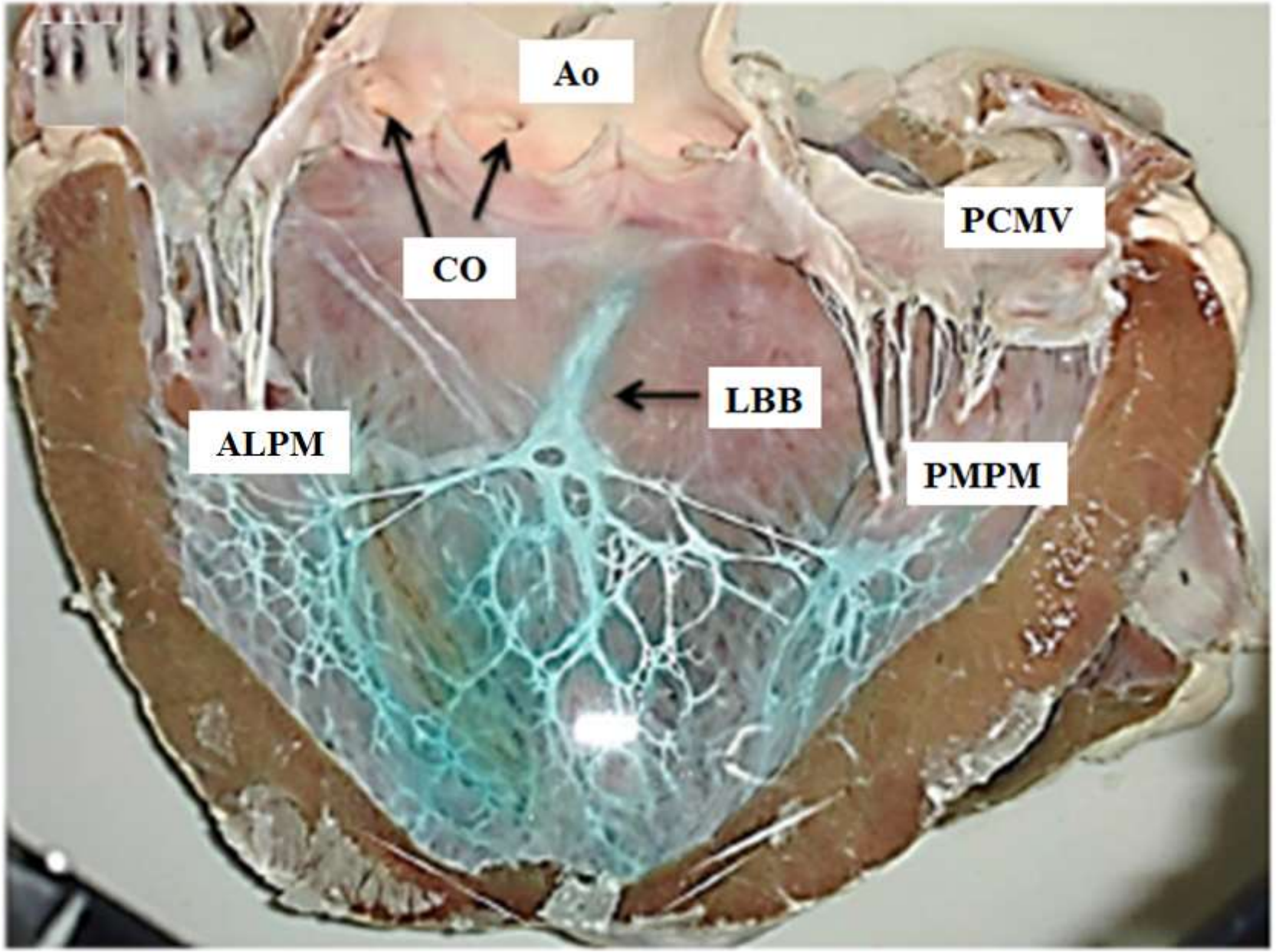


LBB: Left Bundle Branch
LAF: Left Anterior Fascicle
LPF: Left Posterior Fascicle
LSF: Left Septal Fascicle

The 20 normal left His system patterns in human hearts, following Demoulin and Kullbertus (Demoulin 1972;1973 and Kullbertus 1976)



Diagrammatic sketches of the left-sided conduction system as observed in 20 normal human hearts. In this figure we can clearly see 3 fascicles following the LBB (figure reproduced with authorization from American Heart Journal, published by Elsevier).



