

ACUTE CORONARY SYNDROME WITH LEFT
BUNDLE BRANCH BLOCK

SÍNDROME CORONÁRIA AGUDA COM
BLOQUEIO COMPLETO DO RAMO ESQUERDO

SINDROME CORONARIO AGUDO CON
BLOQUEO COMPLETO DE RAMA IZQUIERDA

By Andrés Ricardo Pérez-Riera MD-PhD.

Faculdade de Medicina do ABC Fundação do ABC. Santo André, São Paulo, Brasil.

68 year-old Caucasian man with severe suddenly tightness chest pain at rest radiating to the left shoulder, left arm until the elbow, for more than 20 minutes (3 hours of duration) associated with cold diaphoresis, nausea and vomiting, as well as shortness of breath and light-headedness.

Risk factors antecedents: included hypertension, overweight, hypercholesterolemia, tobacco dependence, and type 2 diabetes mellitus. He was on oral medication for (metformin, 1700mg, rosuvastatin 10mg, amlodipine 10mg, clonazepam 12.5mg, olmesartan medoxomil 40mg, and nebivolol 5mg (intermittent use)).

Physical examination: transient mitral regurgitation murmur, a third heart sound (S3 gallop), hypotension, (PA 95/70mmHg), normal HR (83bpm), diaphoresis, pulmonary rales in lung bases and jugular venous distention.

Which is the clinical diagnosis?

Which is the ECG diagnosis?

Which is the appropriate approach?

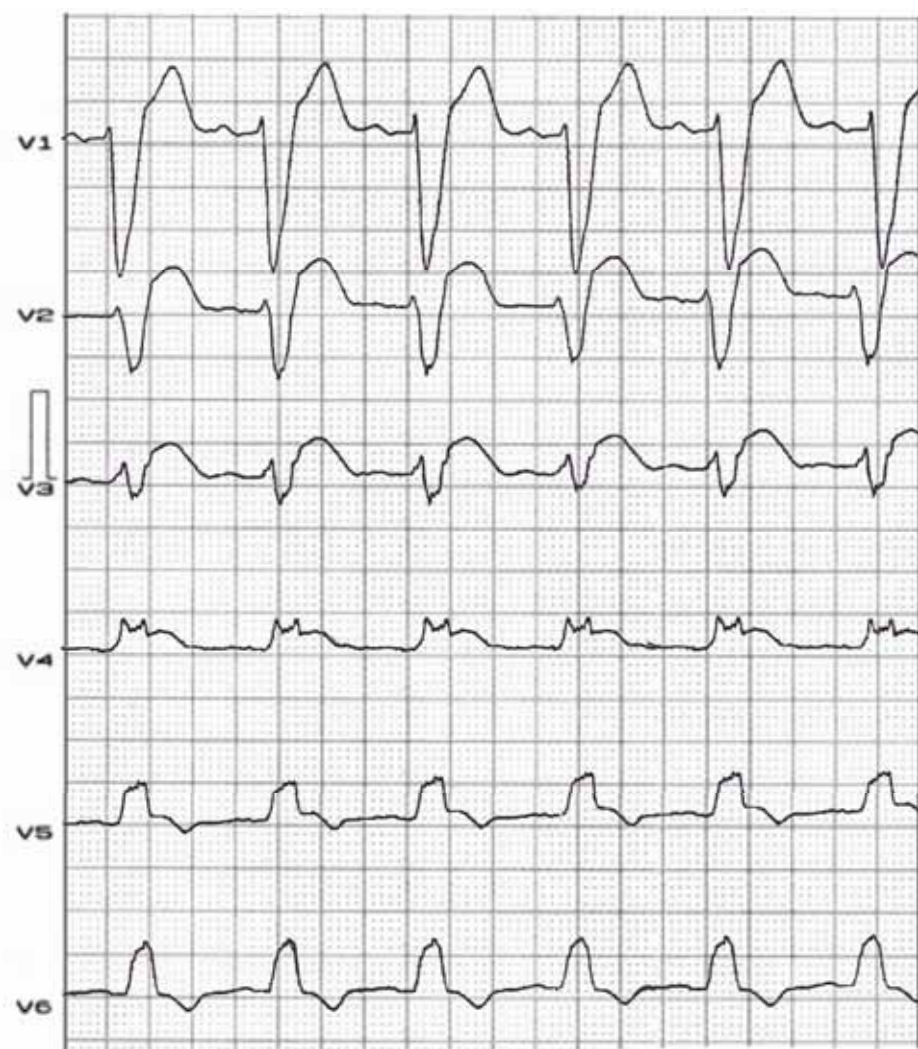
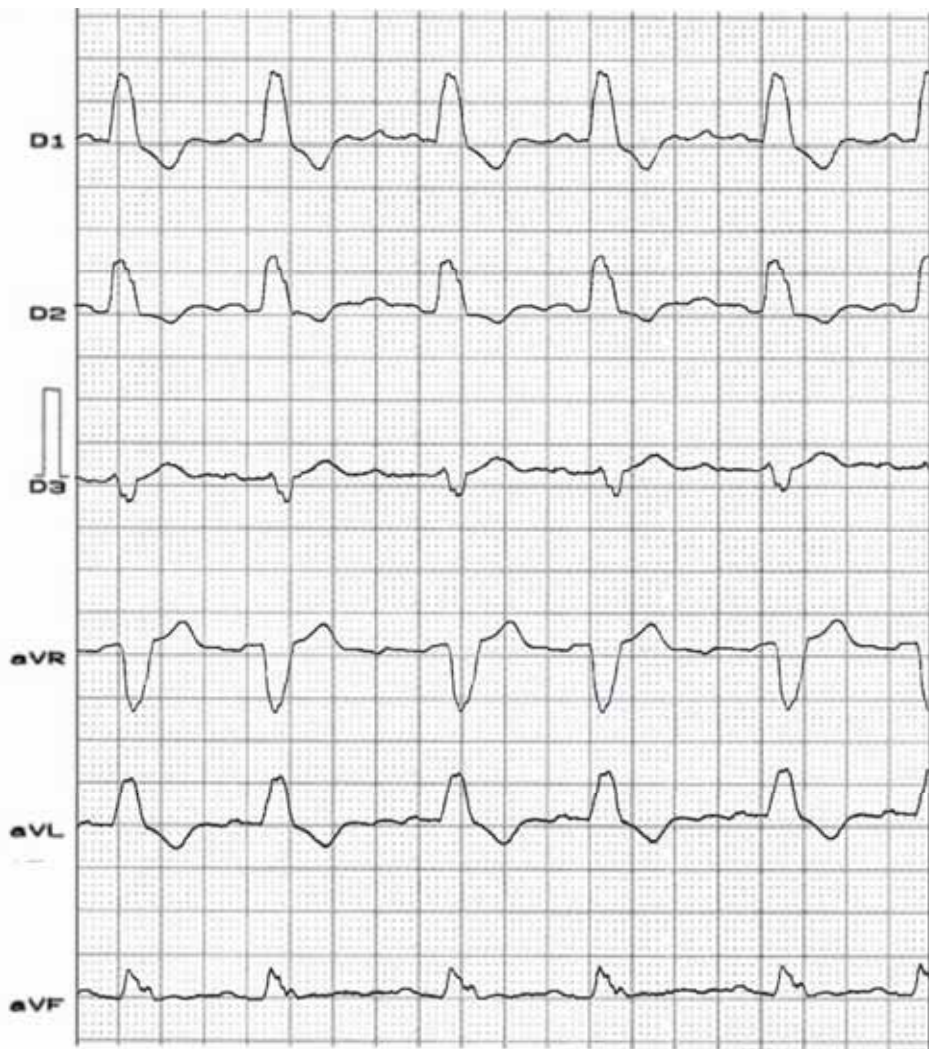
Hombre de 68 años de edad, caucásico, con queja de dolor en el pecho intenso, constrictivo de aparición súbita en reposo, irradiado al hombro y brazo izquierdo, hasta el codo y con más de 20 minutos (3 horas de duración). Acompañado de sudoración fría, náuseas y vómitos, así como disnea y mareos.

