Adult female with instable angina, several cardiovascular risk factors and intermittent Left Bundle Branch Block

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English: Case report

- Female 52-year-old
- History of stable angina for 8 years related to physical strain
- Progression in recent months with more frequent events to minor strain
- Before admission, she presented more prolonged episode accompanied by syncope
- Risk factors: hypertension, DM2 and smoking
- She takes: aspirin, simvastatin 40 mg and glybenclamide 5 mg
- Normal electrolytes and biomarkers

Questions:

1. Which is the clinical-electrocardiographic diagnosis?
2. Which is the most appropriate approach?

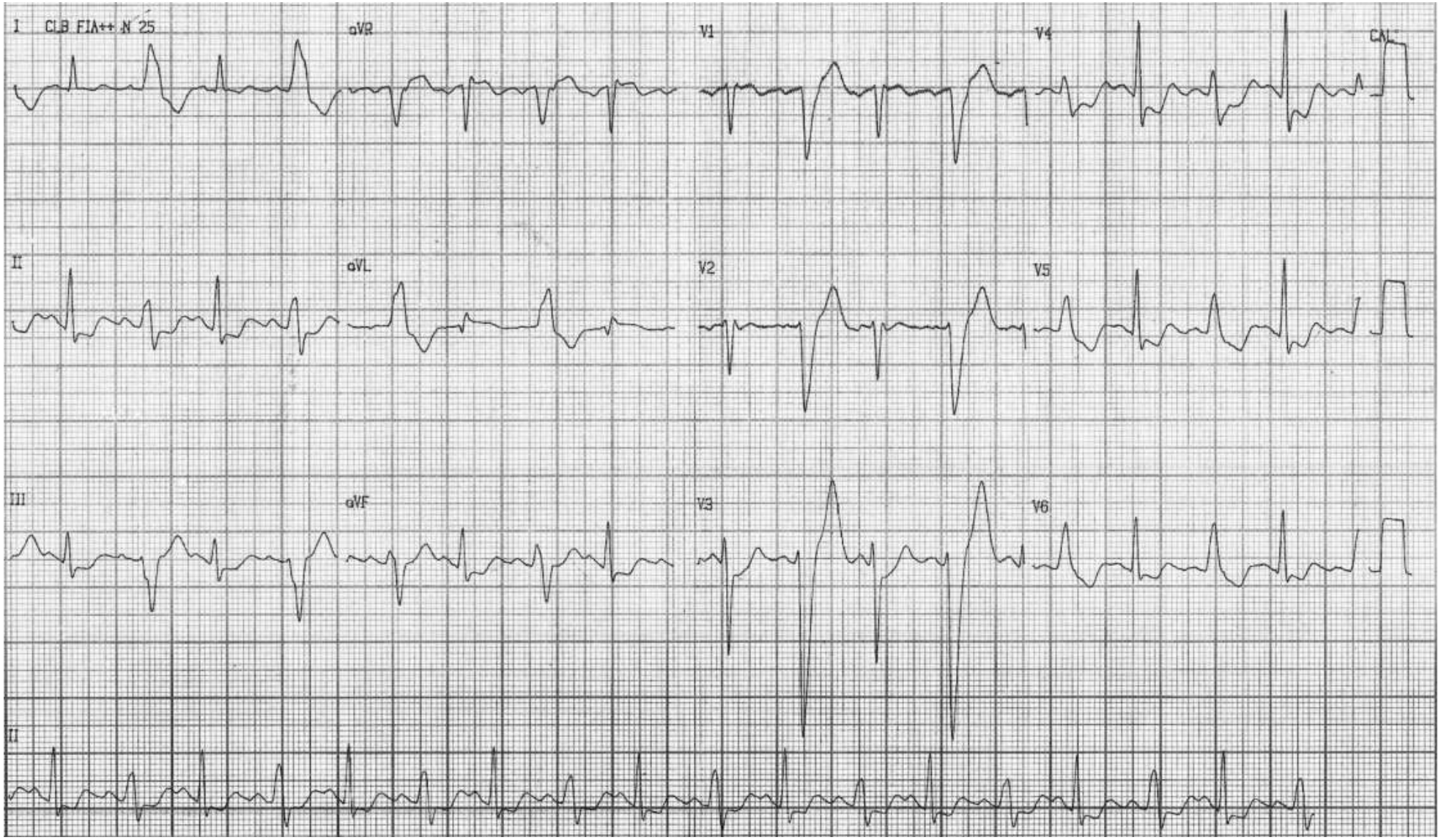
Portuguese: Relato de caso

- Feminino, 52 anos
- História de angina estável há 8 anos relacionada aos esforços físicos
- Progressiva nos últimos meses com episódios mais frequentes aos esforços menores
- Apresentou antes da admissão episódio mais prolongado acompanhado de síncope
- Fatores de risco: Hipertensão , DM tipo 2 e tabagismo
- Faz uso de: aspirina, sinvastatina 40mg e glibenclamida 5mg
- Eletrólitos e biomarcadores normais

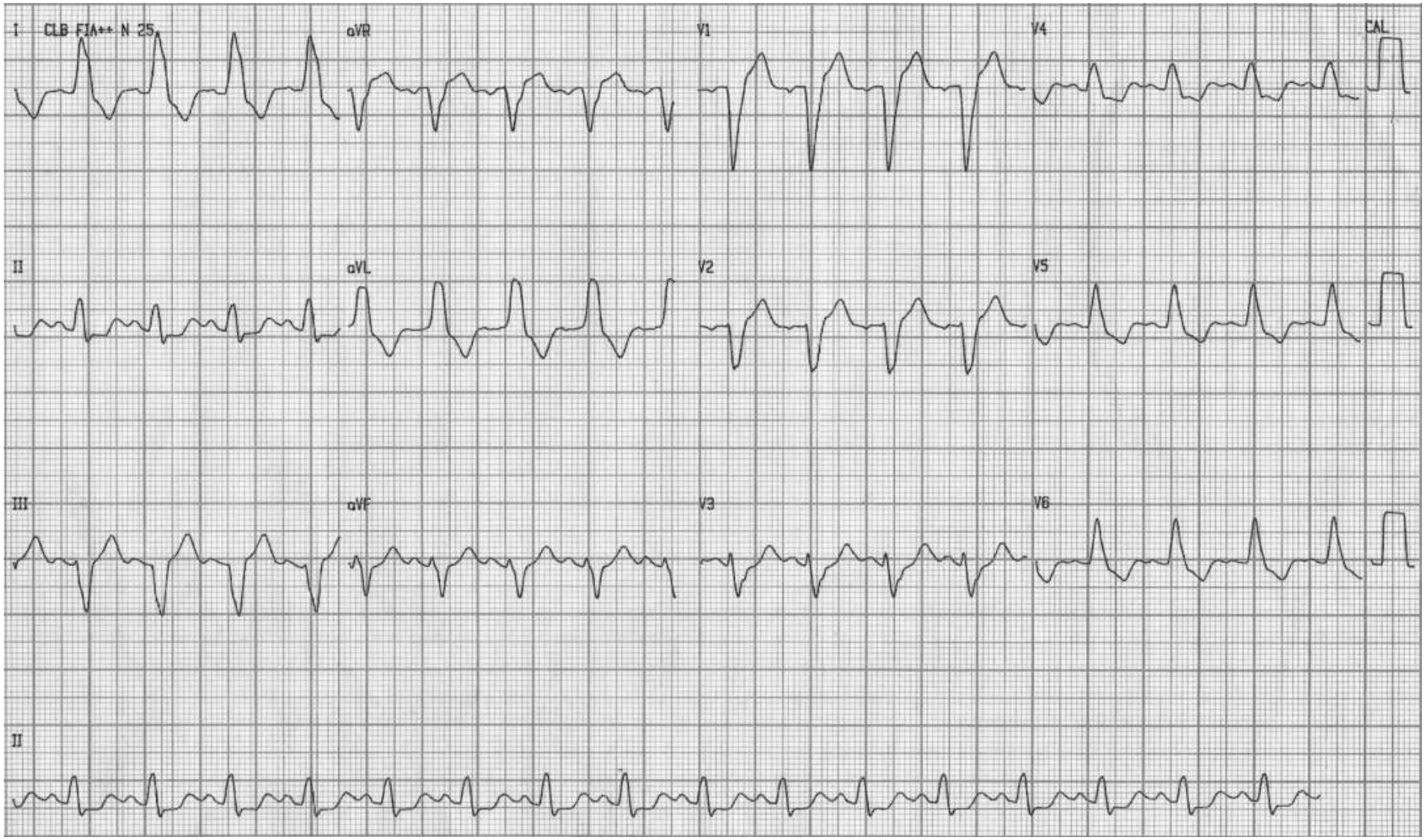
Perguntas:

1. Qual o diagnóstico clínico-eletro-cardiográfico?
2. Qual a abordagem adequada?

With pain / Com dor



Without pain / Sem dor

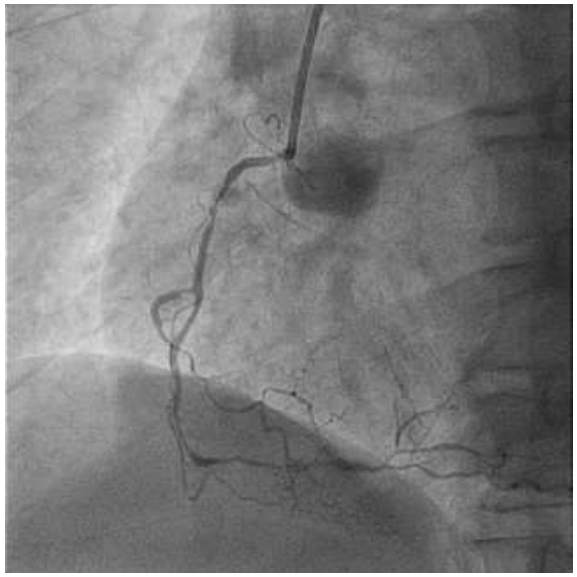
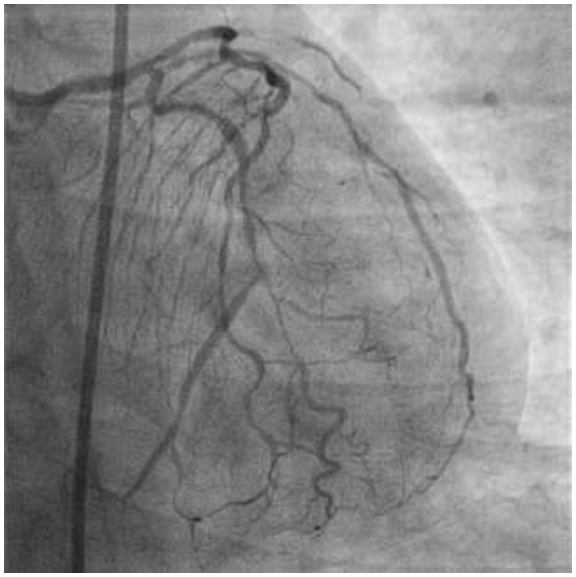
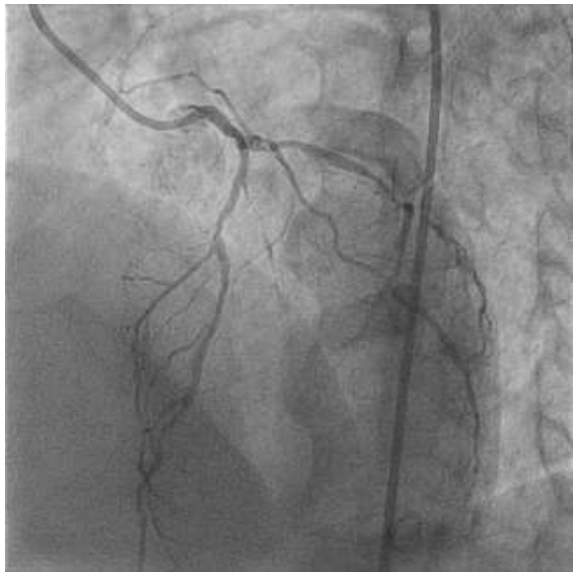
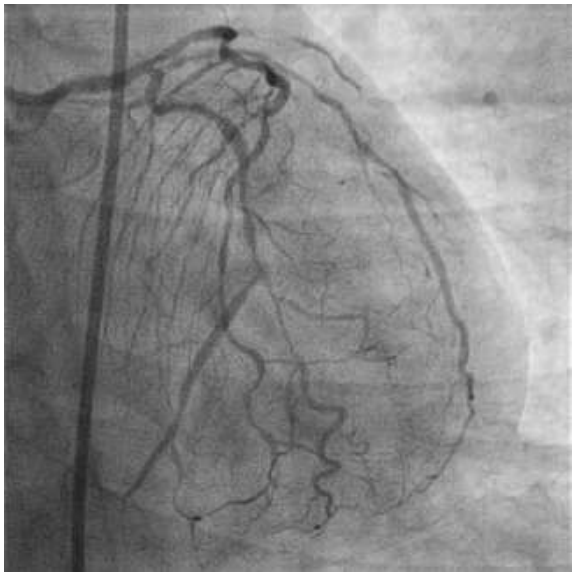


Coronary angiography

- **LMCA = 40% distal**
- **ADA=60% ostial**
- **First diagonal artery: 100% in the middle third**
- **Cx: 80% middle third**
- **Left Circumflex marginal artery = 80% proximal third**
- **RCA = 80% proximal third**
- **Collateral circulation**

Portuguese

- TCE= 40% distal
- DA=60% ostial
- Primeira diagonal: 100% no terço médio
- Cx:80% terço médio
- Mg.Cx.= 80% terço proximal
- CD= 80% terço proximal
- Circulação colateral.