Ao – Aorta; **CO** – Coronary Ostium; **PCMV** – Posterior Cuspid of Mitral Valve; **LBB** – Left Bundle Branch; **ALPM** – Anterior Lateral Papillary Muscle; **PMPM** – Posterior Medial Papillary Muscle (Reproduced with permission of Anatomical Science International) (**de Almeida 2015**)

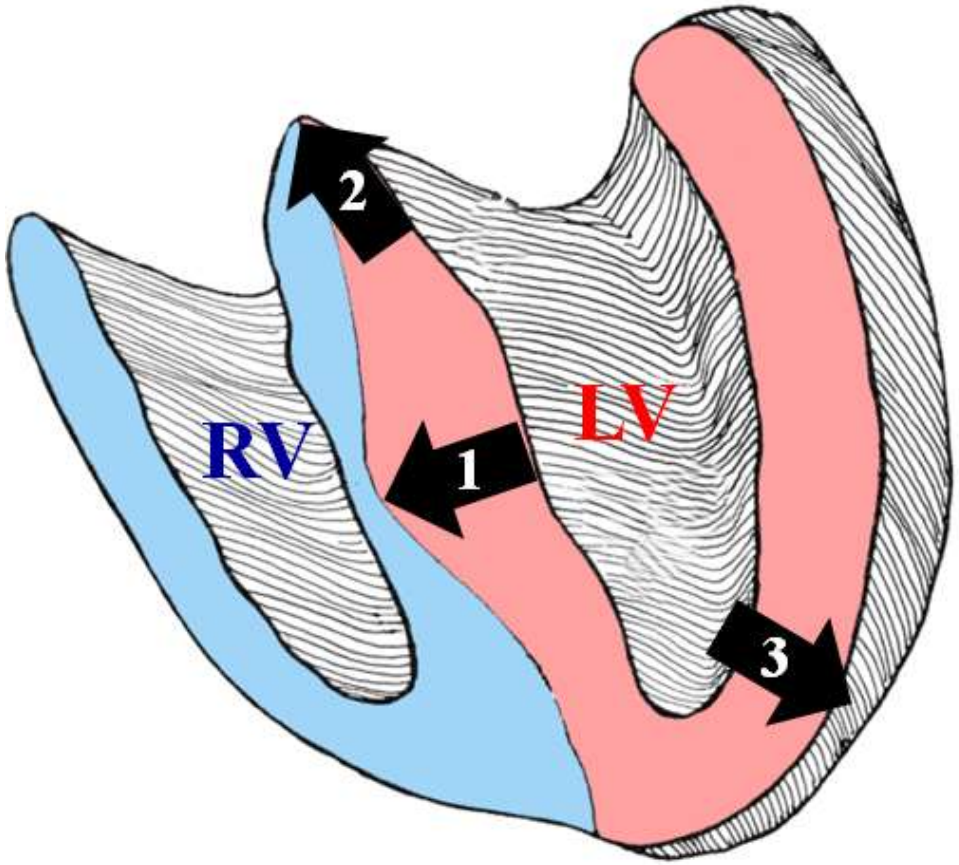
The Durrer concept

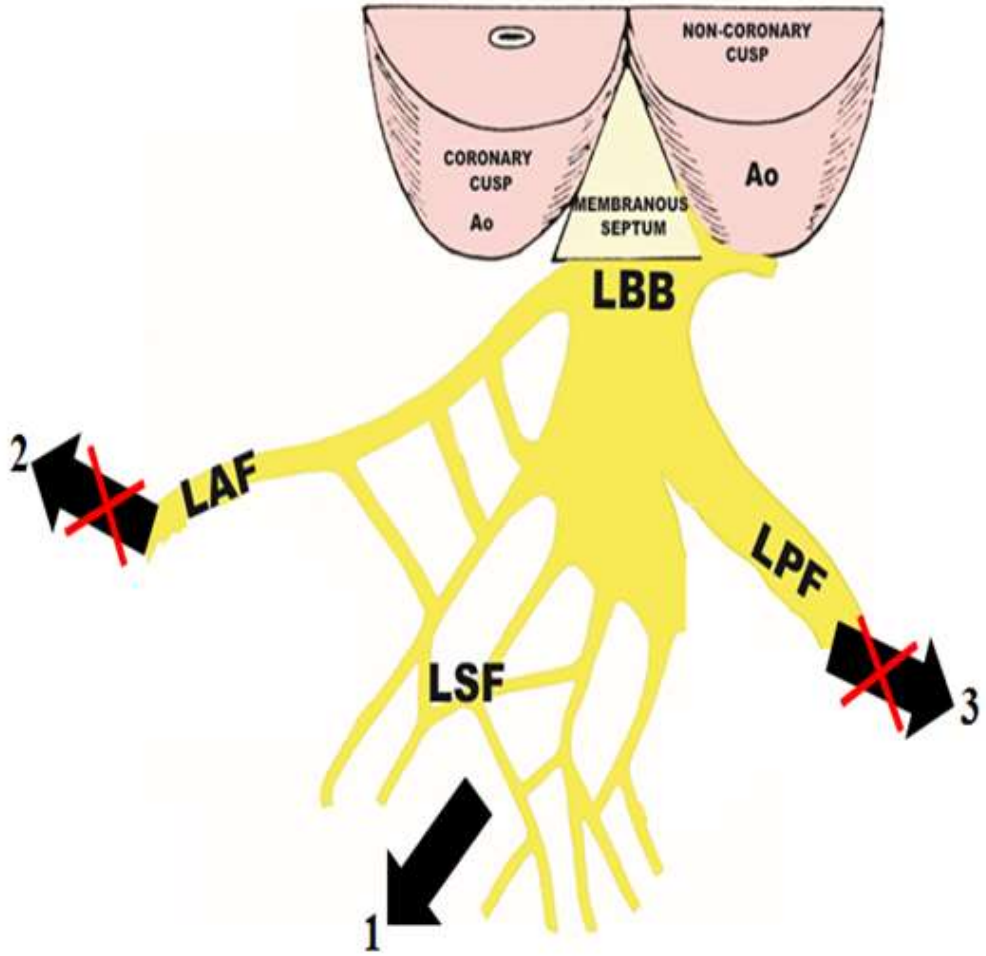
In 1970, Dr. Dirk Durrer et al (**Durrer 1970**) from the University Department of Cardiology and Clinical Physiology at Wilhelmina Gasthuis Hospital in Amsterdam, demonstrated in a classical manuscript, using 870 intramyocardial electrodes in isolated human hearts, that three endocardial areas are synchronously excited from 0 to 5 ms after the start of left ventricle (LV) activity potential (Figure 3). To obtain information concerning the time course and instantaneous distribution of the excitatory process of the normal human heart, the authors studied isolated human hearts from seven individuals who died from various cerebral conditions, but who had no history of cardiac disease. The first excited LV areas were:

- High on the anterior paraseptal wall just below the attachment of the ALPM where the LAF ends;
- Central on the left surface of the IVS where the LSF ends. Septal activation started in the middle third of the left side of the IVS, somewhat anteriorly and the lower third at the junction of the IVS and posterior wall. The normally functioning LSF, the left middle septum surface and the inferior two-thirds of the septum, originate the first vector (vector 1 or first anteromedial (1_{AM}) vector (**Peñaloza 1955**), and left inferior two-thirds of the IVS (second vector or vector of the inferior $^{2}/_{3}$ of IVS) (**Moffa 1996**);
- The posterior paraseptal wall is about one third of the distance from the apex to the base near the base of the PMPM, where the LPF ends. The posterobasal area is the last part of the LV to activate.