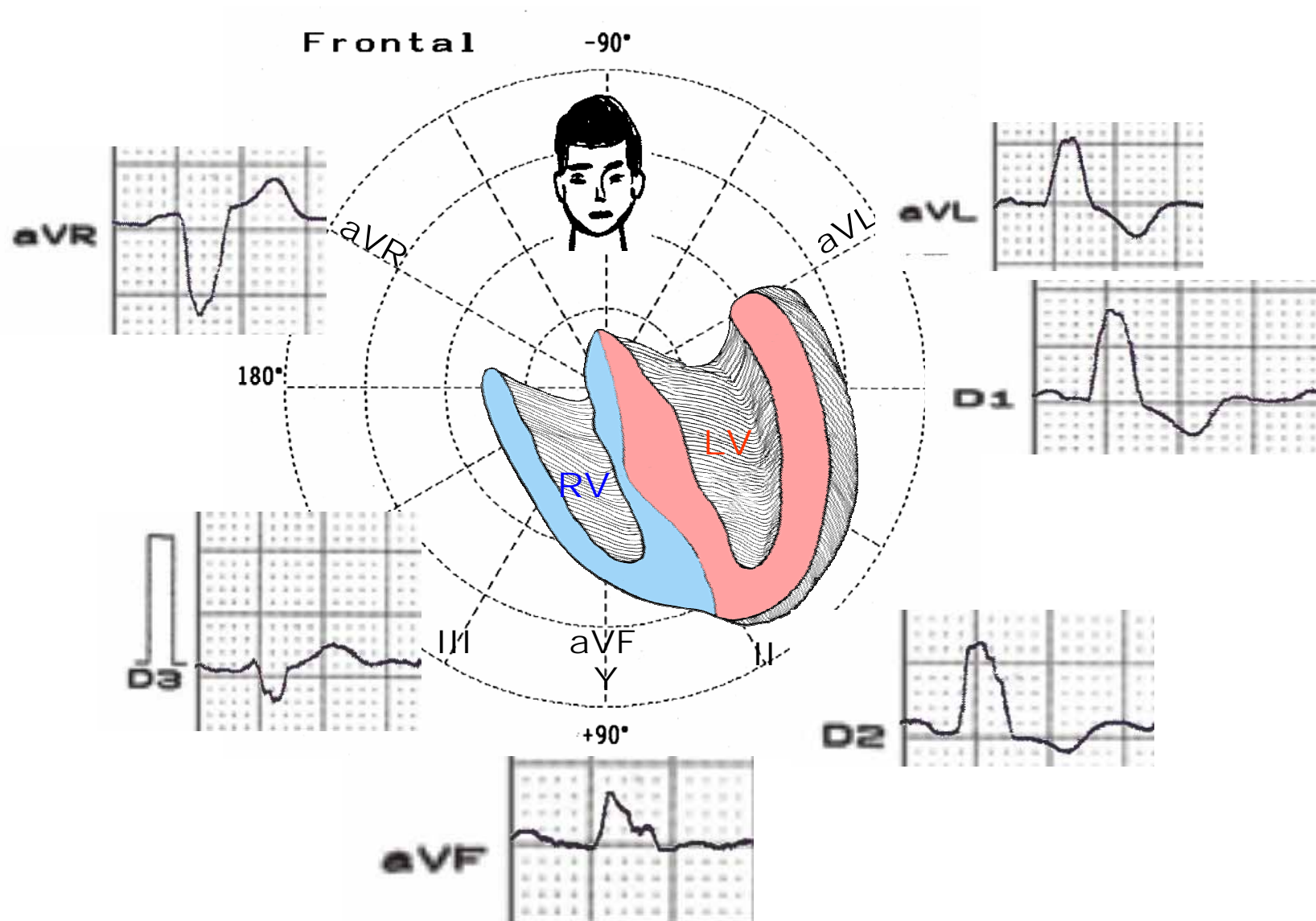
Factores de riesgo: hipertensión, sobrepeso, hipercolesterolemia, tabaquismo y diabetes mellitus tipo 2. Estaba en uso intermitente de metformina, 1700 mg, rosuvastatina 10mg, amlodipina 10mg, clonazepam 12,5 mg, olmesartán medoxomilo 40 mg y nebivolol 5 mg (uso intermitente).

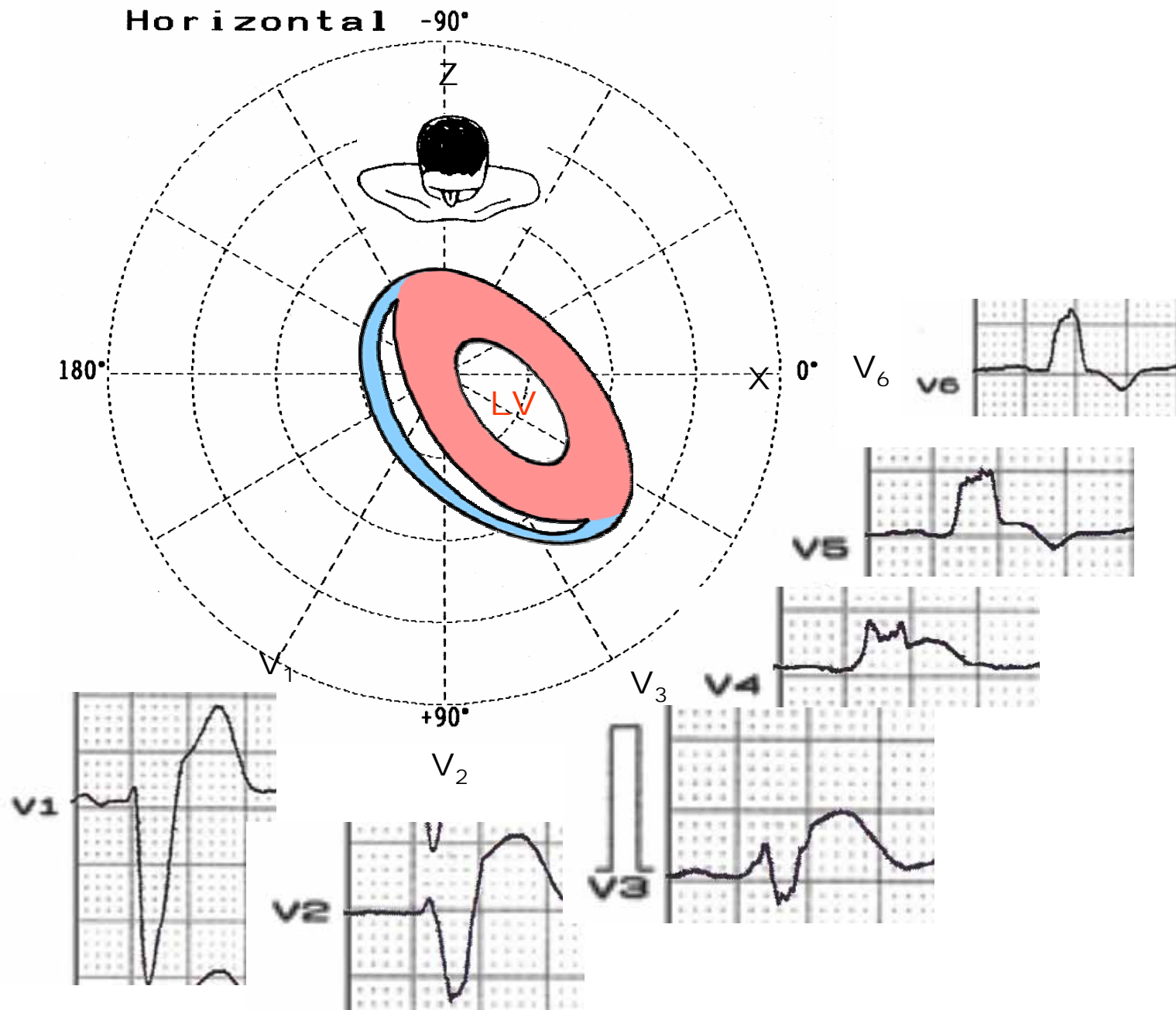
Examen físico: soplo regurgitación mitral, tercer ruido con cadencia de galope S3 hipotensión (PA 95/70mmHg), FC normal (83bpm), diaforesis, estertores pulmonares en bases y distensión venosa yugular.

¿Cuál es el diagnóstico clínico? ¿Cuál es el diagnóstico electrocardiográfico?

¿Cuál es el abordaje adecuado?







Colleagues opinions

In my opinion, this patient should have immediate coronary angiography. There is LBBB with ST elevations in V1-V5, probably as a sign of acute occlusion of the LAD. We would handle the case as STEMI.

Kjell Nikus, Tampere, Finland

En mi opinión, este paciente debería realizar inmediata coronariografía. Hay un BCRI con elevación del segmento ST en V1-V5 probablemente señal de obstricción aguda de la arteria descendente anterior. Estaríamos en manos de un caso infarto agudo con elevación del segmento ST.

Professor Andrés

De acordo com os critérios do Dr. Leo Schamroth e Dra Sgarbossa o ECG é compatível com BRE complicado com IAM anterior. Terapia de reperfusão está indicada (fibrinolítico ou ATC, depende da disponibilidade). Tempo de evolução > 3 horas a angioplastia é mais eficaz. Resumindo: laboratório de hemodinâmica para colocação de marcapasso provisório + angioplastia com stent.

Raimundo

Professor Andrés:

According to the Leo Schamroth and Dr. Sgarbossa criteria the ECG is compatible with anterior MI complicated by LBBB. Reperfusion therapy is indicated (ATC or fibrinolytic, depending on availability). If the time evolve > 3 hours angioplasty is more effective. In summary: In hemodynamic laboratory implantation of provisional pacemaker + angioplasty and stent placement.

Raimundo

Dear Andres: The clinical story of chest pain, diaphoresis, nausea/vomiting and dyspnea suggests an acute coronary syndrome. In the setting of complete LBBB, acute myocardial infarction is indicated in this case by the concordant ST segment elevation (≥ 1 mm) in leads V4-5. This is the best of the three criteria discussed by Sgarbossa et al (NEJM 1996;334:481) for recognizing acute myocardial infarction in the presence of LBBB. Also, there is an unusual amount of discordant ST segment elevation in leads V2-3. These findings indicate transmural anterolateral left ventricular injury due to obstruction of the left anterior descending coronary artery. Emergent revascularization therapy with coronary revascularization or thrombolytic therapy is the preferred treatment for this individual.

I look forward to your comments and those of your colleagues.

Regards,

Frank G. Yanowitz, MD. Professor of Medicine University of Utah College of Medicine Cardiology Director, Intermountain Health & Fitness Institute 440 D Street, Suite 206 Salt Lake City, UT 84103 801-408-5302 frank.yanowitz@imail.org

Caro Andres: A história clínica de dor no peito, sudorese, náuseas/vômitos e dispnéia sugere uma SCA. No cenário de BRE completo, IMA é sugestivo neste caso pela elevação do segmento ST concordantes (≥ 1 mm) em V4-5. Este é o melhor dos três critérios postulados por Sgarbossa et al (NEJM 1996; 334:481). Para o reconhecimento de IMA na presença de BRE Além disso, há uma grau incomum de elevação do segmento ST discordantes nas derivações V2-3. esses achados indicam lesão transmural do VE anterolateral devido à obstrução da artéria descendente anterior.

Terapia de revascularização emergencial com revascularização coronária ou terapia trombolítica é o tratamento de escolha para essa pessoa. Estou ansioso para seus comentários e os de seus colegas. Atenciosamente, Frank

Este es un bloqueo completo de rama izquierda troncular crónico muy probablemente consecuencia de una complicación de la hipertensión arterial con un infarto agudo anteroseptal sobrepuesto por obstrucción de la arteria coronaria descendente anterior distal a la primera diagonal. Porque ? por la escasa profundidad de las ondas S en V2,V3 y V4. En los BCRI tronculares con hipertensión las ondas S precordiales derechas pueden pasar de los 30mm. Esta poca profundidad de las S se expresa en infartos anteroseptales sin bloqueos de rama izquierda con R altas y ST-T elevado, pero en este caso la escasa profundidad de las ondas S expresa el mismo mecanismo electrofisiológico.