Colleagues opinions

Hello.

Regarding the case ” Adult female with unstable angina, several cardiovascular risk factors and intermittent LBBB”, it seems that she has signs of severe coronary artery disease in the beats with narrow QRS recorded during pain (ST elevation in aVR, widespread ST depression maximal in V4-V5). Could be 3-vessel disease or left main disease. I have no electrophysiological explanation for the intermittent LBBB, probably not supernormal conduction? She should have coronary angiography without delay.

Best regards

Kjell Nikus

Kjell Nikus, MD, PhD, Doctor, Cardiology Department, Heart Hospital, Tampere University Hospital, Tampere 33520, Finland

- He has 140 indexed Pubmed manuscripts
- His main study focus is coronary heart disease and its electrocardiographic manifestation
- He is one of the world's most recognized researchers in coronary artery disease and ECG.

Dear Andres and Raimundo,

Thank you for sending this interesting case.
Using the calipers, I think the RR intervals of the narrow complexes are the same as the wide complexes. The PR interval is the same with narrow and wide complexes. I think this is alternate aberration into the left bundle.
Since there is a clear P wave in front of narrow and wide QRS complexes, it fits alternate LBB aberration, and not PVCs.
Looking forward to receiving your comments.

Best regards,
Mohammad

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Maya Smith
Assistant to Dr. Mohammad

Dear Andrés and Raimundo,
Very interesting case of a 52 year old woman with chronic stable angina pectoris.
The ECG 'with pain' shows sinus tachycardia (~120 bpm) with alternating QRS morphology and durations due to 2:1 LBBB. The narrow QRS complexes show evidence of circumferential subendocardial ischemia with ST segment elevation in aVR and ST segment depression in II, III, aVF, V3-6. This ischemic ST segment pattern is usually associated with partial left-main coronary occlusion and /or severe multivessel coronary artery disease.
The ECG 'without pain' shows sinus tachycardia (~110 bpm) and complete LBBB. Of interest, the heart rate, as expected, is slightly slower than when not in pain, but all the QRS complexes show LBBB. I still think this a tachycardia-dependent LBBB where the left bundle branch starts to fail at heart rates around 100-120 bpm. It is generally true that in tachycardia-dependent bundle branch block the bundle branch fails as heart rates accelerate at a higher heart rate threshold and recovers conduction at a lower heart as the heart rate decelerates (so-called 'hysteresis effect').
From a management perspective, she should undergo coronary angiography and receive the appropriate revascularization strategy based on coronary anatomy.I look forward to your comments and those of our colleagues.
Warmest personal regards,

Frank G. **Yanowitz, MD**
Professor of Medicine
University of Utah School of Medicine
Cardiologist, LiVe Well Center
Intermountain Healthcare
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Professor Frank is a renowned educator in the field of electrocardiology. We are honored to have his friendship.
Andrés and Raimundo

Português

A paciente apresenta bloqueio de ramo esquerdo(BRE) intermitente durante a dor, e o mesmo se torna persistente sem dor. Os complexos QRS largos durante a dor parecem apresentar alteração isquêmica de repolarizacão. No ECG com dor o intervalo PR com complexos QRS largos parece se manter semelhante aos intervalos PR de complexos QRS estreitos. Uma explicação possível para os QRS estreitos seria concomitante retardo da condução pelo ramo direito ao BRE já instalado. Assim, o padrão de QRS estreito se deveria a um bloqueio intra-Hissiano acometendo os dois ramos de forma que um compense o outro, estreitando o QRS. Pensaria encontrar na cinecoronariografia uma lesão proximal localizada na artéria descendente anterior próxima do primeiro ramo perfurante septal

Provavelmente a seguir realizaria um estudo eletrofisiológico para confirmar minha hipótese.

Forte abraço

Bruno **Valdigem**

valdigem@gmail.com

English

During chest pain the patient has intermittent left bundle branch block (LBBB), and it becomes persistent without pain.

During pain, wide QRS complexes appear to show ischemic repolarization pattern.?

In ECG with pain the PR interval with wide QRS complexes seems to remain similar to the PR intervals of narrow QRS complexes.

A possible explanation for narrow QRS complexes would be concomitant delayed conduction on right bundle-branch, thus, the narrow QRS pattern should be due to an intra-Hissian block involving the two branches(LBB and RBB) so that one compensates for the other, narrowing the QRS.

I think we will find a proximal lesion in the LAD artery next to the first septal perforator branch(S_1)

I would do an invasive electrophysiological study in order to confirm my hypothesis

Big hug

Bruno **Valdigem M.D.PhD**.

Clinical and Invasive Electrophysiologist of the Albert Einstein Hospital. Arrhythmia Center Assistant Physician of the Electrophysiology Sector of the Dante Pazzanese Institute of Cardiology. Doctor of Science, Federal University of São Paulo. Member of the Editorial Board of Medscape in Portuguese.

This is a 52 year old female with unstable angina and syncope.
Her ECG without pain shows a left bundle branch block. During pain she has alternating LBBB with normally conducted QRS complexes. In addition, she has ST depression in the antero-lateral and inferior leads that are more pronounced in the QRS complexes without LBBB. Cardiac memory should have resulted in opposite T wave changes in lateral leads. This case shows how LBBB can partially obscure the ECG manifestations of ischemia.
I would recommend cardiac catheterization with coronary angiography.

Mario D. Gonzalez, MD
Penn State Hershey Heart and Vascular Institute
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Dear Andres and Raimundo,

Thank you for sending this very interesting case. In the ECG labeled “With pain”, there are wide and narrow QRS complexes on alternate beats. All the beats are preceded by P waves and a constant PR interval, so that the wide QRS complexes represent LBBB, rather than ventricular beats. In support of this, the beats with the narrow QRS complexes have small Q waves in Leads I, II, III, aVL and aVF and these Q waves are absent in the beats with the wide QRS complexes. In addition, the beats showing the LBBB have another important feature – their QRS complexes in Leads II, III and aVF terminate in prominent S waves. This suggests that the LBBB is due to post-divisional, rather than the more common pre-divisional block. Evidence of conduction disturbances in both the anterior and posterior fascicles of the left bundle branch suggests more extensive conduction system disease than would be the case if the block were confined to a circumscribed lesion above the bifurcation of the left bundle branch into its two fascicles. This pattern is confirmed in the tracing labeled “Without pain” in which all the beats show post-divisional LBBB.

Returning the “With pain” ECG, the narrow QRS complexes also have terminal S waves in Leads II, III and aVF. These S waves are not deep enough too produce marked left axis deviation, but I believe that they nevertheless indicate the presence of left anterior hemiblock. This is confirmed by examining the terminal R waves in Leads aVR and aVL, in which the timing of the peak of the R wave in aVL precedes that of the peak of the R wave in aVR. These findings are consistent with left anterior hemiblock in the beats with the narrow QRS complexes.

(Warner RA, Hill NE, Mookherjee S, Smulyan H. Improved electrocardiographic criteria for the diagnosis of left anterior hemiblock. Am. J. Cardiol. 51:723-726, 1983. and (Warner RA, Hill NE, Mookherjee S, Smulyan H. Electrocardiographic criteria for the diagnosis of combined inferior myocardial infarction and left anterior hemiblock. Am. J. Cardiol. 51:718-722, 1983.)

In the “Without pain” ECG, the consistent appearance of the post-divisional LBBB may be rate dependent. In any case, because of the occurrence of syncope in conjunction with ECG evidence of extensive intraventricular conduction system disease, I would recommend the insertion of a permanent pacemaker. Unless the patient has evidence of left ventricular systolic dysfunction, e.g. as evidenced by an abnormally low ejection fraction by echocardiogram, a single-lead permanent pacemaker would probably suffice.

Sincerely,

Bob Warner, MD

Final comments by

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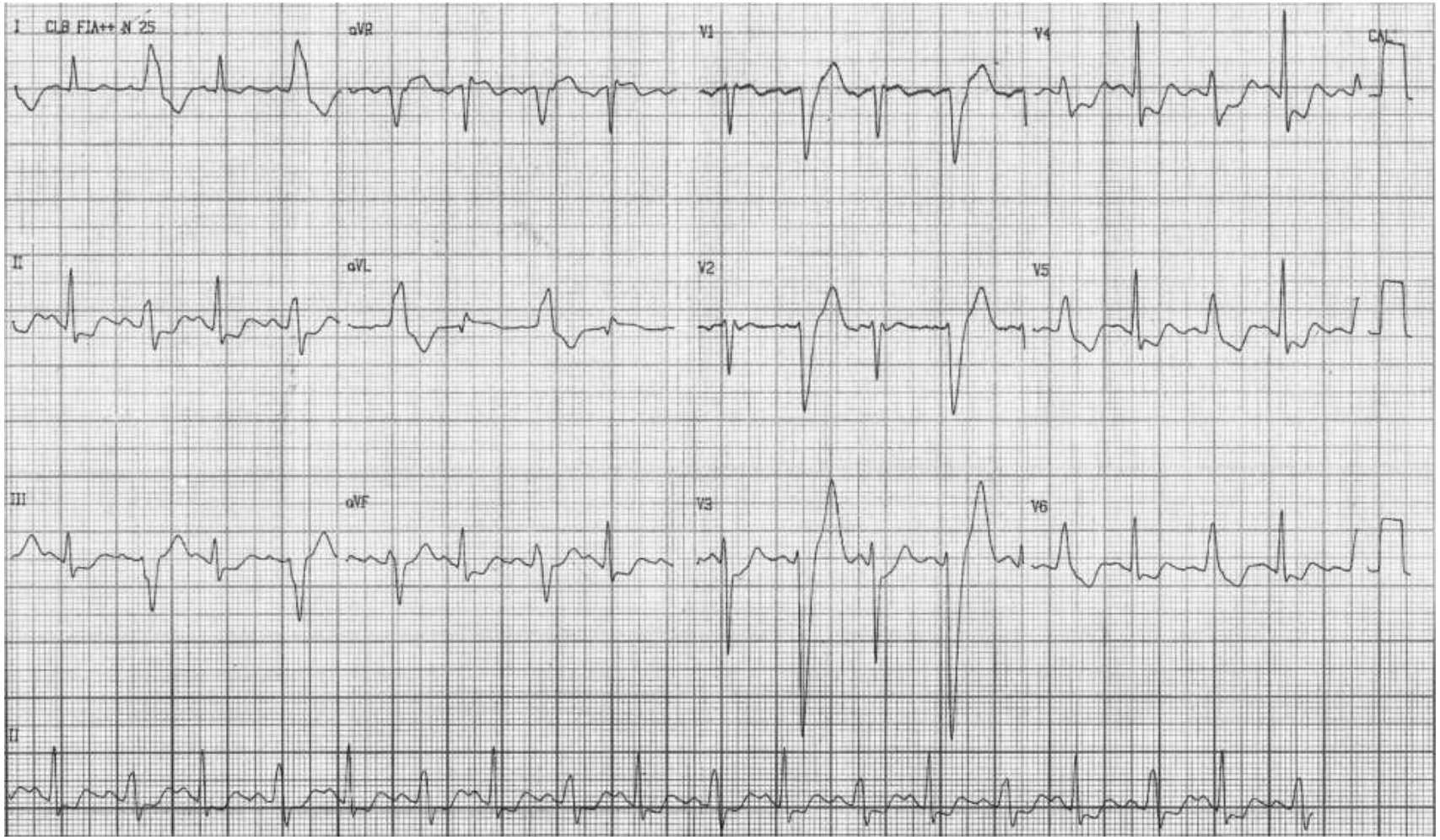
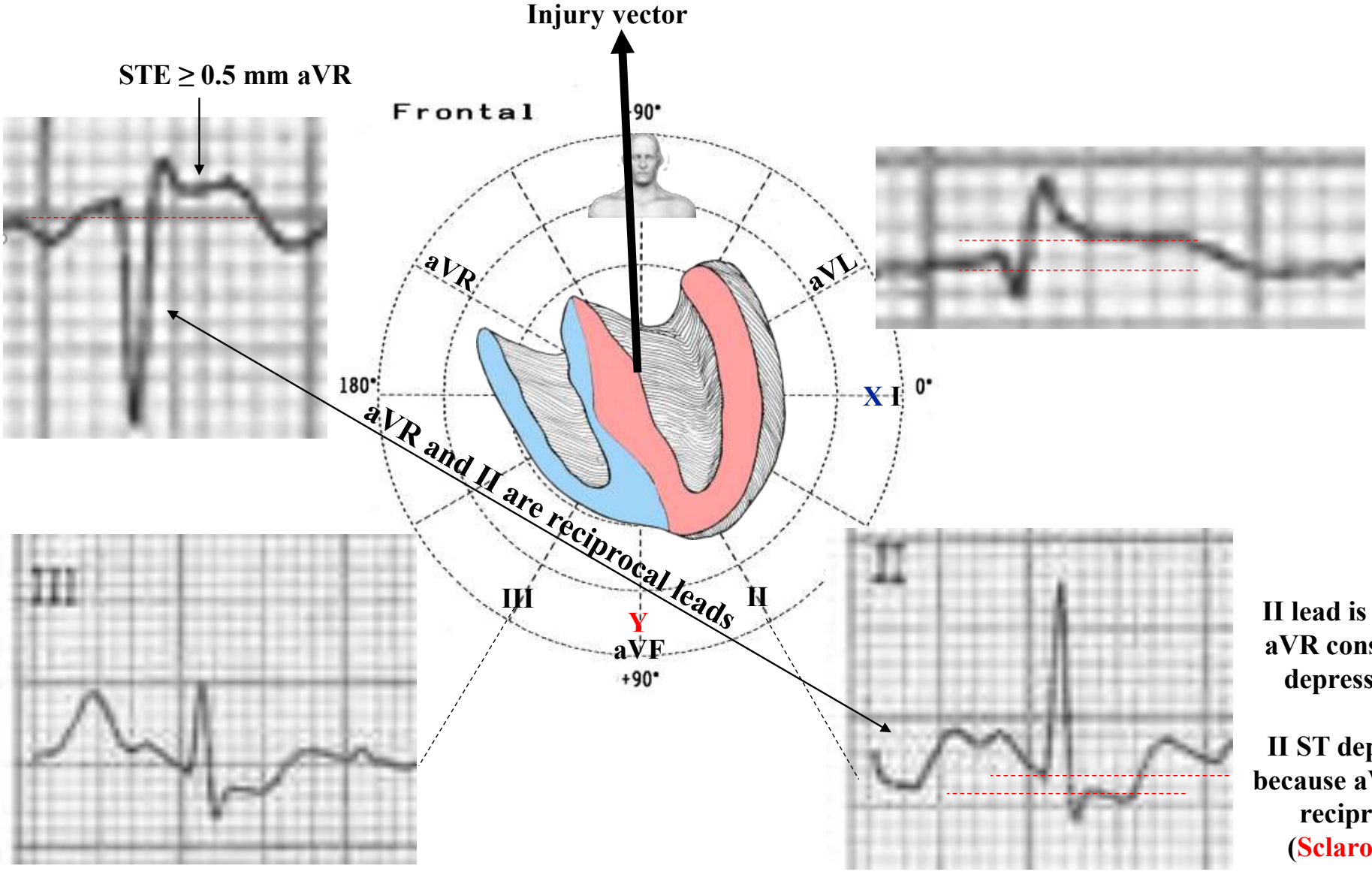
With pain / Com dor