Note: This research might be considered as the beginning of the clinical electrophysiological setting for the stage of programmed electrical stimulation and registration of the human heart.

Sequential representation of normal initial ventricular activation (0 to 10 ms)



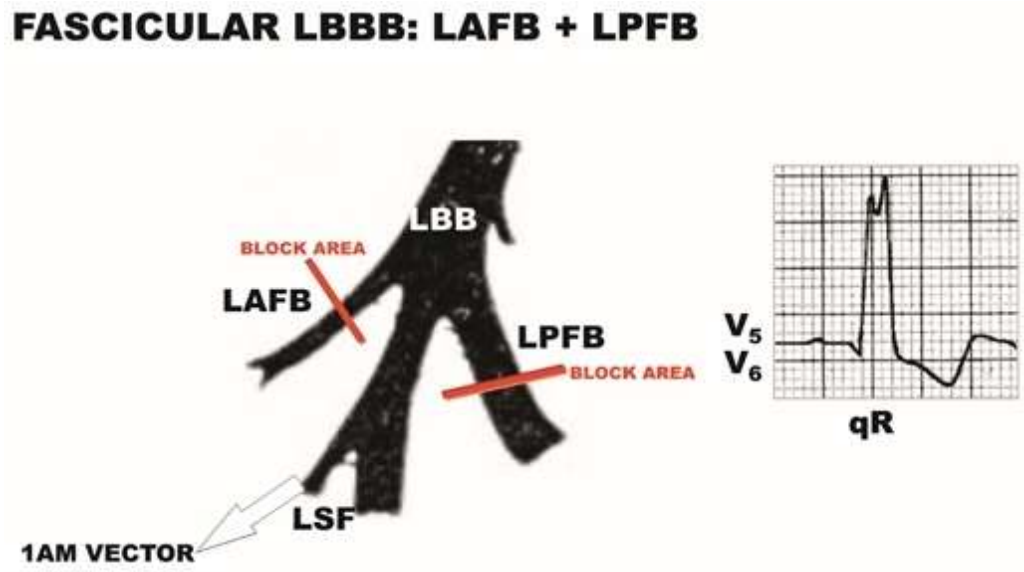


Initial ventricular despolarization from 0 to 10 ms
1) Central & apical region dependent of LSF 2) Anterosuperior region dependent of LAF 3) Posteroinferior region dependent of LPF

Note: In normal conditions vectors 2 and 3 have opposite directions, and consequently cancel each other. So, the only vector that expresses is vector 1 of the middle third of the IVS, vector 1_{AM} (anteromedial) or Tranchesi and Peñaloza vector which is directed forward and rightward (80% of cases) or leftward (remaining 20%).