La elevación del segmento ST en V3, V4 y la separación de los vectores R1 y R2 en estas derivaciones indican enlentecimiento de la conducción en septo inferior y apex

Desde el punto de vista pronóstico, el BCRI no tiene significado, y el tratamiento es angioplastia directa o trombolisis

El PR normal indica que el sistema intraventricular derecho esta intacto

Lo unico que podria agravar el pronóstico si se afectara la rama derecha por una isquemia aguda

Querido amigo lo felicito por las extraordinarias figuras que presenta

Un fraternal abrazo

Samuel Sclarovsky

Complemento:

Queridos amigos foristas quisiera agregar algunos pensamientos sobre el caso que envio nuestro maestro profe Andres Ricardo Perez Riera, de un infarto agudo en presencia de un bloqueo izquierdo cronico. A pesar de la imagen dramática de este caso, el infarto esta muy limitado a un al septo anteroseptal, y no es de emergencia critica. Se puede estudiar por el trazado que la coronaria circunfleja izquierda, esta bien desarrollada, porque protege cara anterolateral, y este factor es importante para determinar la extensión del infarto

Si el miocardio esta bien reperfundido, a los 3 dias comenzara a verse que las onda T en las precordiales comienzan progresivamente a negativisarse, primera la segunda parte de la onda T y luego la punta de esta onda ,quedando la imagen de ST elevado ,con apice de la onda T invertida Tambien las ondas S,s se van profundizando progresivamente hasta alcanzar su maximo al 7 dia

Porque todo esto? Porque despues del lavado de potasio acumulado en el epicardio, el Q-T de esta zona se alarga con respecto a la capa

endocardial . ya que el endocardio no hay lavado de potasio por reperfusion .La reperfusion del endocardio se se hace atravez de la microcirculacion , que sufre de las isquemia por largo tiempo, mientras que el epicardio esta irrigados por ramas de los grandes vasos

Las ondas S,s se van profundizando porque se reestablece el sistema de conexina , y por lo tanto la conduccion intramiocardica

Ustedes queridos amigos ,se preguntaran porque yo vuelvo siempre ,como el viejo Cato en el senado romano volvia siempre a repetir en sus discursos "Hay que destruir Cartago "

Pero yo aprendi de unos de los grandes biologos neodarwinista ,Ernst Mayer, su famoso libro This is biology" que ciencia comienza no preguntando que es esto ,sino PORQUE es estos

Seguramente nuestro maestro nos dara un larga leccion de este sabio ,que escapo de los nazis en 1933 y se radico en Toronto Canada

A los 94 anios siguio escribiendo sobre Ornitologia , desde el punto de vista evolutivo, y murio en 2004 a los 100 anios Vale la pena leer este sabio que fue el que introdujo las nuevos conceptos de las nuevas filosofia en la biologia moderna

Sin mas , mis queridos amigos

un fraternal abrazo

samuel sclarovsky

Hola Andres,

Tu caso de acuerdo a la presentación y tus interrogantes, mi opinión es la siguiente:

Clinicamente esta cursando un infarto agudo de miocardio, con falla de bomba, tal vez con alguna complicacion mecánica como insuficiencia mitral o CIV.

Electrocardiograficamente es un BCRI con supradesnivel del ST anterior. siempre es la gran duda si el ST es debido al BCRI o a injuria miocárdica.

Sgarbosa en el gusto 1 (creo) definió los criterios para ese diagnóstico diferencial.

Probable oclusión proximal de la DA. Posiblemente tenga otros vasos con lesiones severas por todos los factores de riesgo que posee.

La conducta es urgente CCG y angioplastia del vaso culpable.

Gran abrazo.

Oscar A. Pellizzon.

Mis saludos como anda la fracción de eyección de esta paciente si desde D1veo las dos cámaras cardíacas bien dilatadas, tiene antecedentes de cardiomiopatía dilatada. Se ve a nivel V1,V2,V3 PARA EMPAZAR EL DETERIORO PROGRESIVO DE SU CARDIOPATÍA.

Gregorio Maslivar

Estimado Maestro Perez Riera:

1. Diagnostico: SCA. Angor con irradiacion a MSI, y cambios ECG. Inestabilidad hemodinamica con signos de IC al ingreso. Reportes indican mayor mortalidad en los pacientes con bloqueos persistentes (19,4%) que en los transitorios (5,6%). En los casos de BCRI persistente, mortalidad 36,4% reportada.

2, ECG: RS en la primera tira de ritmo 78 por min (en las derivaciones de los miembros), en las derivaciones precordiales RS 100 por min. CAI. Eje electrico 60°, PR 0,20 seg. BCRI en las derivaciones de los miembros 160 mseg de duracion, en las precordiales presenta mayor duracion cercana a los 200 mseg.

Presenta supradesnivel del segmento ST de V1 a V4 de 5 mm opuesto al QRS (criterios de Scarbosa). y en V3 empastamiento de la deflección terminal de la onda S (signo de Cabrera), en V4 y V5 presenta patron RR', al igual que en aVF (signo de Champman). Los cambios electrocardiograficos progresivos en este mismo ECG (aumento de la duracion del QRS, no disponemos de ECG seriados, son indicadores de probable ischemia anteroaseptal aguda e IAM posteroinferior secuelar.

3. Conducta? estudio hemodinamico y angioplastia de rescate.

Indicaciones de la ACTP primaria Indicaciones

Clase I:

1. Como alternativa a la terapia fibrinolítica

Elevación del segmento ST o bloqueo de rama izquierda nuevo o presumiblemente nuevo En 12 horas de los síntomas o pasadas las 12 horas si persiste el dolor En tiempo adecuado (tiempo puerta balón 90±30 minutos) 2. En los pacientes con shock cardiogénico <75 años, en las primeras 36 horas de evolución del infarto, si la ACTP se realiza en las primeras 18 horas de evolución del shock

Un cordial saludo

Martin Ibarrola

Hola:

Estoy de acuerdo con la descripción del ECG por el Dr Ibarrola y le haría cinecoronariografía de urgencia (para hacer PTCA) y ver válvula mitral para descartar insuficiencia mitral aguda por necrosis del músculo papilar que nos da el soplo de regurgitación y el ritmo de galope, cuadro de insuficiencia cardíaca aguda por la insuficiencia valvular antes mencionada. Estaría bueno hacer un eco de urgencia. Tener en stand by quirófano para CCV de urgencia.

saludos

Marilina

Prezado prof Andrés:

Diagnóstico clínico: SCA c/ elevação de ST

ECG: Ritmo Sinusal FC = 88 bpm PR = 0,18ms (P = 120 c/ entalhes ("Melladuras": Borderline Distúrbio da condução intra-atrial) QRS = 0,160ms c/ entalhes variados (melladuras, tower e notches) ausencia de q em D1 e AVL Alterações secundárias de repolarização Ângulo QRS/ST = +/- 180° Exagerada elevação de ST em V2 > 5mv Elevação de ST de convexidade superior em V2 até V5 (parede ântero-lateral) QRS/ST = 1 em V1 (Schamroth)

Notch em rampa ascendente de S V3 - Sinal de Cabrera. Sgarbossa = 5 pts em V3 e 2 pts em V5

Diagnóstico Eletrocardiográfico

- 1. Distúrbio da Condução intratrial**
- 2. Bloqueio completo do Ramo Esquerdo**
- 3. Infarto Agudo do Miocárdio na parede ântero-lateral**