ECG diagnosis: sinus tachycardia, HR 120 bpm, alternating 1:1 narrow and wide (LBBB) QRS complexes. The QRS complexes without LBBB (narrow) show ST segment depression in inferior leads, and from V3-V6 with concomitant ST-elevation in aVR. This ischemic ST segment pattern is usually associated with partial or subocclusion LMCA, proximal LAD or equivalent severe multivessel disease (see ludic explanation in the next two slides)

Multivariate analysis showed that ST-segment elevation in lead aVR of ≥ 0.5 mm was the strongest predictor of LMCA/ three-vessel disease (3-VD) (**Kosuge 2005**). ST-segment elevation in lead aVR of ≥ 0.5 mm and positive troponin T on admission are useful predictors of LM/3-VD.



II lead is reciprocal to aVR consequently ST depression II > III

II ST depression >III because aVR and II are reciprocal leads (Sclarovsky 2002)

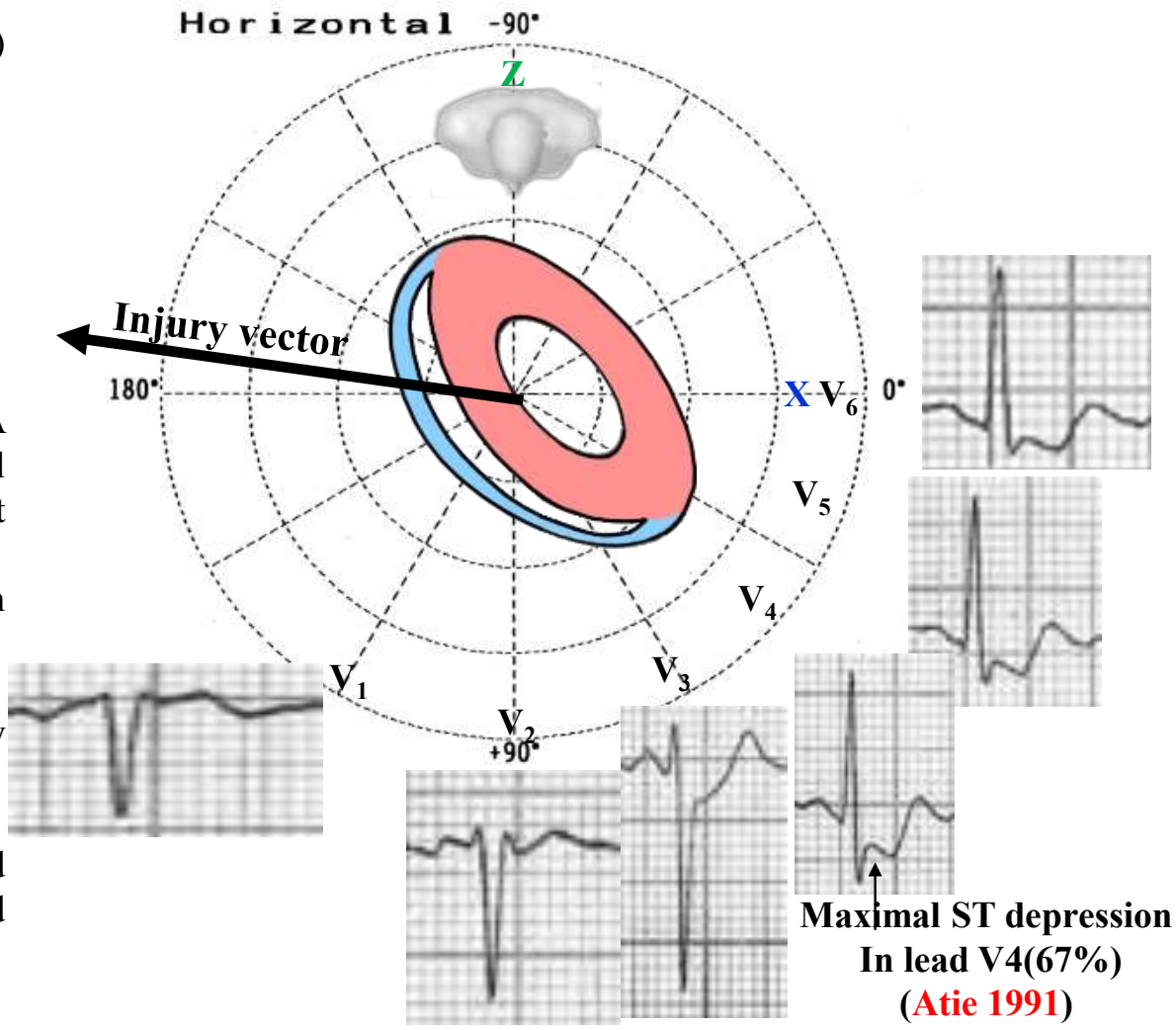
These ECG modifications are predictors of LMCA subocclusion or equivalent or three-vessel disease (3-VD) in patients who have acute coronary syndromes with non–ST-segment elevation (**Nikus 2010; Kosuge 2005; Nikus 2011: Birnbaum 2012**).

Clinical scenarios with possible similar ECG patterns
Common pitfalls (Birnbaum 2012)

- Aortic stenosis (LVH)
- Tachycardia
- Anemia
- Fever
- Hypertension

Rare pitfalls (Gotzmann 2009)

- Mediastinal tumors Acute LMCA stenosis due to a mediastinal tumor can be treated by direct stenting.
- Left main coronary artery spasm (**Rumoroso 1995**)
- Cocaine user (**Mongeon 2008**)
- Traumatic coronary artery dissection (**Harada2002**)
- Takayasu's arteritis (**Amir 2006**)
- Hibernating myocardium caused by isolated, radiation induced LMCA stenosis (**Ellis 1997**)

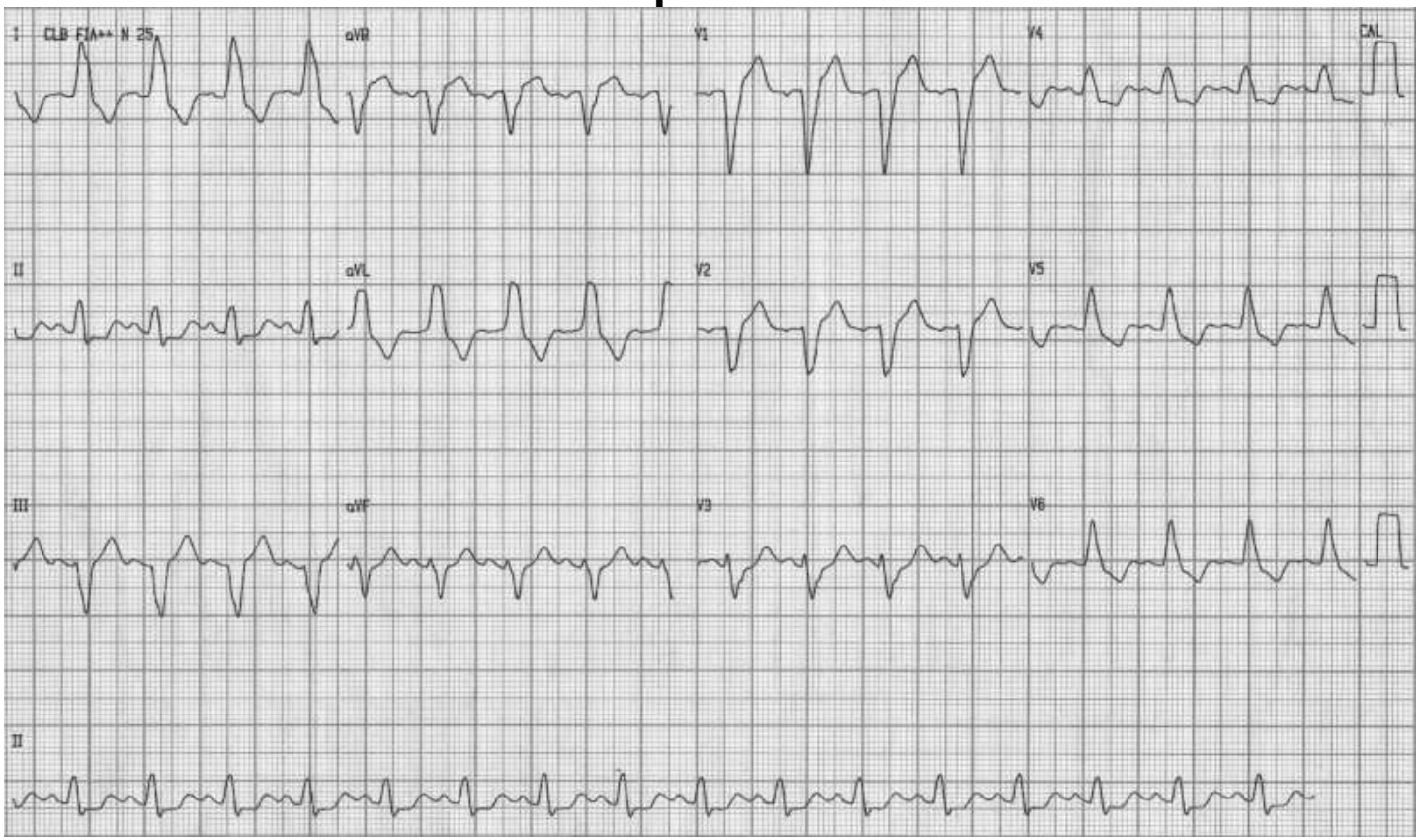


LMCA stenosis by vascular pathologies

Pathology and cause	Diagnostic tests	Treatment
Spasm		
LMCA spasm	Intracoronary nitroglycerin reduces LMCA stenosis	Nitroglycerin, nitrates, calcium channel blockers; percutaneous coronary stenting; perhaps CABG
Dissection		
LMCA dissection†	Intravascular ultrasound, computed tomography	Aspirin, other antiplatelets; nitrates, beta-blockers; percutaneous coronary stenting; CABG
Inflammatory vasculitis		
Takayasu's arteritis (**Amir 2002**)	Medical history, complete blood count, CRP, ESR, histopathological examination	Aspirin, steroids, immunosuppressants; CABG
Kawasaki's disease (**Prêtre 2000**)	Medical history, CBC, CRP, ESR, anti-endothelial cell antibodies; angiogram (stenosis and aneurysms); histopathological examination	Aspirin, immunoglobulins; percutaneous coronary stenting; CABG
Giant-cell arteritis (**Mitnick 1990**)	Medical history, CBC, CRP, ESR, histopathological examination	Steroids, immunosuppressants; CABG
Iatrogenic		
Radiation-induced (**Ellis 1997**)	Medical history	CABG

CABG, coronary artery bypass grafting; CBC, complete blood count; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; LMCA, left main coronary artery. For example:* idiopathic (**Rumoroso 1995**), catheter-induced (**Takahashi 1996**), cocaine-induced (**Mongeon 2008**); †spontaneous (**Missouris 2000**), traumatic (**Harada 2002**).