Several anatomical, histological, pathological (**Demoulin 1972;1973. Kulbertus 1976**), electrocardiographic (**Riera 2008;2008**), vectorcardiographic (**Kullbertus 1976; Picollo 1980; Acunzo 2013; Pérez-Riera 2011**), body surface ECG (**MacAlpin 2012**), and electrophysiological (**Piccolo 1980; Dhala 1996; Perrin 2012**) publications have shown that the left bundle branch (LBB) splits into three fascicles or in a "fan-like interconnected network" in the vast majority of human hearts (**Mirvis 2014**) (approximately 2/3 of the cases in human hearts) (**Surawicz 2009**). There are multiple anatomic variations of the left septal fascicle that may depart from the other left fascicles (mainly the left posterior fascicle) and none from the main LBB and even may be absent. The left His system is trifascicular with a left anterior, a left posterior, and a left septal fascicle. Consequently, the classic term "hemiblock", to describe the block of one of the fascicles, established several decades ago by Rosenbaum's school, should be updated. Electrovectorcardiographic changes resulting from conduction abnormalities of the left anterior and left posterior fascicles are commonly diagnosed, mainly by their changes in the frontal plane. However, the existence of conduction defects of the left septal fascicle remains controversial. The ECG/VCG hallmark of the left septal fascicular block is prominent anterior QRS forces (PAF) on the horizontal plane. Proof of transient LSFB (**Riera 2008; Pérez-Riera 2015; 2106; 2017**), as part of the requisites to recognize a new ECG dromotropic disturbance, is considered mandatory. There were cases of transient ischemic LSFB in literature (associated with proximal obstruction of the left anterior descending coronary artery before the first septal perforating branch or ischemia triggered during an exercise stress test (**Uchida 2006**), additionally we described a phase-4 rate-dependent or bradycardia-dependent mechanism of LSFB. Rate dependent blocks are the best models to study any new conduction disturbances in the conducting tissue, as they are free from possible "contaminants" associated with transient injuries to the surrounding tissue, as it happens in ischemia (**Ibarrola 2014**). The ECG/VCG phenomena of prominent anterior QRS forces should be distinguished from other conditions that also produce anterior QRS shift in the HP as: normal variants (athlete heart and CCW rotation around longitudinal axis), right ventricular hypertrophy (types B and A VCG), misplaced precordial leads (**MacKenzie 2004**), lateral myocardial infarction (ancient dorsal), RBBB, type A Wolff-Parkinson-White preexcitation (type A has a positive delta wave and QRS complexes are predominantly upright in the precordial leads with R/S > 1 in V_1). The dominant R wave in lead V1 may be misinterpreted as RBBB, obstructive and non-obstructive forms of hypertrophic cardiomyopathy, diastolic left ventricular hypertrophy, combined or biventricular hypertrophy, endomyocardial fibrosis, Duchenne muscular dystrophy, dextroposition or pseudo dextrocardia, left septal fascicular block or a combination of the above. The two highly frequent etiologies of LSFB are: coronary artery disease (CAD) with critical proximal obstruction of the left anterior descending coronary artery before the first septal perforating branch, and in Latin America, chronic Chagasic cardiomyopathy. Another strong argument is raised by the electrophysiological explanation of "atypical" LBBB cases. There are divisional or fascicular LBBB cases (LAFB + LPFB) that display a q wave in the left leads, making the electrocardiographic pattern of LBBB atypical. Rosenbaum called them "left intraventricular blocks without changes in the initial part of QRS" and, Alboni (**Alboni 1976**).

"LBBB with normal septal activation". At first, this colossal work did not find an explanation for these cases, and state in the above mentioned book, that they are "difficult to explain" (**Rosenbaum 1968**). Three years later Medrano et al (**Medrano 1970**) (Figure below) proposed that in these atypical LBBB cases, the fibers of the LSF would originate prior to the site or area of the block in the LPF and LAF, so the middle-septal activation is preserved (1_{AM} vector); this explained the q waves of the left V5-V6 leads, and turned the LBBB into an atypical one.. The same explanation was provided by Alboni six years later (**Alboni 1976**).



There is conclusive evidence of a left human trifascicular His system in at least two thirds of people (**Surawicz 2009**). The isolated left septal fascicular block has been described by several authors using different methodologies (**Mirvis 2014**). Traditional teaching does not include the concept of a trifascicular left system. The authors provided the current acceptable terminology and definitions for electrovectorcardiographic diagnosis of left septal fascicular block. All criteria are validated in absence of RVH, lateral MI (previously called dorsal), septal hypertrophy and other miscellaneous causes of prominent anterior QRS forces. Consequently, isolated LSFB diagnosis must always be clinical-electrovectorcardiographic. The following criteria are proposed by the Guidelines of the Brazilian Society of Cardiology on the Analysis and Issue of ECG Reports: R voltage in V2-V3 ≥ 15 mm, “in crescendo” R voltage in the intermediate precordial leads and decreasing from V5 to V6, QRS duration < 120 ms, absence of QRS shift in the FP and frequently negative T waves in the right precordial leads (**Pastore 2009**). Prominent QRS anterior forces may have many causes including LSF block. A quadrifascicular(**Hoffman 2012**) conception of the intraventricular conduction system should ultimately prevail. The trifascicular model is simple, but simpler than true (**Perrin 2012**). A call is made to the international electrocardiographic societies to generate a position paper or consensus to unify nomenclature and definitions.

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