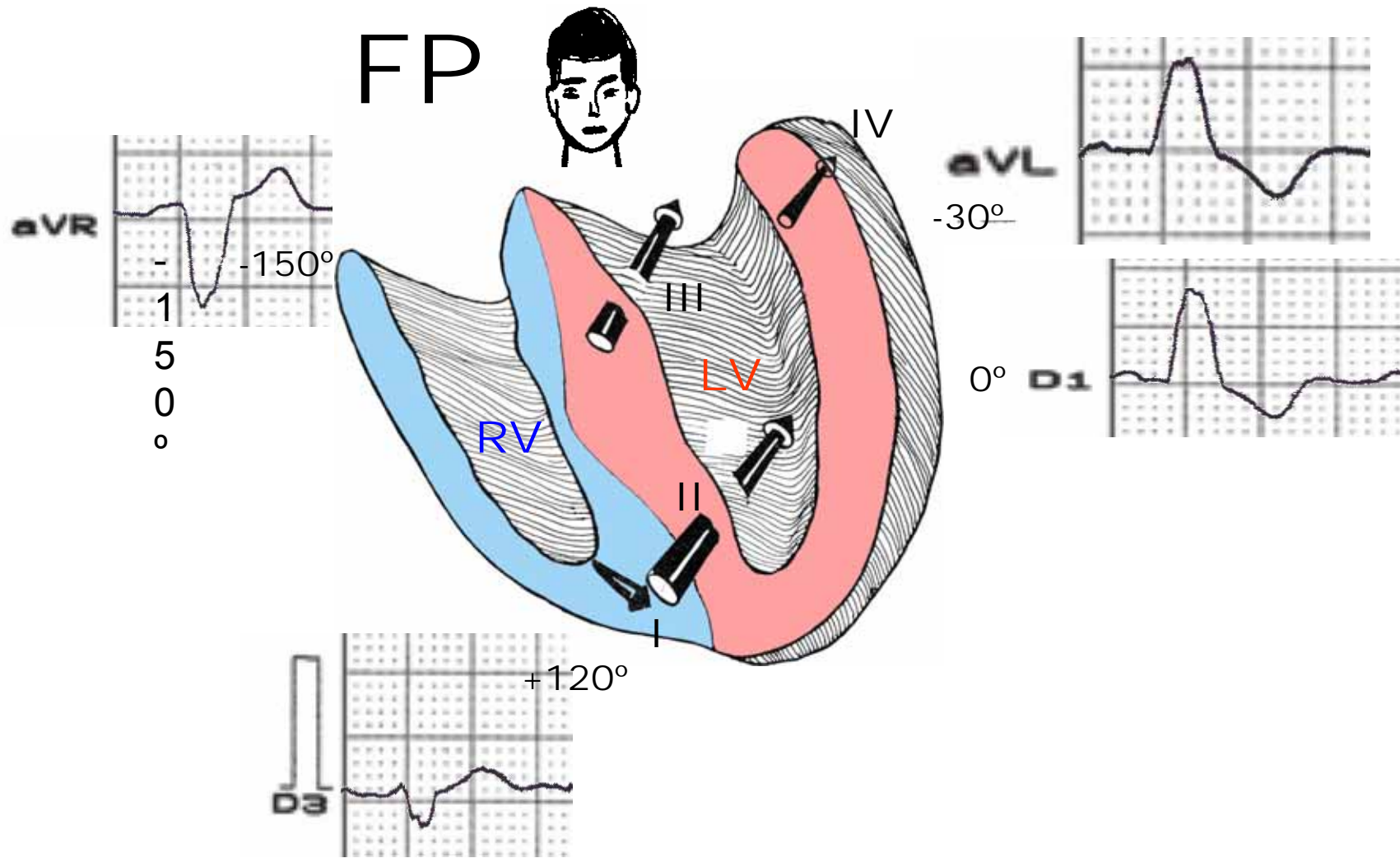
Conduta

Laboratório de hemodinâmica e decidir: Angioplastia c/ Stent ou cirurgia (RM + aparelho valvar mitral se tiver lesão)

Adail - Bahia - Brasil

Final commentaries

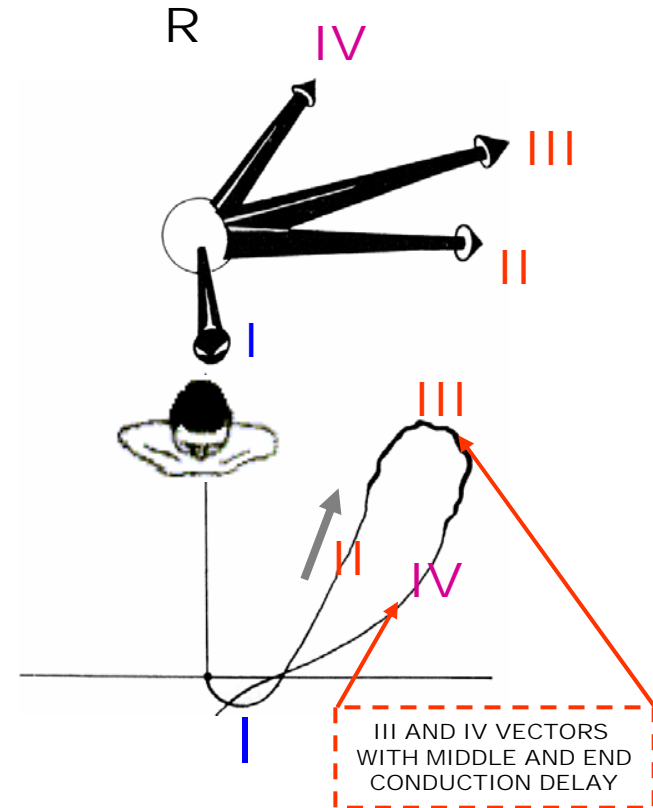
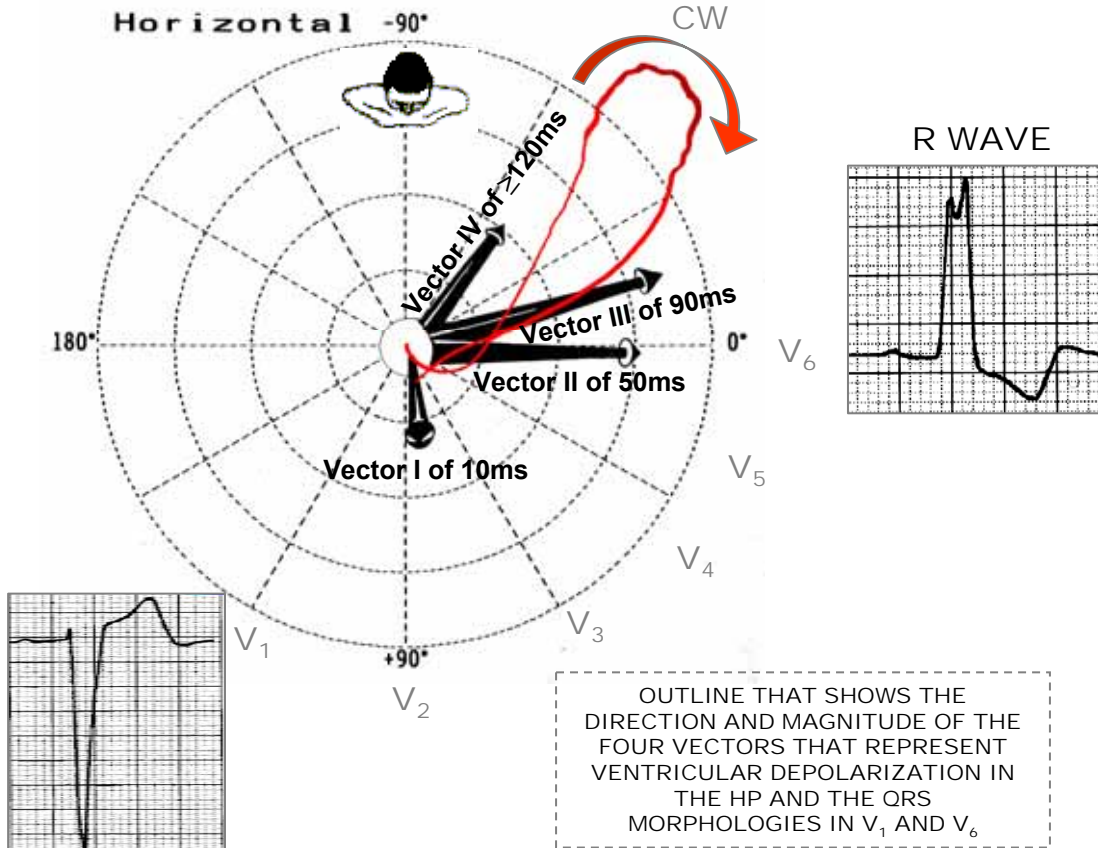
VECTORIAL REPRESENTATION OF VENTRICULAR DEPOLARIZATION IN UNCOMPLICATED LBBB



The four vectors of ventricular depolarization on the frontal plane (FP) in the LBBB.

ECG/VCG CORRELATION IN HORIZONTAL PLANE, DEPOLARIZATION VECTORS, RIGHT (V_1) AND LEFT (V_6) ECG PATTERNS IN UNCOMPLICATED LEFT BUNDLE BRANCH BLOCK

The QRS loop has
clock wise rotation



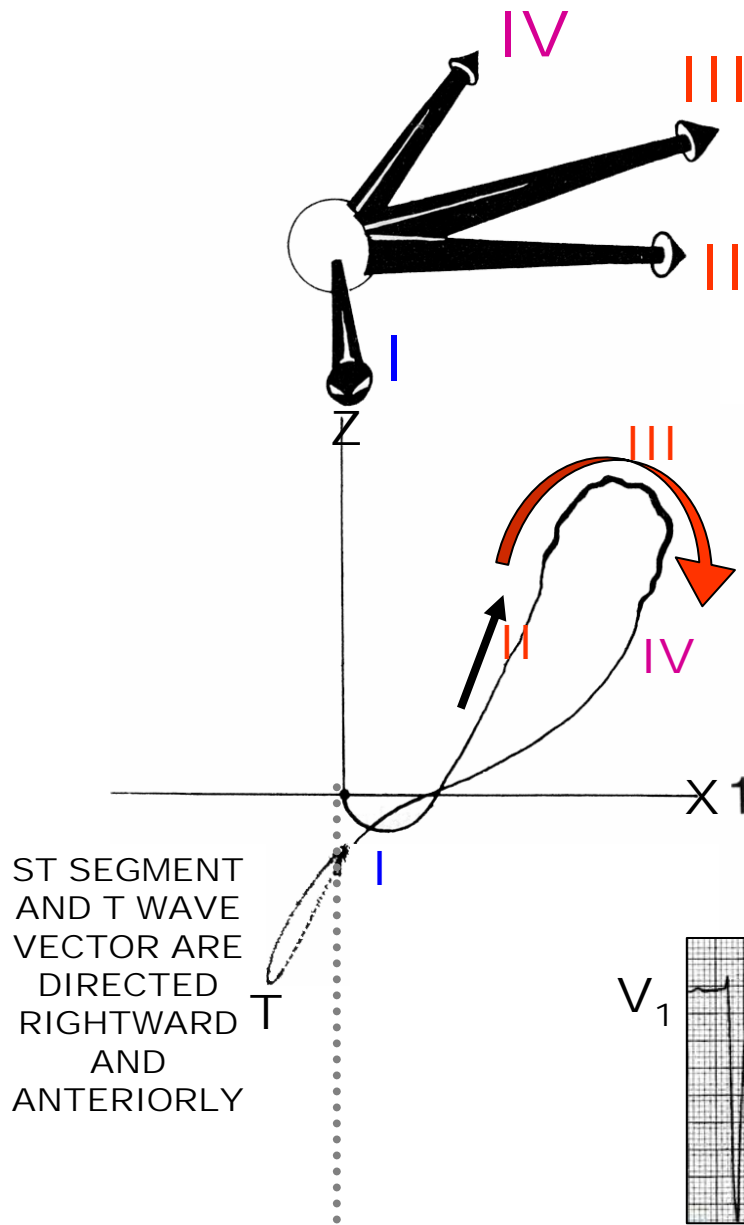
rS or QS PATTERN

CW: COUNTER WISE ROTATION

CHARACTERISTIC OF VENTRICULAR DEPOLARIZATION IN UNCOMPLICATED LBBB

THERE ARE THREE MAJOR COMPONENT IN VENTRICULAR ACTIVATION PROCESSES IN UNCOMPLICATED LBBB:

- I) **THE FIRST COMPONENT:** Right inferior septal activation on the subendocardial region of the anterior papillary tricuspid muscle: Vector I: Directed anteriorly, inferiorly and to the left.
- II) **THE SECOND COMPONENT:** Delayed and anomalous left septal activation: Vectors II and III: Directed posteriorly, superiorly and to the left.
- III) **THE THIRD COMPONENT:** Delayed and anomalous activation of the free left ventricular wall: Vector IV: Directed posteriorly, superiorly and to the left. The last 50ms is made up of activation fronts in the posterolateral wall of the left ventricle.