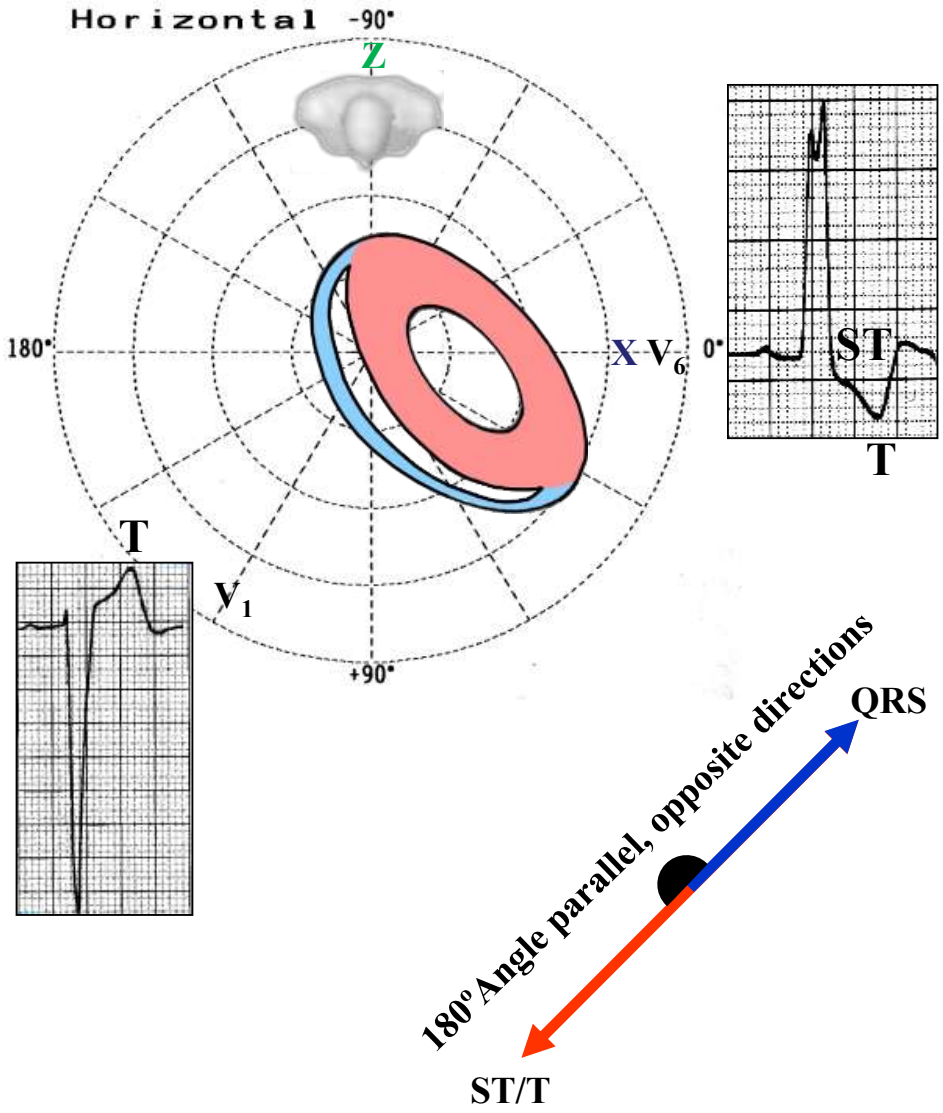
Without pain / Sem dor

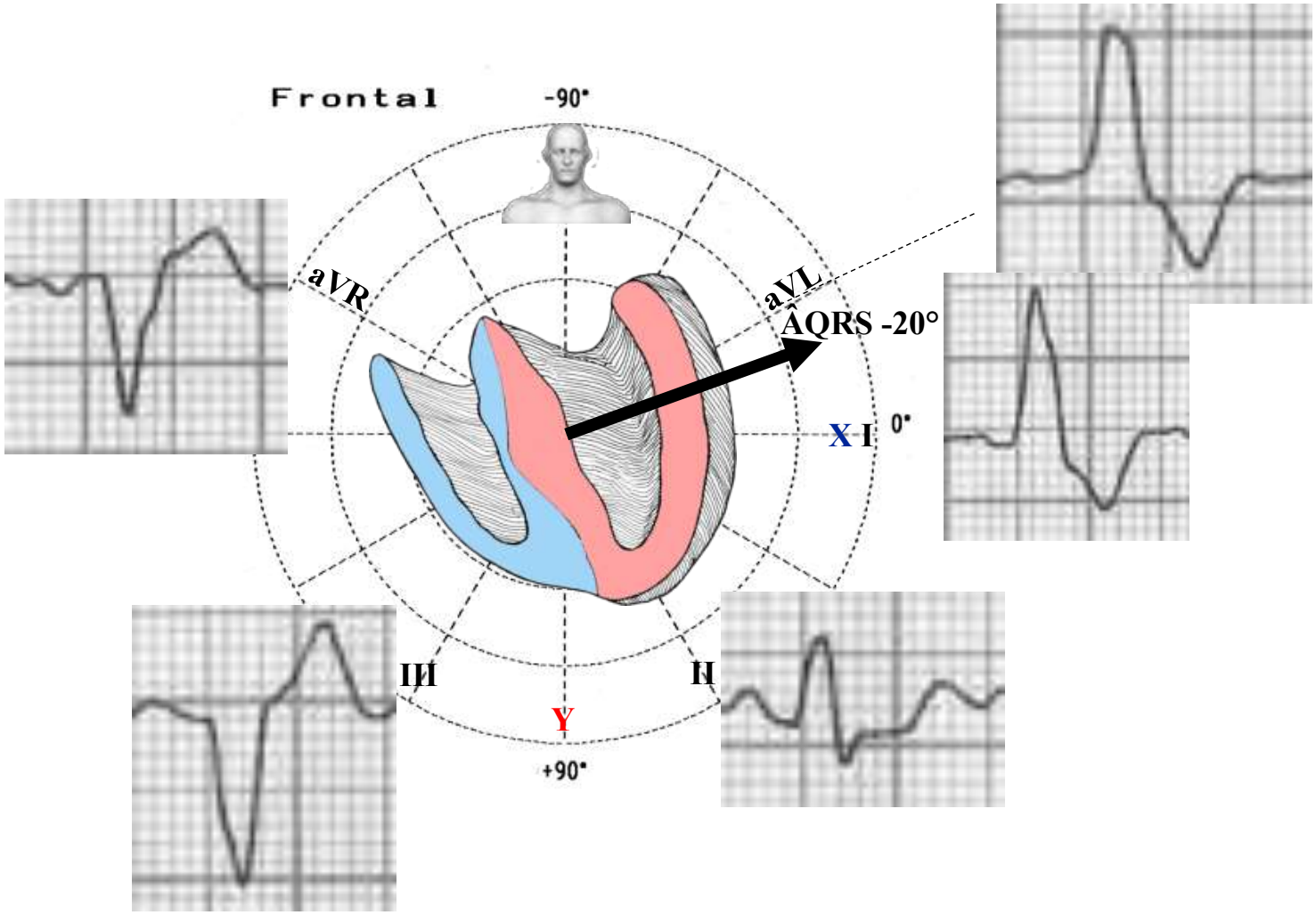


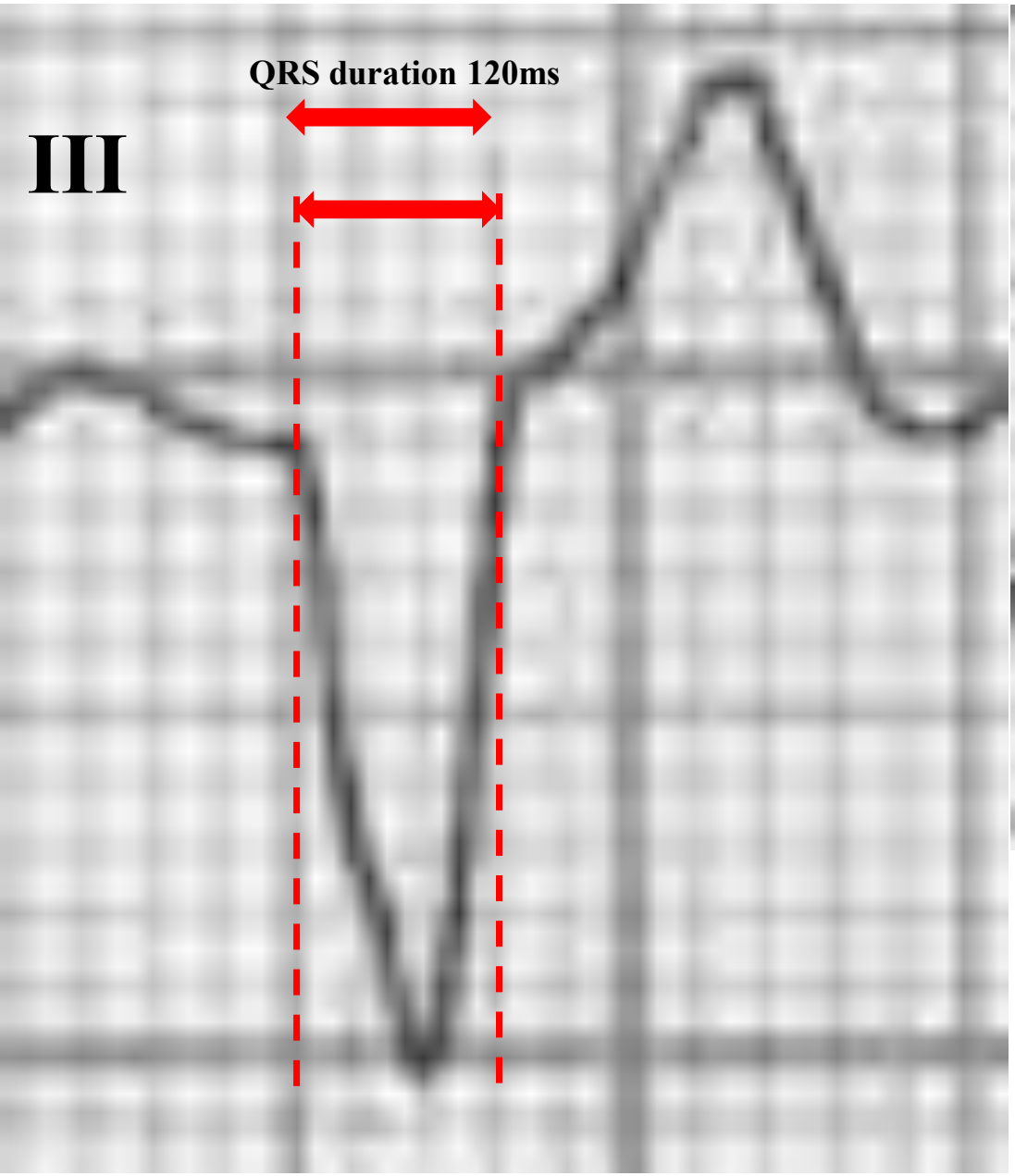
ECG diagnosis: sinus rhythm, HR 110 bpm, P-axis: +60°, P-duration: 80ms, P-voltage: 1mm, PR segment: 145ms, QRS axis: -20°, QRS duration: 120ms, total or predominantly negative QRS complexes in right precordial leads, monophasic, broad (**without notched or slurred)** R waves in left leads I, aVL, V5 and V6, QS pattern in aVR, prolonged ventricular activation time (VAT) in left leads (≥50ms): permanent LBBB, the ST-segment and T-wave vectors are opposite to a greater deflection of QRS: positive from V_1 to V_3 and negative in left leads I, aVL, V_5 and V_6. These are (**appropriate)** Secondary Repolarization Abnormalities with wide QRS-ST-T angle and normal ventricular gradient. These features are clinically relevant because primary abnormalities indicate changes in the repolarization characteristics of ventricular myocytes whereas secondary ones do not. Please see ludic explanation in the next slide…….