The QRS loop duration is $\geq 120\text{ms}$

The QRS loop shape is elongated and narrow

The small short-duration initial 10ms vector directed anteriorly and leftward: **Vector I**

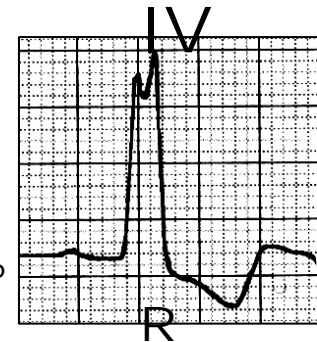
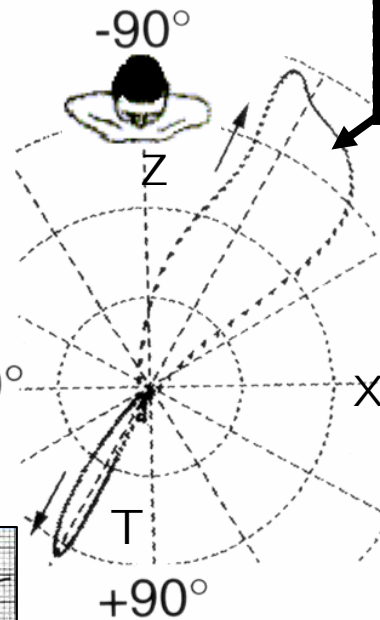
The main body of the QRS loop is inscribed posteriorly and to the left

within the range -90° to -40° .

The main body of QRS loop is inscribed clockwise (CW)

The magnitude of the max QRS vector is increased above normal exceeding 2mV.

CONDUCTION DELAY
NOTED IN THE MID AND
TERMINAL PORTION



Outline that shows the four activation vectors in LBBB in the horizontal plane. There is an ECG/VCG correlation of the QRS loop and the leads V₁ and V₆. Take notice that the middle final delay of the QRS loop in the HP rotates in a clockwise direction.

VCG ON HP IN UNCOMPLICATED LBBB

Sensi. 2
 Timer 2 msec
 Loop All Loop
 Sagittal Right
 Z Axis Back
 Filter Hum
 Muscle
 Drift

Efferente limb
 located to the right

Horizontal -90°

Conduction delay noted in
 the mid and terminal portion

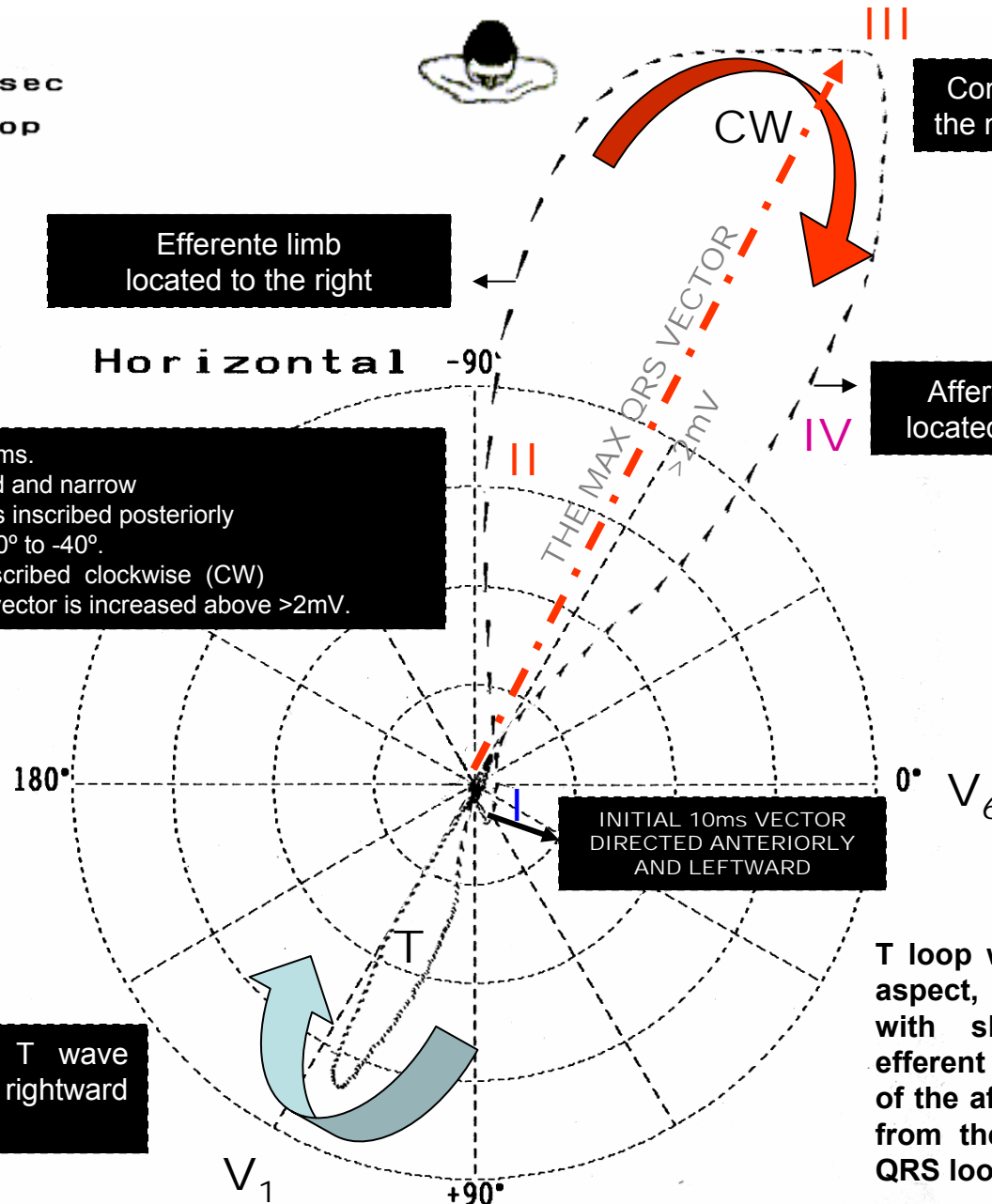
Afferente limb
 located to the left

- ✓ The QRS loop duration is ≥ 120 ms.
- ✓ The QRS loop shape is elongated and narrow
- ✓ The main body of the QRS loop is inscribed posteriorly and to the left within the range -90° to -40° .
- ✓ The main body of QRS loop is inscribed clockwise (CW)
- ✓ The magnitude of the max QRS vector is increased above >2 mV.

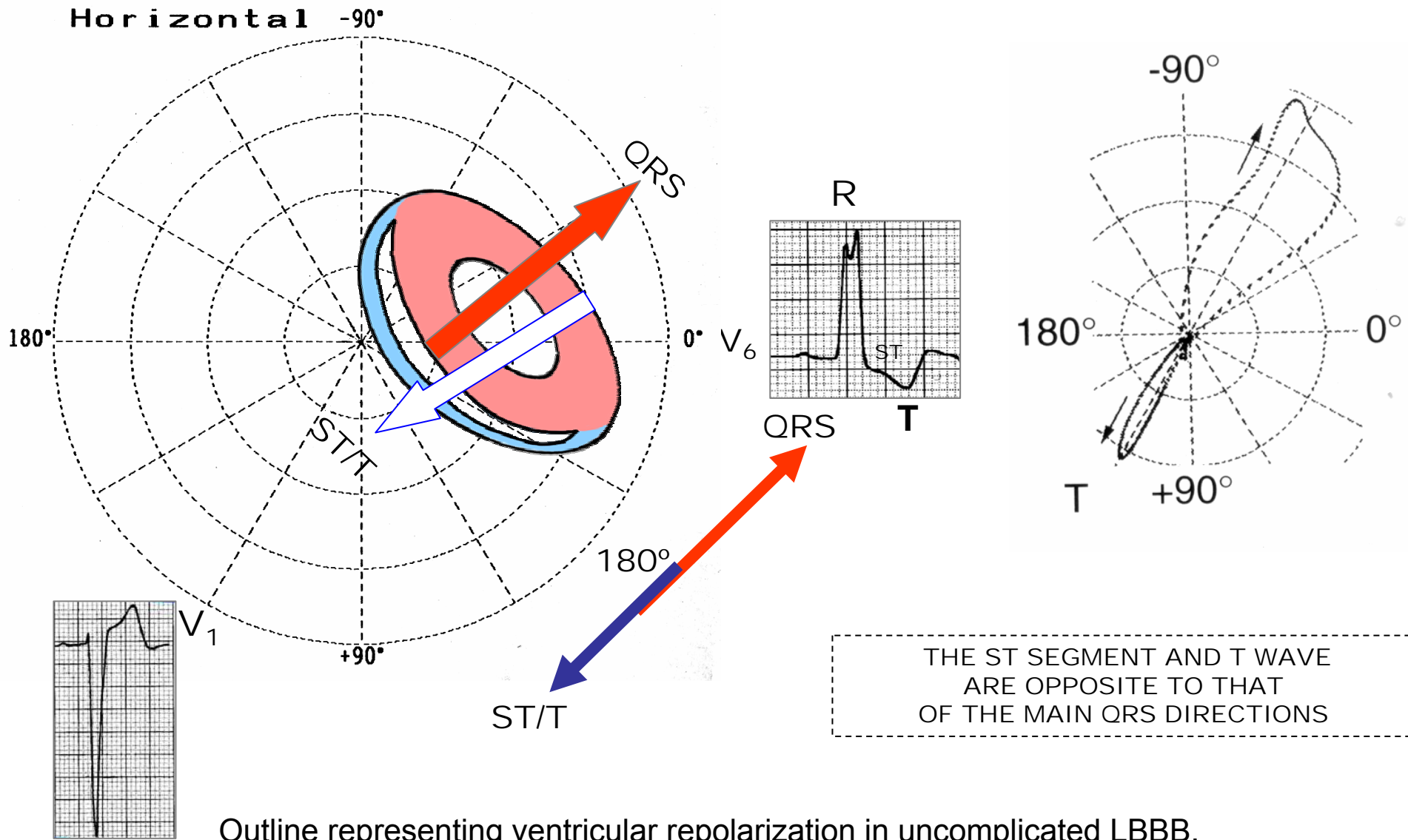
INITIAL 10ms VECTOR
 DIRECTED ANTERIORLY
 AND LEFTWARD