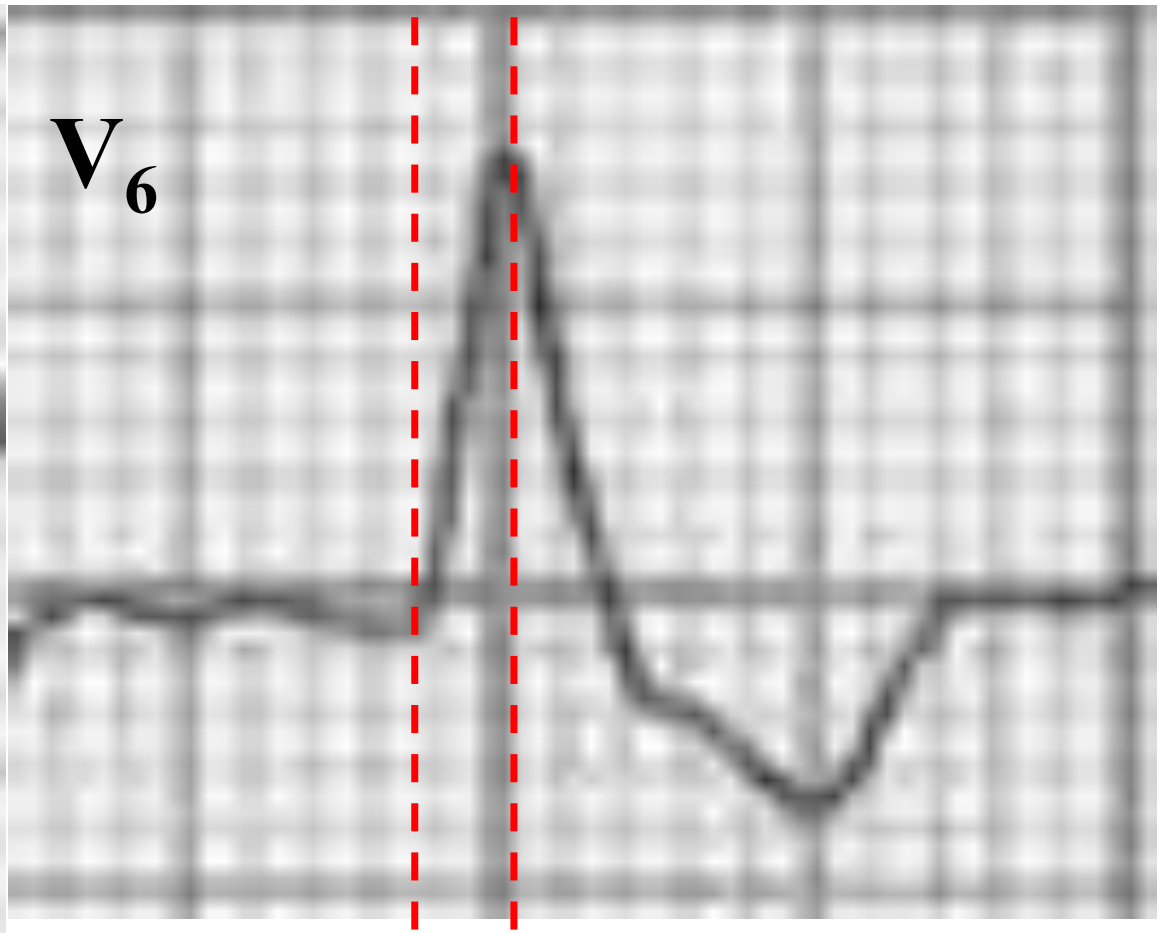
Ventricular repolarization in Uncomplicated Complete LBBB

The ST- segment and T-wave vectors opposite to a greater deflection of QRS: positive from V_1 to V_3 and negative in left leads I, aVL, V_5 and V_6. These are Secondary Repolarization Abnormalities with wide QRS-ST-T angle and normal ventricular gradient. The classic ventricular gradient concept introduced by Wilson et al in 1931 is of some theoretical interest concerning primary versus secondary repolarization abnormalities. Ventricular gradient in a single ECG lead is the net time integral of the ECG voltage from the beginning of the P wave to the end of the U wave. Its spatial counterpart is the ventricular gradient vector determined from the orthogonal XYZ leads. The practical utility of the ventricular gradient in differentiating primary from secondary repolarization abnormalities has not been demonstrated. When the direction of the QRS axis is normal, an abnormal direction of the T-wave axis is generally an indication of primary repolarization abnormalities. The figure bellow representing ventricular repolarization in not complicated LBBB. Secondary alteration of ventricular repolarization is observed with QRS/ST-T angle near the 180º. The ST segment is convex upward followed by negative T wave in left leads and ST segment concave upward followed by positive asymmetric T wave in right leads (**Wilson 1931; Surawicz 1988**).




Frontal
-90°
aVR
aVL
ΔQRS -20°
X
I
0°
III
II
Y
+90°





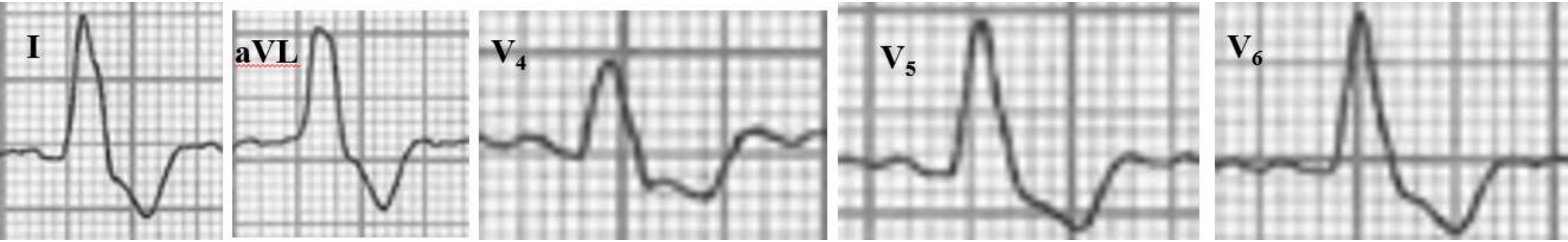
Prolonged Ventricular Activation Time (VAT) in left leads (≥50ms)

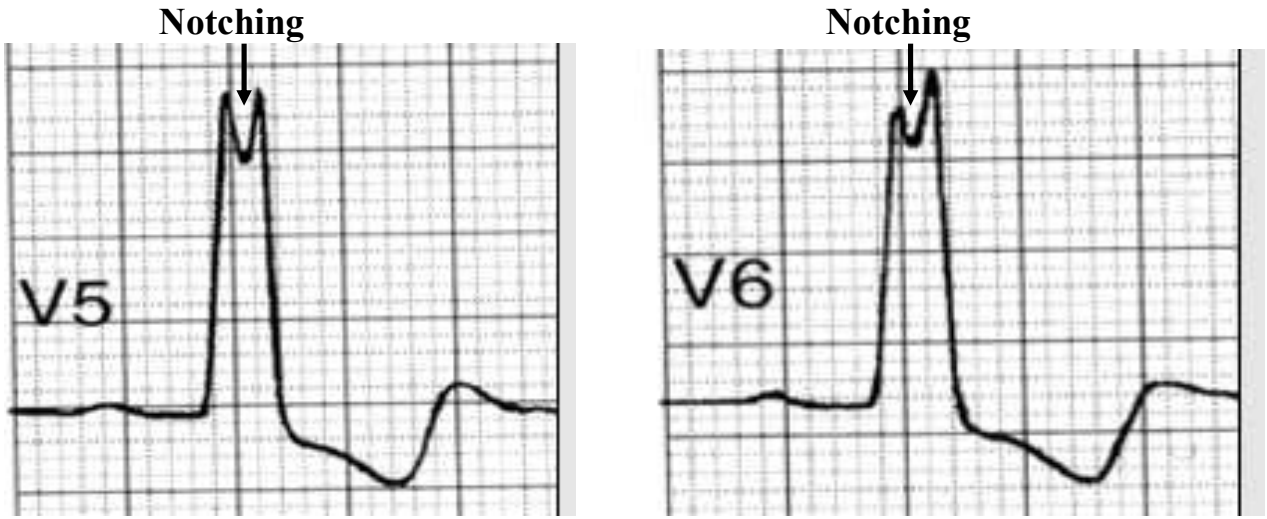
Observation: the second ECG ("without pain") does not meet Strauss's strict new criteria for complete left bundle branch block (**Strauss 2011**):

1) QRS duration ≥ 140 ms for men and ≥ 130 ms for women
2) Mid-QRS notching or slurring in ≥ 2 contiguous leads.

This new values and shape are important for Cardiac Resynchronization Therapy (CRT) indication.

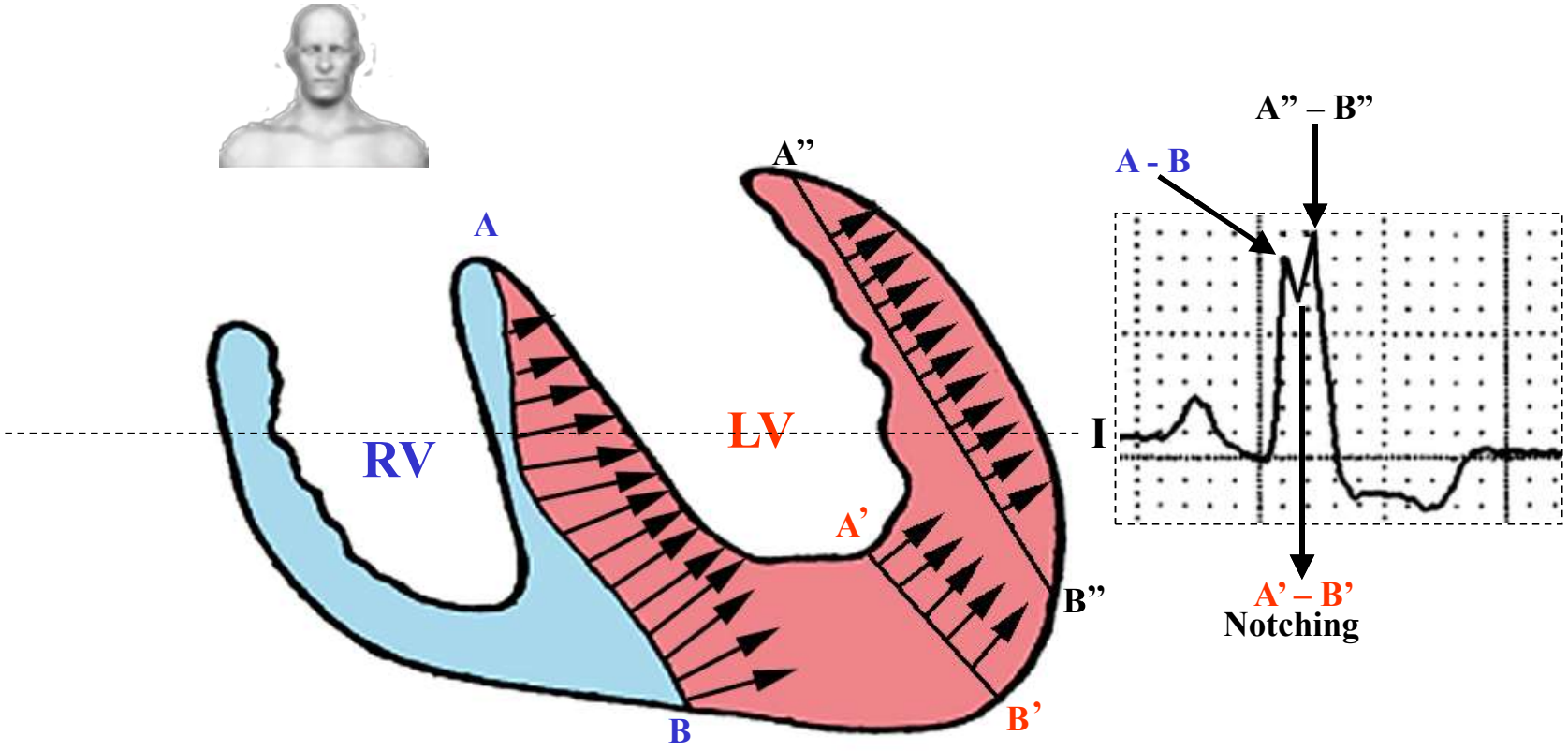
In the present case QRS duration has 120ms and there are not notching or slurring in ≥ 2 contiguous leads at the apex of the R waves in left leads **I, aVL, V_4, V_5, V_6.**





Mid-QRS notching or slurring in ≥ 2 contiguous leads.

Monophasic R wave of slow recording with notching or slurring 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. 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.

Electrocardiographic classification criteria for Left Bundle Branch Block

I- According to the degree:

1. **Criteria (most used in literature):**

- *Incomplete LBBB:* Incomplete Left Bundle Branch Block (QRS duration from 90 to 110 ms)
- *Complete LBBB:* Complete Left Bundle Branch Block (QRS ≥ 120 ms) in adults.
- Strict criteria of Strauss for complete LBBB: QRS duration ≥ 140 ms for men and ≥ 130 ms for women, along with mid-QRS notching or slurring in ≥ 2 contiguous leads. This new values are used for Cardiac Resynchronization Therapy (CRT) (**Strauss 2011**)

1. **Criteria from the Mexican School (Sodi 1964):**

- 1st degree left bundle branch block;
- 2nd degree left bundle branch block: 1st degree & 2nd degree correspond to incomplete LBBB;
- 3rd degree left bundle branch block, advance or complete LBBB.
  - Complete LBBB by classical criteria: QRS duration ≥ 120ms
  - Stricter criteria QRS duration ≥140 ms (men) or 130 ms (women), QR or rS in leads V1 and V2, and mid-QRS notching or slurring in ≥2 of leads V1, V2, V5, V6, I and aVL.

2. **Criteria from the Spanish School (Bayés de Luna 2007). Global left ventricular blocks:**

- Advanced left bundle branch block (ALBBB) or third degree (equivalent to CLBBB - QRS duration ≥ 120 ms),
- Non-advanced global left ventricular blocks:
  - First degree LBBB (partial) corresponds to types I and II of Mexican school: isolated R in V6 with more or fewer slurring but QRS duration < 120 ms.
  - Intermittent or second degree LBBB: corresponds to special type of ventricular aberrancy.