ST segment and T wave
 vector are directed rightward
 and anteriorly

T loop with an elliptical or linear
 aspect, inscribed clockwise and
 with slow inscription of the
 efferent limb and rapid inscription
 of the afferent limb, directed away
 from the terminal vectors of the
 QRS loop.



VENTRICULAR REPOLARIZATION IN UNCOMPLICATED LBBB



THE ST SEGMENT AND T WAVE
ARE OPPOSITE TO THAT
OF THE MAIN QRS DIRECTIONS

Outline representing ventricular repolarization in uncomplicated LBBB.
Secondary alteration of ventricular repolarization is observed: QRS/ST-T angle near 180°.

THE ST-SEGMENT ELEVATION CHARACTERISTICS IN UNCOMPLICATED LBBB

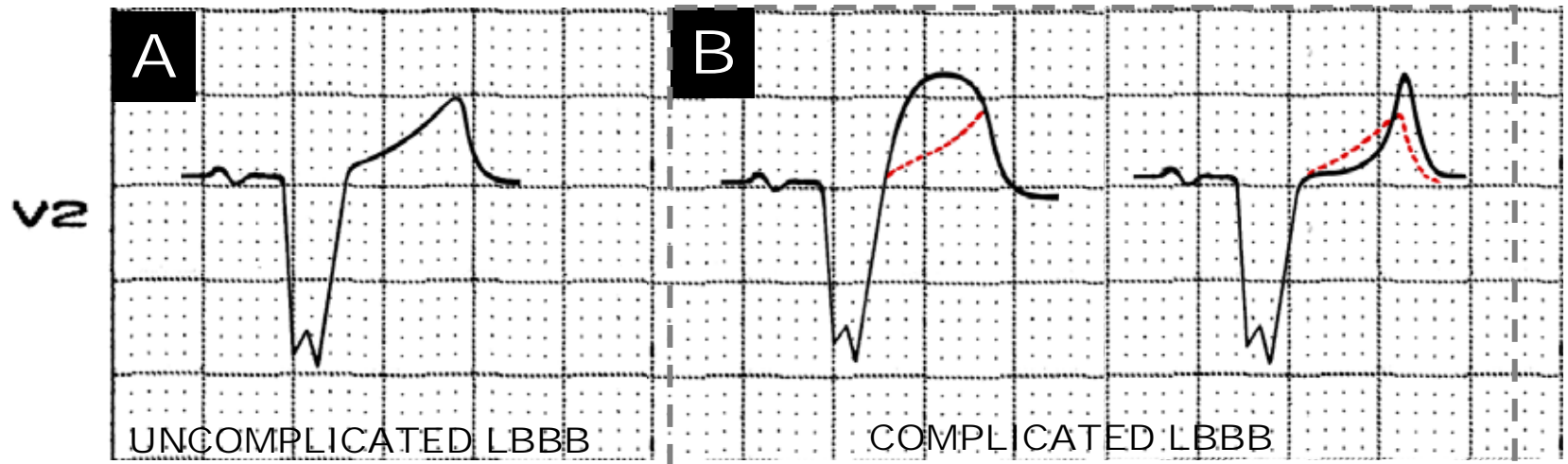
- In uncomplicated LBBB the ST segment and T wave are normally displaced in a direction opposite to that of the main QRS deflection.
- Positive ST segment displacement in the right precordial lead (V_1 and V_2) is much more difficult to evaluate, since ST segment elevation in these leads may occur in uncomplicated LBBB alone.
- Stable ≥ 5 mm ST elevation are occasionally found in leads with predominantly negative QRS complexes, particularly of large amplitude in the absence of AMI. In such patients presenting with symptoms suggestive of AMI, further non-ECG confirmation of probable underlying AMI should be sought¹.

AMI: Acute Myocardial Infarction

1) Madias JE, et al. A critique of the new ST-segment criteria for the diagnosis of acute myocardial infarction in patients with left bundle-branch block. Clin Cardiol. 2001; 24: 652-655.

IDENTIFICATION OF ACUTE CORONARY SYNDROMES IN PATIENTS WITH LEFT BUNDLE BRANCH BLOCK

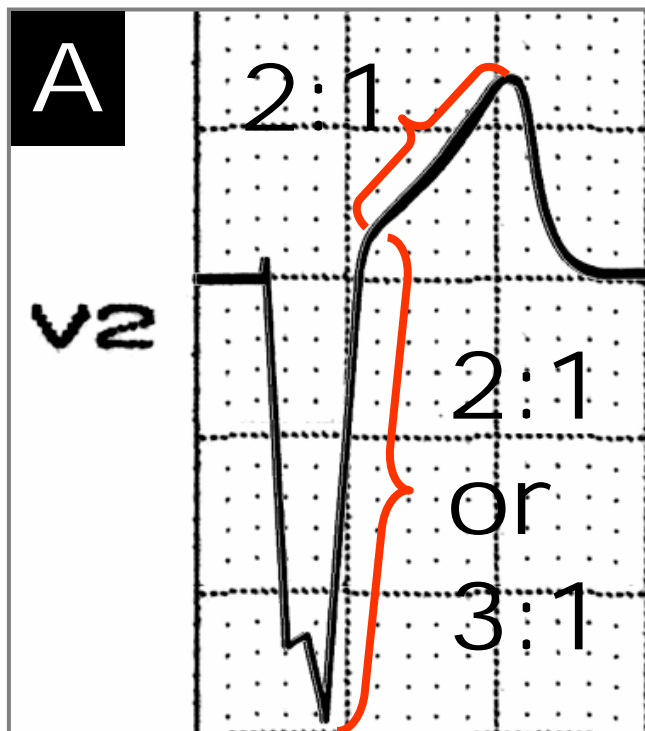
ST-SEGMENT AND T WAVE CHARACTERISTICS ON RIGHT PRECORDIAL LEADS (V_1 - V_2) IN UNCOMPLICATED(A) AND COMPLICATED LBBB(B)



- **A:** The elevated ST segment has a straight upward slope, or an upward slope that is minimally concave-upwards.
The T wave is upright, with asymmetrical limbs and a relatively blunt apex.
- **B:** With AMI the ST segment elevation is exaggerated (≥ 5 mm) in the right precordial leads and becomes coved, convex-upward.
The T wave becomes inverted and/or its limbs tend to become more symmetrical.

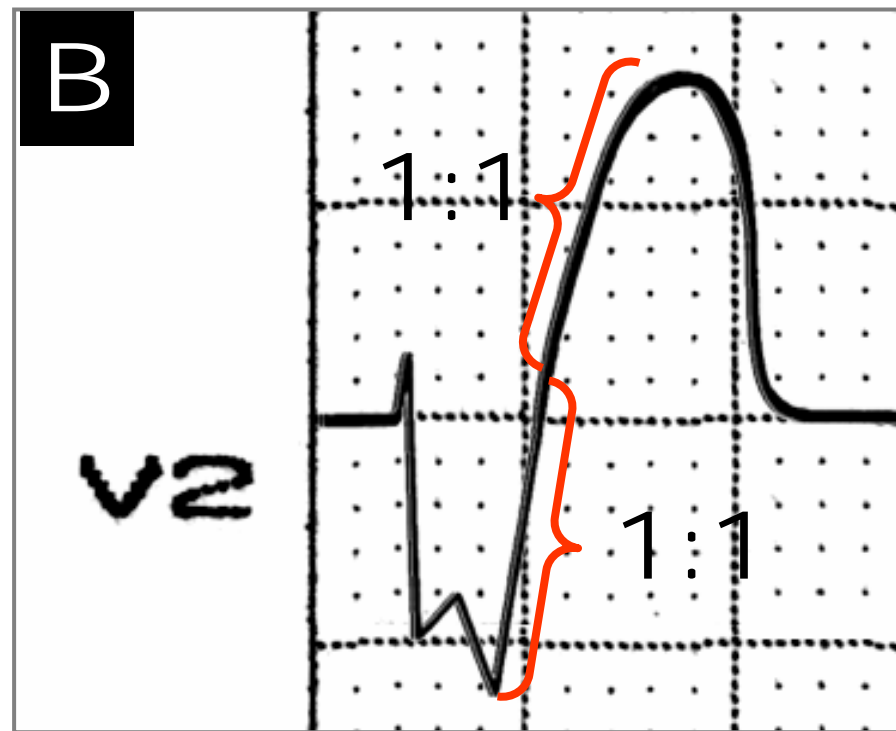
OTHERS ECG CRITERIA IN LBBB COMPLICATED WITH AMI

UNCOMPLICATED LBBB



A: Ratio of QRS_{ST-T} amplitude, 2:1. ST upwardly concave.