II- According to topography:

1. **Predivisional** (90% of cases) QRSd = 120 to 160 ms Observation: The intermittent forms are nearly always pre-divisional.
   - Of the left His bundle
   - Of the truncus of the left bundle branch
2. **Post-divisional** (10% of cases)
   - **Fascicular or divisional:** by unequal dromotropic involvement of divisions or fascicles of the left bundle branch: left anterior fascicle (LAF), left posterior fascicle (LPF) and left septal fascicle (LSF)
   - **Parietal, global Purkinjean, diffuse intraventricular, intramyocardial or intramural** (in the Purkinje-muscle union). Characterized by: wider QRS, clockwise rotation of the QRS loop in the HP and pan conduction delay of QRS loop. In general, they point out greater myocardial involvement.

III- According to steadiness:

1. Permanent or definite: most of them.
2. Intermittent, transient, episodic or of second degree LBBB that could be:
   - **Rate-dependent intermittent LBBB (Arias 2006):** the most - common cause of episodic LBBB is the change in heart rate (tachycardia or bradycardia). Rate-dependent BBB has also been attributed due to the "linking" phenomenon Specifically, concealed delayed retrograde activation from the contralateral bundle branch maintains the functional antegrade block (**Caldwell 2013**).
     - **Tachycardia-dependent or in "phase 3":** occurs when an impulse arrives at tissues that are still refractory caused by incomplete repolarization. Transient LBBB is less common than right bundle branch block (only 25% of phase 3 aberration is of the LBBB type).
     - **Bradycardia-dependent or in "phase 4":** Rosenbaum et al. (**Rosenbaum 1973**) showed that bradycardia dependent intermittent BBB is related to hypopolarization of the involved fascicle in the presence of spontaneous diastolic depolarization.
     - **Concealed conduction (Issa 2012):** it is an aberration caused by concealed transeptal conduction occurs in several situations including perpetuation of aberrant conduction during tachyarrhythmias, unexpected persistence of acceleration-dependent aberration and alteration of aberration during atrial bigeminal rhythm.

- **Ischemic rate dependent LBBB**: has been described in patients with new onset LBBB and ischemic chest pain and therefore sometimes represents a clinical challenge (**Chakrabarti 2013**). A high prevalence of CAD and/or heart failure in patients who exhibit exercise-induced BBB is possible (**Stein 2011; Williams 1988**). Noninvasive testing appears to have limited ability to detect or exclude CAD in patients with exercise induced LBBB (**Munt 1997**). Besides the appearance of LBBB during exercise, the opposite phenomenon, namely transient loss of complete BBB has also been reported (**Kligfield 1995**). The underlying mechanism may be the exercise induced alternating conduction delay in the opposite bundle branch leading to more synchronous depolarization of alternating beats (**Kligfield 1995**). Vieweg et al. (**Vieweg 1976**) reported a case of angina with rate dependent LBBB and a normal coronary arteriogram. Later, a case series of symptomatic rate dependent LBBB in patients with normal coronary arteriograms was presented (**Virtanen 1982**). The underlying mechanism in such cases is suggested to be the microcirculatory ischemia or Prinzmetal angina (**Bozkurt 2001**). In some cases, the angina due to intermittent LBBB is attributed to the sudden ventricular dysynchrony rather than to myocardial ischemia (**Perin 1991; Shvilkin 2016**), but Prinzmetal angina may be the cause in patients without coronary artery obstruction (**Alhaji 2013**). Shvilkin et al. described 4 patients with painful LBBB syndrome and systematically analyzed 46 additional similar patients that have been reported in the literature. Importantly, certain criteria for the diagnosis of painful LBBB syndrome were proposed by Shvilkin:
  1) Concomitant onset of chest pain with LBBB development;
  2) Resolution of symptoms when LBBB disappears;
  3) Normal 12-lead LBBB (although cardiac memory T wave changes may be evident);
  4) Absence of myocardial ischemia;
  5) Normal LV function and low precordial S/T ratio during LBBB (**Shvilkin 2016**).

  Exercise training is an effective treatment in symptomatic exercise induced LBBB (**Anderson 2014**). Right or biventricular pacing based on EPS evaluation may be an effective measure to manage the painful LBBB syndrome (**Shvilkin 2016**).

Source: Ann Noninvasive Electrocardiol. 2016;21(2):117-25.

- **Intermittent LBBB independent from heart rate. Mechanisms:** Mobitz type I; Mobitz type II by Wenckebach phenomenon; and by significant hypopolarization.

Etiologies: athletic heart syndrome (**Chapman 1977**), general, local, and epidural anesthesia (**Pratila 1979**); acute pulmonary embolism (**Kasmani 2009**); cardiac interventions (**Shimamoto 1998**); mad honey poisoning (**Sayin 2012**); acute pancreatitis (**Pezzilli 1999**); drugs effect; coronary fistulas between pulmonary artery and coronary arteries (**Juraschek 2011**), chest contusion (**Pizzo 2005**), cervical wound (**Ishikawa 2014**), Guillain-Barre syndrome (**Serrano Junior 1987**), takotsubo cardiomyopathy (**Di Cori 2010**), Graves-Basedow hyperthyroidism (**Lubczynska-Kowalska 1971**), hemorrhagic stroke (**Martynov 2004**), myocarditis in enteric fever (**Prabha 1995**), myocarditis along with acute ischemic cerebellar, pontine and lacunar infarction following viper bite (**Bhatt 2013**), electroconvulsive therapy (**Adams 2014**), endoscopic retrograde cholangiopancreatography (**Kounis 2003**), tachycardia induced cardiomyopathy (**Senoo 2014**), endoscopic brachytherapy for lung cancer (**Vasic 2009**), and propionic aciduria (**Ardoin 2009**).

- **Intermittent LBBB during Anesthesia:** intermittent LBBB during general, local, and epidural anesthesia was described. LBBB during anesthesia may be related to blood pressure perturbations (**Pratila 1979**), and due to tachycardia (**Sunaguchi 1998; Nonaka 2004; Tyagi 2004**) or bradycardia (**Mizuno 2009**) while its occurrence poses a diagnostic dilemma with regard to the presence or not of intraoperative myocardial ischemia or AMI (**Reyford 1994**). In some cases, the occurrence of LBBB during anesthesia has been attributed to the patients' medications such as carbolithium (**Azar 1977**) or cardiotoxic drugs for cancer treatment (**Sunaguchi 1998; Tagliente 1989**). The anesthetic drugs may affect the cardiac conduction system (**Tagliente 1989**). Intermittent LBBB during general anesthesia is possible in cases of epidural (**Asao 1996**), local (**Harioka 1988**) or combined general and epidural anesthesia (**Adams 2013**). Previous history of exercise-induced LBBB may portend the appearance of LBBB during anesthesia (**Elterman 2014**). Athlete's heart has been associated with the appearance of anesthesia induced LBBB (**D 2013**). Intermittent LBBB with disappearance during anesthesia is possible (**Mishra 2009; Garcia 1997**).

- **Intermittent LBBB in Acute Pulmonary Embolism (APE):** RBBB is a common ECG feature of APE (**Costantini 2004**). Intermittent LBBB secondary to sinus tachycardia can rarely be the prominent conduction abnormality in this condition (**Kasmani 2009**). Intermittent LBBB disappearance in APE is observed (**Athar 2002**). A possible mechanism for this apparently bizarre phenomenon is the conduction delay along the RBB which may result from APE and RV strain. However, the appearance of a bradycardia after APE is another possible explanation either because slower heart rates (HRs) can slow conduction along the RBB or because the previous "normal" HR is higher than the critical HR leading with that way to a rate-dependent LBBB which disappears in slower HRs (**Athar 2002**). A higher HR of complete AV block requiring permanent pacemaker implantation while it predicts a failure of LVEF improvement and poorer functional status after the procedure (**Urena 2014**). A case of Watchman device dislocation unmasked by intermittent LBBB was described (**Perez Matos 2014**). In this case the LA appendage device was dislocated to the LVOT. The presence of intermittent RBBB or LBBB after performing an accessory pathway ablation for Wolff-Parkinson-White syndrome was observed (**Fuenmayor 2013**). The BBB that appears after ablation is frequent, intermittent, and benign while it is not associated with further consequences and seems to be a manifestation of cardiac memory (**Fuenmayor 2013**).

- **LBBB during Cardiac Interventions:** Intermittent or permanent LBBB is a rare complication of left cardiac catheterization (**Shimamoto 1998**). Given that the trunk of LBB is short and divides immediately it is believed that it is resistant to trauma whereas RBB is sometimes injured during right sided catheterization (**Shimamoto 1998**). It has been demonstrated that suture less aortic valve replacement (AVR) with the Perceval S bioprothesis is complicated by intermittent LBBB in a ≈11% of cases (**van Boxtel 2014**). Transcatheter Aortic Valve Implantation (TAVI)-induced LBBB is intermittent in ≈35% of cases (**Poels 2014**). TAVI induced LBBB negatively affects cardiac function and hospitalization, while its impact on mortality is still controversial (**Poels 2014**). TAVI-induced persistent LBBB associated with higher mortality rates compared to transient LBBB (**Houthuizen 2014**). Urena et al. observed that new onset persistent- LBBB after TAVI is not associated with increased risk of mortality or rehospitalization (any cause or HF) at 1-year follow up but only with a higher rate of complete AV block requiring permanent pacemaker while it predicts a failure of LVEF improvement and poorer functional status after the procedure (**Urena 2014**).

- **Intermittent LBBB and Mad Honey Poisoning, grayanotoxin poisoning, honey intoxication, or rhododendron poisoning:** Mad honey poisoning occurs after intake of honey derived from the nectar of Rhododendron species containing grayanotoxins (**Sayin 2012; Saritas 2011**). Several conduction disturbances including AV blocks, sinus bradycardia, nodal rhythm, asystole, QT interval prolongation, and even AMI due to mad honey poisoning is possible (**Saritas 2011**). Intermittent LBBB caused by tachycardia, bradycardia, or hypotension after mad honey poisoning were described (**Sayin 2012; Saritas 2011**).

- **Intermittent new-onset LBBB induced by drugs:** A single oral dose of propafenone or flecainide can be administered to terminate acute-onset AF outside the hospital once treatment has been proved safe during hospitalization for selected patients ("pillin- the-pocket" strategy) (**Camm 2010**). Intermittent LBBB and prolonged PR interval with propafenone 600 mg in a patient with a history of hypertension and recurrent paroxysmal AF and paroxysmal supraventricular tachycardia treated with carvedilol was resported (**Patane 2008a**). Intermittent LBBB induced by drugs includes propoxyphene hydrochloride poisoning (**Heaney 1983**), intravenous lisuride (**Capria 1993**), propafenone and antineoplastic agents association (**Capria 1993**), flecainide (**Shvilkin 2016**), trastuzumab (**Tu 2009**), trimethaphan (**Di Leo 1984**), digoxin intoxication (**Singh 1976**), phenothiazines, tricyclic antidepressants (**Fowler 1976**), and chloroquine (**Fourcade 2014**).

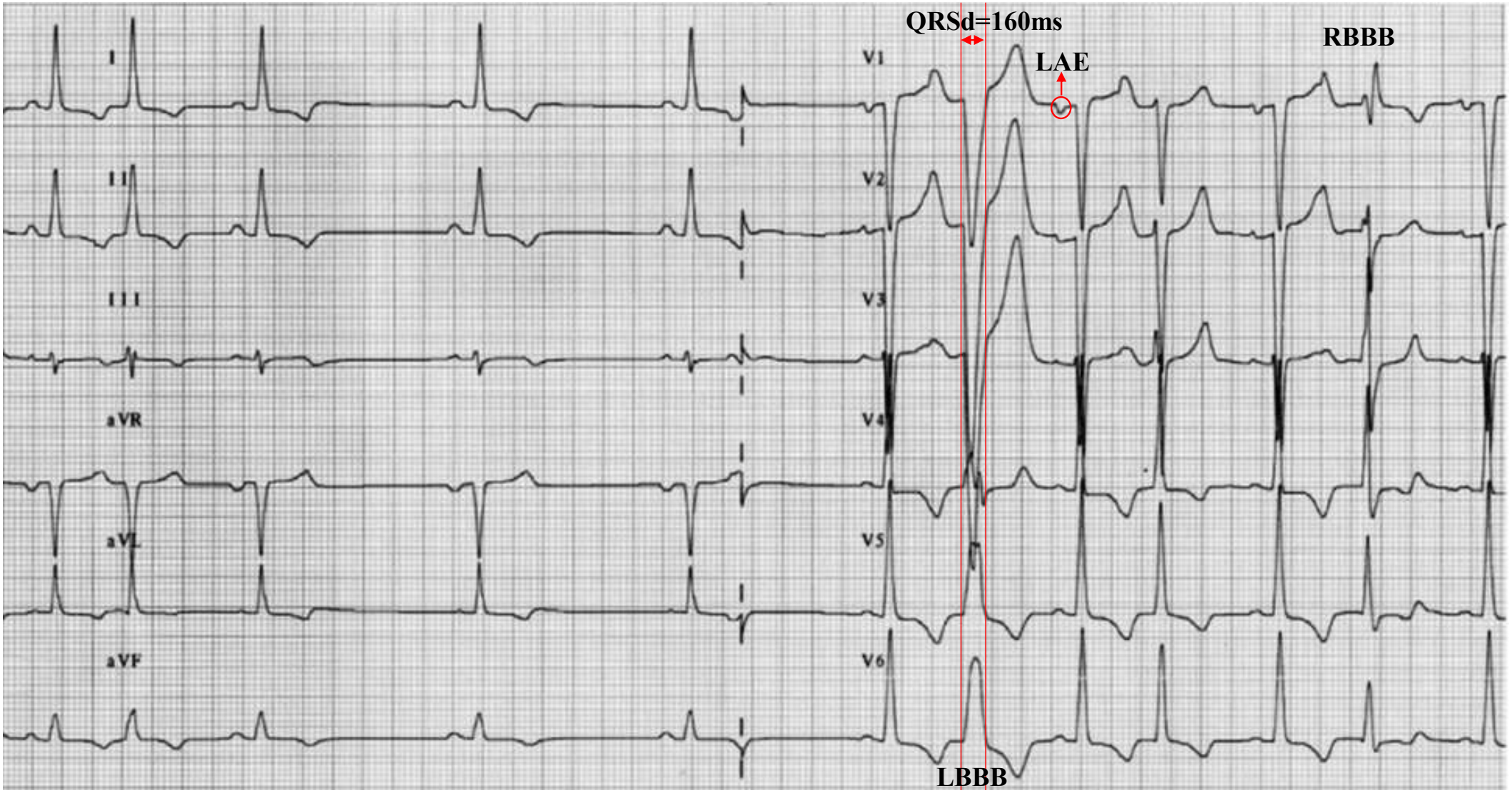
- **LBBB consequence of changing intrathoracic pressure:** Chow et al. (**Chow 2012**) observed a senior man with an intermittent LBBB triggered by laughter without HR changes. The patient had syncope immediately after a crisis of laughter and coughing. The coronary angiography showed triple vessel CAD with severe stenosis of the LCx, and revascularization was successfully performed with successful deployment of a drug-eluting stent. The exercise stress testing with a "laughter challenge" 6 weeks later showed no evidence of inducible LBBB. The authors speculated that intermittent ischemia, exacerbated by elevated intrathoracic pressure during laughter, may have contributed to intermittent LBBB.

Different ECG manifestations of left ventricular hypertrophy in presence of intermittent LBBB and RBBB

LVH is commonly found in patients with hypertension and aortic stenosis and associated with worse prognosis. In patients with severe aortic stenosis LVH is found in ≈90% of cases, whereas in patients with treated hypertension its prevalence is estimated to be between 17% and 32% (**Seiler 1996; Mancia 2002**). Most often LVH is diagnosed with the use of echocardiography or quantified in detail by CMRI, but ECG detection of LVH also has been subject for many clinical trials. Currently ECG diagnosis of LVH is implemented in the model proposed by the European Society of Cardiology and European Society of Hypertension for detection of end-organ damage in hypertensive patients (**Mancia 2013**). A variety of proposed LVH diagnostic criteria may be found. Currently used guidelines for ECG LVH diagnosis are based on limb lead voltage and/or precordial lead voltage, with or without including into the criteria the QRS duration (**Hancock 2009**). Because LVH detection is highly dependent on the intraventricular conduction pattern, therefore the criteria are different for patients with LBBB and RBBB. Depolarization patterns occurring in those setting are very different from those observed in the physiological conditions. ECG criteria for LVH in the presence of intraventricular conduction abnormalities requires knowledge on the electrophysiology of the heart muscle and the determinants of the observed changes. In the presence of LBBB one of the factors contributing to the QRS morphology is the depolarization of the RV occurring significantly earlier than depolarization of the LV. In the normal settings vectors responsible for the LV and RV depolarization have opposite sense and therefore attenuate each other's amplitude. In LBBB the LV depolarization amplitude is increased because its vector is not countered by the RV force, since it has already been depolarized; and the thick inferobasal wall is activated prior to the thinner anterolateral wall. This causes leftward shift in the frontal-plane QRS axis and results in S waves of greater amplitude observed in the right precordial leads (**de Luna 2012**).

In the presence of RBBB, depolarization of the RV is delayed and occurs after the onset of the heart electrical activation and the depolarization of LV, which causes QRS widening. The initial part of the QRS complex in the right precordial leads is “pseudo-normal” due to the initial depolarization of the septum (with septal q waves in I, aVL, and V6) and r waves with relatively normal voltage and morphology. The second part of the QRS complex results from the depolarization of the LV. Vector responsible for this part has a sense that results in the S waves in V1-V2, and R waves in I, aVL, V6. In RBBB we observe a significant reduction in “left ventricular” QRS amplitudes, caused by a reduction in LV mid-QRS forces caused by altered septal and delayed RV depolarization (**Chan 2006**). The vector responsible for RV depolarization in these settings is facing anteriorly and right-side, and appears late (approximately after 80 ms) causing a high-amplitude R (R′) wave observed in the right precordial leads (**de Luna 2012**). ECG is a relatively inexpensive and widespread method, but has several limitations. A variety of LVH criteria were described. Generally, their sensitivity is quite low (<50%) whereas the specificity is high (85-90%) (**Hancock 2009; MacFarlane 1988**). For the Sokolow–Lyon index used in the presented case to describe LVH in beats without aberrations in intraventricular conduction the sensitivity is 15–30% and the specificity is 73–100% (**Hancock 2009**).

For the description of LVH in LBBB the most often used criteria are referred to as Kafka's criteria and were also used in this patient.
Kafka et al (**Kafka 1985**) selected and used 5 ECG parameters in cumulative fashion for the diagnosis of LVH in the presence of LBBB:
1.RaVL ≥11 mm;
2.QRS axis ≤40º or SII greater than RII;
3.SV1 + RV5 to RV6 ≥40 mm;
4.SV2 ≥30 mm
5.SV3 ≥25 mm.
This cumulative approach was superior to using single conventional criterion such as SV1 + RV5 or RV6. When LVH was defined as an M-mode index of at least 115 g/m2, the sensitivity was 75% and specificity 90%. Using M-mode, a mass of at least 215 g was the standard, the sensitivity was 73% and the specificity 66%.
LVH can be diagnosed by ECG criteria in the presence of LBBB at least as reliably as in normal conduction.
The presence of LBBB on 12-lead ECG may obscure the diagnosis of LVH.
The criterion of SV2 + RV6 greater than 4.5 mV demonstrated a sensitivity of 86% and a specificity of 100% for LVH diagnosis in the presence of LBBB.
QRS duration greater than 160 ms plus left atrial enlargement strongly supports the diagnosis of LVH in the presence of LBBB (**Klein 1984**).
There are no differences in limb lead voltage, intrinsicoid deflection, or mean frontal plane QRS axis.
The following criteria can be helpful in left bundle branch block: QRS voltage increase, left atrial enlargement, QRS duration >155 ms (**Oreto 2007**).
LVH can be diagnosed in the presence of LBBB with an accuracy at least similar to that observed in patients without this conduction defect.
Computer-assisted interpretation of the ECG may be useful in the diagnosis of LVH as it enables the implementation of more accurate algorithms.
Diagnostic algorithms, voltage-duration products, and certain compound criteria had the best sensitivities (**Rodríguez-Padial 2012**).
LA abnormality is significantly diagnostic of LVH in the presence of LBBB. Age, body mass index, body surface area, frontal axis, and QRS duration are also significant predictors of LV mass (**Metha 2000**).



ECG showing the criteria of left atrial enlargement (LAE) + LVE in the presence of intermittent LBBB (QRS duration = 160ms) and RBBB. The RBBB causes significant drop in the S wave amplitude, while LBBB unmasks LV abnormalities causing higher S wave amplitude in the V1–V3 leads. In beats with LBBB and QRS duration ≥160 ms plus LAE strongly supports the diagnosis of LVH in the presence of LBBB.(**Klein 1984**)

Clinical impact of intermittent LBBB

Individuals who develop exercise-induced LBBB during exercise stress testing have a significantly higher all-cause mortality compared to both individuals with normal and those with abnormal ST-segment depression (**Stein 2011**). Exercise-induced LBBB independently predicts a higher risk of death (29%) and major cardiac events (19%) compared with matched control group (**Grady 1998**). Moreover, the rate in which exercise-induced LBBB occurs is suggested to be a prognostic factor (the onset of exercise-induced LBBB at a HR between 120–125 bpm is correlated with the occurrence of CAD while the appearance of exercise-induced LBBB at a HR of 120–125 bpm is correlated with a normal coronary arterial tree and a better prognosis (**Hertzeanu 1992**). In patients without structural heart disease, exercise-induced LBBB have better prognosis (**Stein 2011**). The prognosis of patients with exercise-induced LBBB and angina having normal coronary arteries is better compared to patients with LBBB and CAD (**Candell Riera 2002**). The development of exercise-induced LBBB is predictive of subsequent progression to permanent LBBB (**Lilly 2015**). In a case series, 5 of the 8 patients with exercise-induced LBBB without CAD and 5 of the 12 patients with exercise-induced LBBB and CAD developed permanent LBBB. Progression to complete AV block with the consequent need of pacemaker was observed rarely (**Candell Riera 2002**). LBBB is associated with asynchronous LV wall motion and secondary changes in the filling pattern (**Takeshita 1974; Xiao 1994**). These abnormalities can lead to intermittent systolic and diastolic dysfunction and are reversible after restoring the normal conduction (**Xiao 1994**). Donzeau et al. (**Donzeau 1994**) studied 3 cases where sustained rate-dependent LBBB could induce syncope while no syncope happened when the same tachycardia at the same HR was associated with narrow QRS complexes.

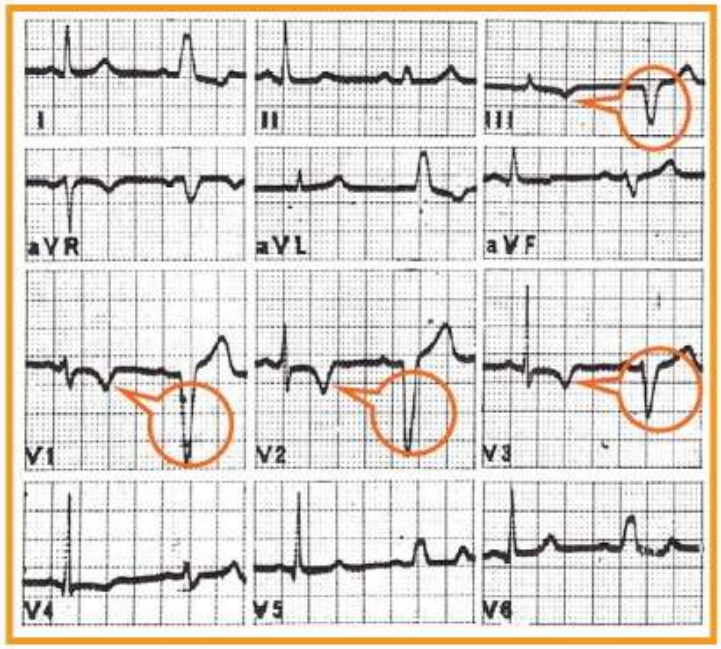
The prognosis of patients with painful LBBB is generally favorable (**Shvilkin 2016**). Patients with intermittent LBBB frequently have T wave inversions in the right and mid-precordial leads during normal conduction that usually do not reflect CAD (**Denes 1978; Cicogna 1985**).

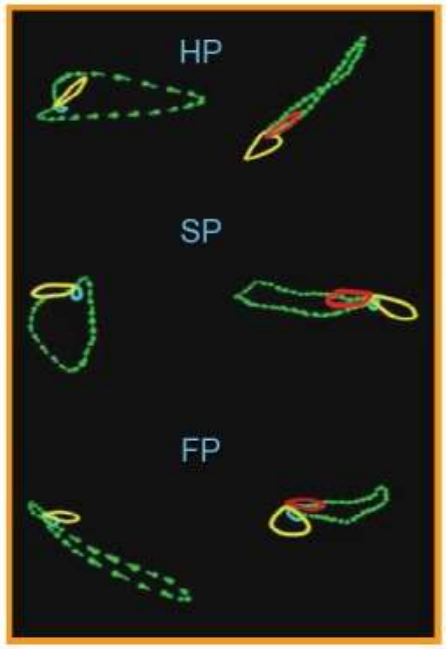
The incidence of T wave inversions is observed in ≈50% in patients with intermittent LBBB (**Abben 1979**). This specific ECG abnormality can be explained on the basis of cardiac memory phenomenon (**Van de Heyning 2011; Byrne 2010**). This refers to ECG T wave abnormalities that occur on resumption of normal ventricular activation after a period of abnormal ventricular activation (**Littmann 2014**). The presence of cardiac memory has been observed in: intermittent ventricular pacing, intermittent ventricular preexcitation, frequent PVCs, and prolonged episodes of VT (**Littmann 2014; Jeyaraj 2010**). Sometimes the ECG changes associated with intermittent LBBB may mimic anterior wall ischemia due to high-grade LAD coronary artery obstruction (Wellens' sign) (**Kershaw 2014**). The discrimination between cardiac memory and myocardial ischemia is of great importance and some ECG clues have been suggested (**Gautschi 2003; Vakil 2014**). In summary, cardiac memory or T wave "memory" is a particular variety of cardiac remodeling caused by a episodic change in the ventricular depolarization due to ventricular pacing, rate-dependent intraventricular block, ventricular preexcitation or tachyarrhythmias with wide QRS complexes. It is manifested by inverted T waves that appears when normal ventricular activation is restored. This phenomenon is cumulative and occurs earlier if the ventricular myocardium has previously

been exposed to the same conditioning stimuli. It is also shown that cardiac memory may induce not only negative pseudo-primary T waves but also a reversal of primary and pseudoprimary T waves leading to "normalization" of ventricular repolarization. The knowledge of these dissimilar consequences of T wave memory is essential to assess the characteristics of ventricular repolarization (**Chiale 2014**).