LBBB COMPLICATED WITH ANTERIOR MI

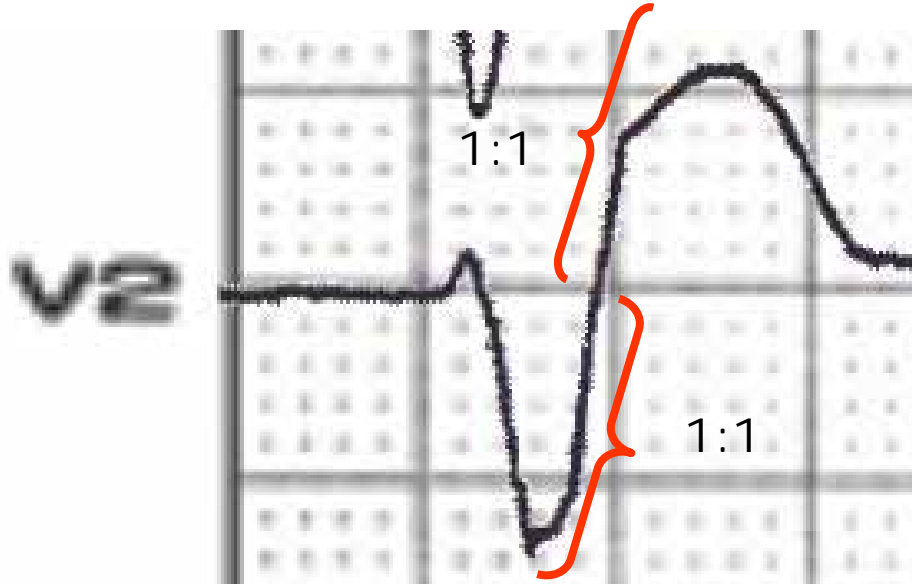


B: Ratio of QRS_{ST-T} amplitude 1:1. ST upwardly convex.

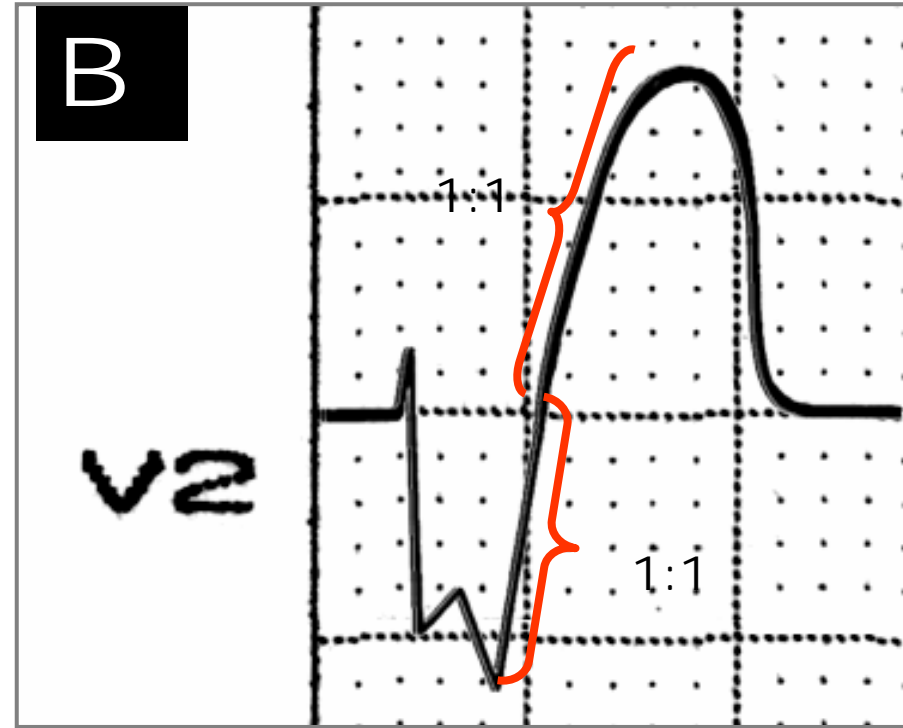
Diminution of QRS_{ST-T} ratio in lead V2: In uncomplicated LBBB, the ratio of QRS voltage to the ST segment voltage is always greater than 1. Usually 2:1 or 3:1 in V2 lead¹. During AMI the elevation of ST segment with concomitant eventual reduction in the QRS voltage results in a QRS_{ST-T} ratio near to 1:1.

- 1) **Schamroth L. The Electrocardiology of Coronary Artery Disease Myocardial Infarction Associated with Left Bundle Branch Block 1975; Chapter 10 pp: 93 Blackwell Scientific Publications.**

THE PRESENT CASE



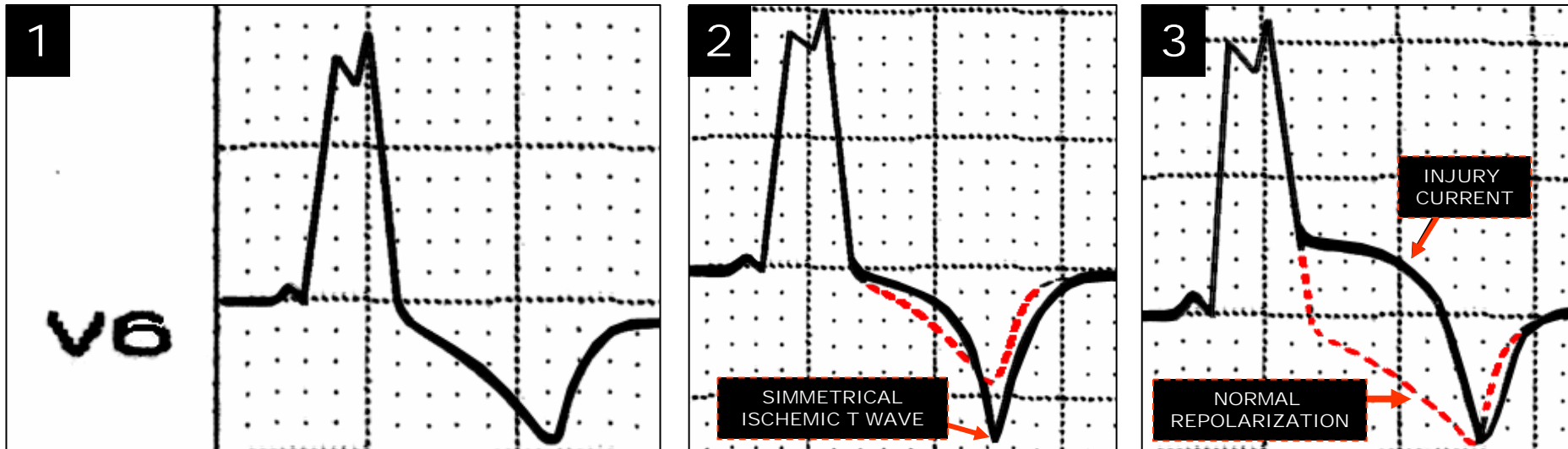
LBBB COMPLICATED WITH ANTERIOR MI



Diminution of QRS/ST-T ratio in lead V2: In uncomplicated LBBB, the ratio of QRS voltage to the ST segment voltage is always greater than 1. Usually 2:1 or 3:1 in V2 lead. During AMI the elevation of ST segment with concomitant eventual reduction in the QRS voltage results in a QRS/ST-T ratio near to 1:1.

LEFT PRECORDIAL LEADS (V_5 - V_6) IN UNCOMPLICATED LBBB (1) AND LBBB ASSOCIATED TO ACUTE CORONARY SYNDROME (2, 3)

V_6 lead in uncomplicated LBBB and associated to injury, ischemia and/or infarction.



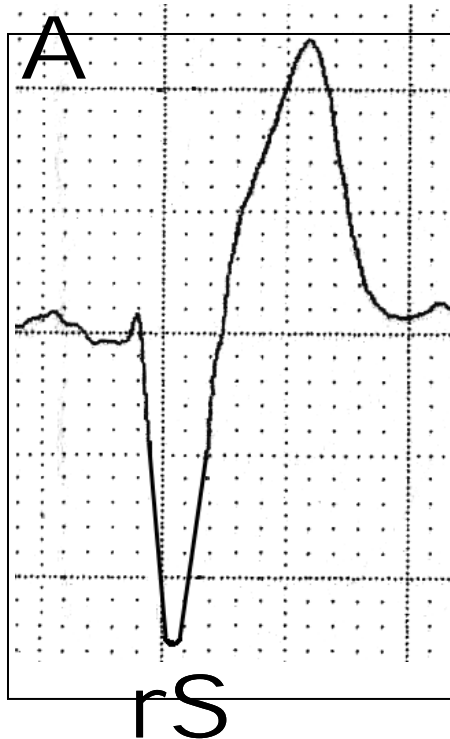
1: Habitual QRS-S/T in uncomplicated LBBB in V_6 Lead.

2: LBBB with ischemia (red dots indicate normal T shape).

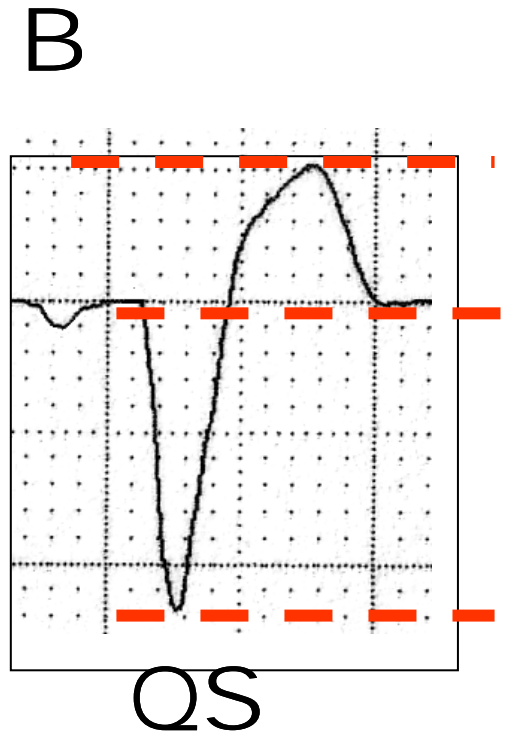
3: LBBB Associated to Antero-lateral Infarction: ST segment elevation convex to the top: subepicardial injury (red dots represent normal repolarization).