The clinical implications of cardiac memory include malignant ventricular arrhythmias such as TdP, predisposition to adverse mechanical remodeling, and abnormal response to antiarrhythmic therapy (**Vakil 2014**). Cardiac memory is associated with altered ion channel properties and therefore the action of antiarrhythmic drugs on specific ion channels is affected as memory evolves (**Plotnikov 2001**). Haverkamp et al. (**Haverkamp 1998**) described a case of repolarization abnormalities appearance after catheter ablation which was attributed at least in part due to cardiac memory and contributed significantly to proarrhythmia. The adverse mechanical remodeling is thought to arise from dyssynchronous activation of the LV which activates a cascade of signaling pathways that cause adverse structural remodeling (**Jeyaraj 2010**).

In memorian





This was the last manuscript related cardiac memory of the admired friend Pablo Ambrosio Chiale, a giant of non-invasive electrophysiology.

Example of “memory-induced” T waves in a patient with intermittent LBBB (ECG on left and VCG on right). The red loop of the T wave at right (superimposed to the VCG QRS loop of the LBBB) corresponds to the yellow loop of T wave on left (normal ventricular conduction). This composition highlights that the T wave spatial direction follows that of the QRS aberrancy.

Together 2 great colleagues, Pablo Chiale (in memorian) and Adrian Baranchuk (current editor in chief Journal of Electrocardiology), FIAI 2012

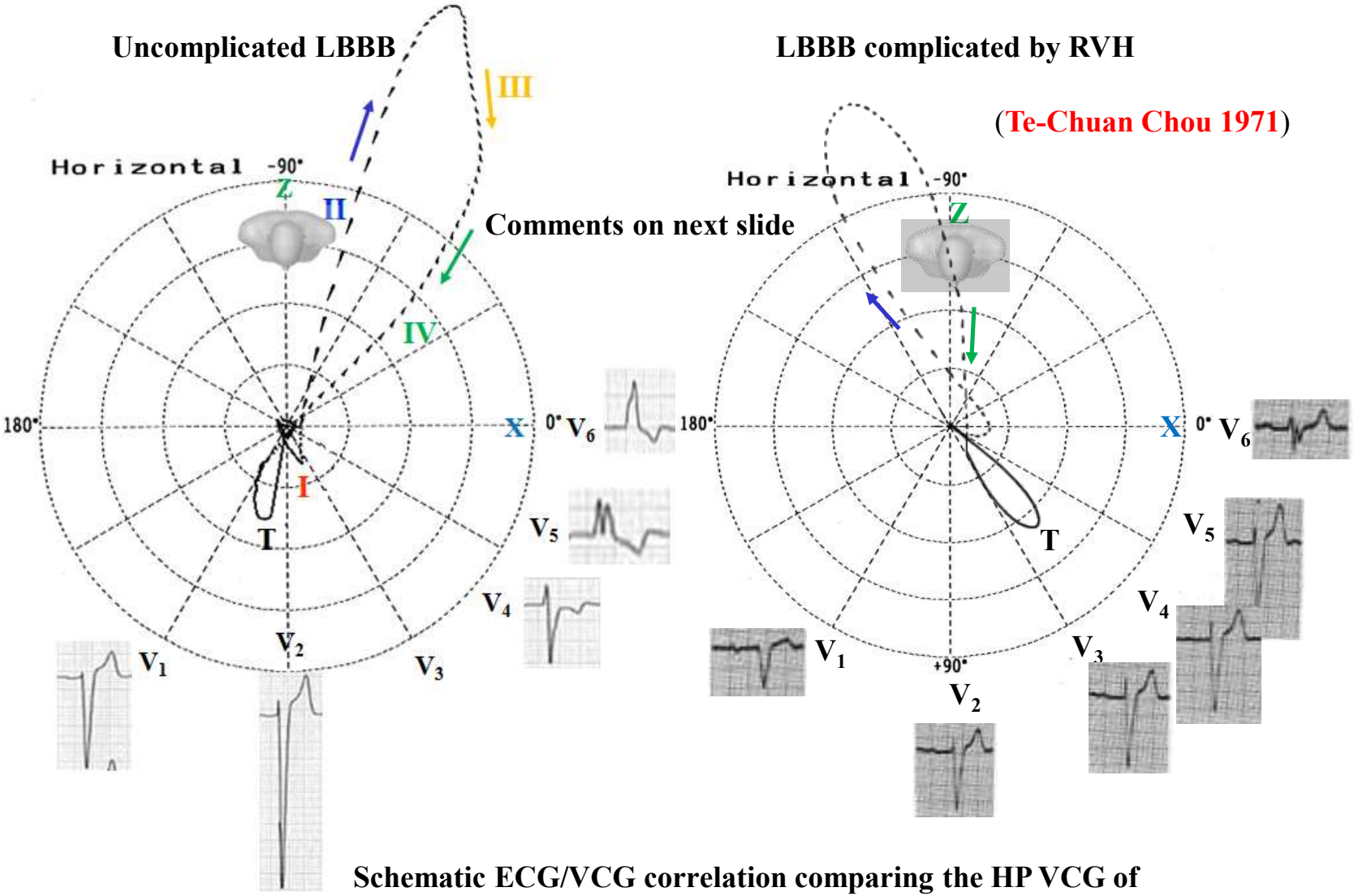
Dear Pablo, we want to tell you that we miss you more and more each passing day. Adrián, how gratifying to have your friendship.
Andrés and Raimundo

Transient changes in the sequence of ventricular activation may either induce or normalize abnormal T-wave. The background of preceding ventricular depolarization needs to be taken into account before determining the clinical significance of a given pattern of ventricular repolarization (**Chiale 2010**).

Chiale et al. demonstrate that, under certain conditions, brief periods of ventricular pacing can also normalize or improve primary or pseudo-primary T wave abnormalities. In order for this to occur, pacing should be performed from specific sites that result in QRS complexes whose polarity is opposite to that of the abnormal T waves. This phenomenon described by the first time by Pablo Ambrosio Chiale in 2010 can be referred to as "memory-induced" normalization of ventricular repolarization. This normalization can be obtained by pacing from the RVOT in patients with negative T waves in inferior and left precordial leads or from the base of the LV in patients with abnormal T waves in most precordial leads. All the above provoked changes in the T waves may be attributed to the influence of the pacing-induced shift of ventricular depolarization. The potential role of variations in the HR was prevented by maintaining a constant atrial cycle length before and after ventricular pacing.

The cellular and electrophysiological basis of the T wave is still a matter of debate (**Opthof 2009; Patel 2009**). Under normal conditions, the concordance of the T wave with the R wave in the surface ECG indicates that the repolarization sequence proceeds in the opposite direction to that followed by the depolarization process (**Wilson 1931; Noble 1978; Franz 1987**). This has been attributed to electrotonic interactions operating during the excitation process, by which repolarization lasts longer at sites where depolarization begins and is shorter at sites where depolarization ends (**Toyoshima 1978; Hoffman 1982**), thus creating regional repolarization gradients. As the course of ventricular repolarization depends on the sequence of ventricular depolarization, a shift of the latter, such as the one induced by pacing, evokes an instantaneous modification in the T waves, whose polarity tends to be opposite to that of abnormal QRS complexes (secondary T waves) (**Surawicz 1983**). Progressive changes in ventricular repolarization remain masked by the secondary changes and only become manifest once normal ventricular activation are restored. In fact, "memory-induced" T wave changes reveal a modification in the sequence of ventricular repolarization that follows the spatial direction of the main electrical forces of the previously altered depolarization process. Thus, any repetitive change in the pattern of ventricular activation is accompanied by cumulative regional changes in the repolarization process that modify the ventricular gradient.

ECG / VCG difference between LBBB and LBBB associated to RVH in thee HP



Schematic ECG/VCG correlation comparing the HP VCG of typical LBBB with LBBB complicated by RVH

VCG characterization of right ventricular hypertrophy in the presence of LBBB

The VCG characteristics are:

1. QRS loop duration with prolongation;
2. Slow inscription of the mid and late portion of the QRS loop;
3. Leftward and inferior orientation of the initial QRS vectors;
4. Posterior and rightward displacement of the maximum QRS vector;
5. Clock-wise inscription of the major portion of the QRS loop in the HP;
6. Anterior and leftward orientation of the ST vector and T-loop.

Final comments:

The changes in the HP VCG differed from the typical LBBB pattern only in the rightward displacement of the QRS loop and leftward orientation of the ST vector and T-loop.

	Isolated LBBB	LBBB + RVH
HP QRS loop	Leftward displacement	Rightward displacement
ST vector and T-loop	Righward orientation	Leftward orientation
ECG lead I	Monophasic R wave	Presence of S wave
QRS axis	From -30º to +60º (≈ 65% to 70% of cases) From –30º to -90º (≈25% of cases)	Beyond +90º (< than 1% of cases)

IV- LBBB classification according to electrical axis of QRS complex in the Frontal Plane. See figure next slide.

a) With QRS axis not deviated: between -29º and +60º (≈ 65-70% of cases)

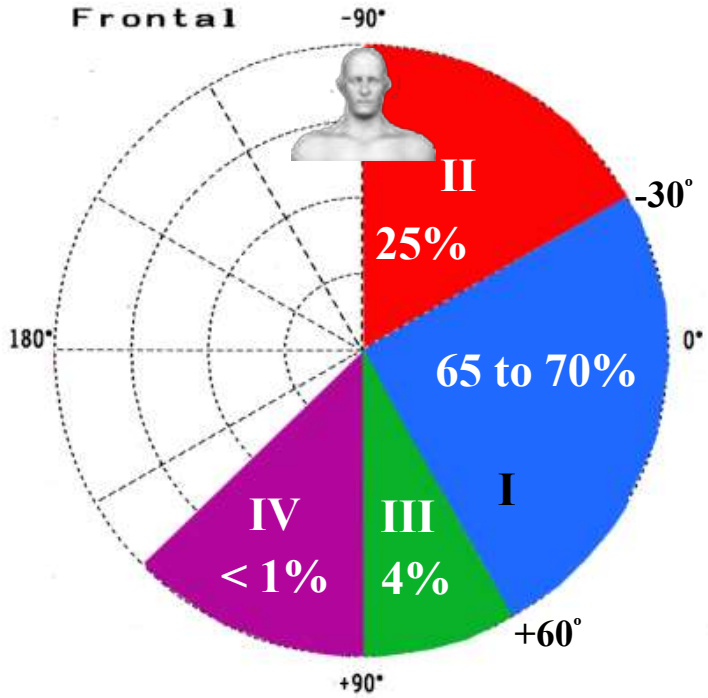
b) With QRS axis with extreme deviation to the left: beyond –30º: between -30º and -90º (**Parharidis 1997**) (≈25% of cases). The presence of left axis deviation had a 41.9% sensitivity and a 91.6% specificity for the presence of organic heart disease. Aortic valve disease in LBBB pts seems to be frequently accompanied by left axis deviation. In LBBB patients, those without left axis deviation seem to benefit more from cardiac resynchronization therapy with defibrillator (CRT-D) than those with left axis deviation (**Brenyo 2013**).

c) With QRS axis deviated to the right: between +60º and +90º (≈3.5-5% of cases)

d) With QRS axis with extreme deviation to the right: beyond +90º (<1% of cases). It is named "paradoxical type of Lepeschkin" (**Lepeschkin 1951**). The majority of subjects had dilated cardiomyopathy with biventricular enlargement (**Childers 2000**). The uncommon combination of LBBB and right axis deviation is a marker of severe myocardial disease, specially primary congestive cardiomyopathy. The mechanism of production of this ECG pattern appears to be diffuse conduction system involvement in advanced myocardial disease (**Nikolic 1985**). Causes that determine paradoxical complete LBBB:

- Complete LBBB associated to right ventricular hypertrophy/enlargement or severe cardiomyopathy with biventricular enlargement. or diffuse advanced myocardial disease.(3) >98% of cases.
- Fascicular Complete LBBB (LAFB + LPFB) with a higher degree of block in the postero-inferior division. In presence of AF LBBB with intermittent right axis deviation is explained by an additional LPFB accompanying predivisional LBBB (**Patenè 2008b; 2012**)
- LBBB in Wegener granulomatosis (**Khurana 2000**)
- Complete LBBB associated to lateral infarction (free wall of left ventricle)
- Complete LBBB with accidental exchange of limb electrodes
- Complete LBBB associated with true dextrocardia (**Salazar 1978**)

Types of CLBBB according to electrical axis of QRS complex in the FP



- With QRS axis not deviated: between -30º and +60º (≈65-70% of cases)
- With QRS axis with extreme deviation to the left: beyond –30º (≈25% of cases)
- With QRS axis deviated to the right: between +60º and +90º (≈ 3.5-5% of cases)
- With QRS axis with extreme deviation to the right: beyond +90º (<1% of cases). It is named "paradoxical type of Lepeschkin“ (**Lepeschkin 1951**). Causes that determine paradoxical complete LBBB:

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