POSSIBLE QRS MORPHOLOGIES IN RIGHT PERICARDIAL LEADS (V_1 - V_2) IN UNCOMPLICATED LBBB

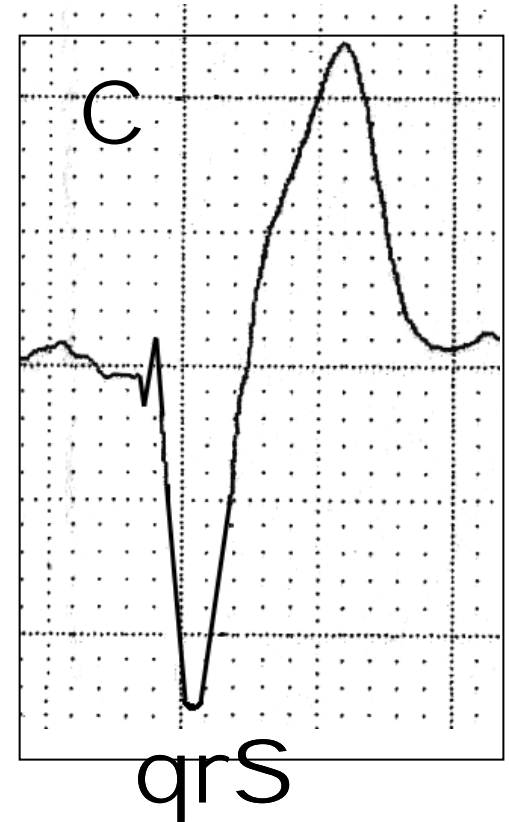
70%



>29%



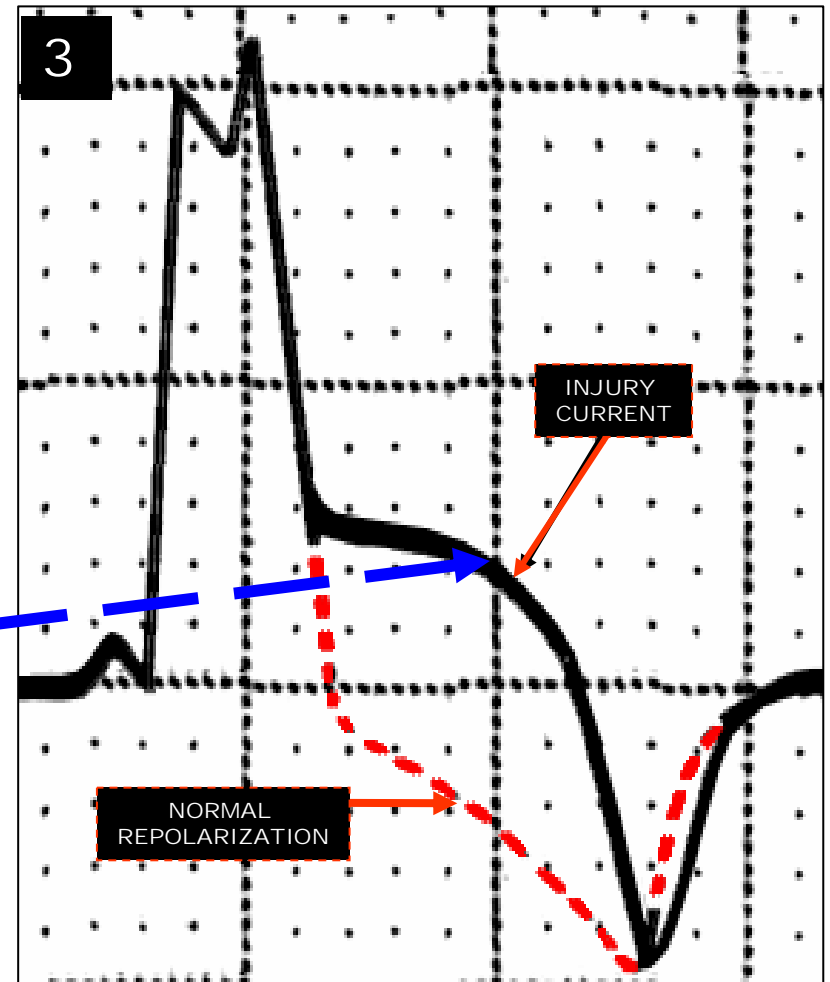
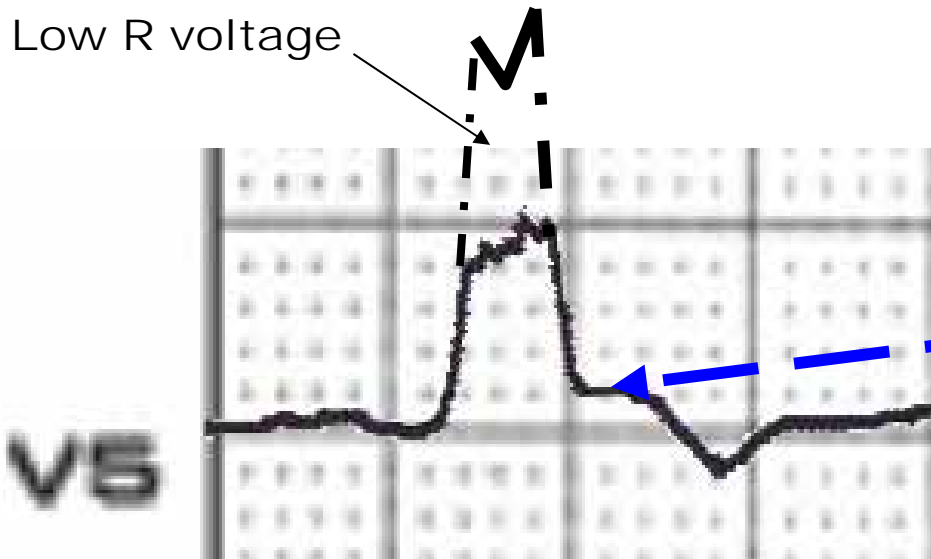
<1%



The three possible QRS morphologies in $V_1 - V_2$ in uncomplicated LBBB:
rS (70%), QS (>29%) and qrS (<1%).

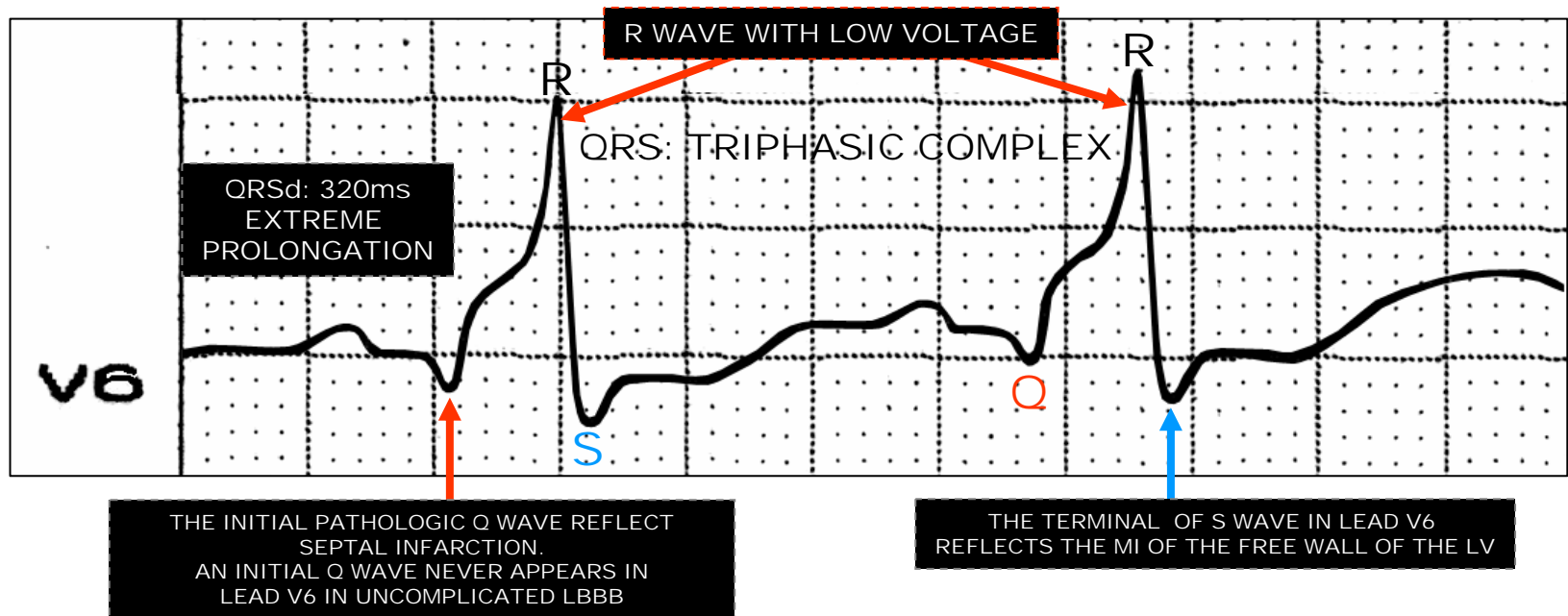
The present case

Low R voltage



ST-segment elevation of ≥ 1 mm in the presence of a positive QRS complex ≥ 1 mm of concordant ST-elevation (in the same direction as the majority of the QRS complex) in at least one lead. Low R voltage= transmural necrosis.

THE "DOME AND DART" QRS COMPLEX CONFIGURATION: LBBB COMPLICATED WITH MASSIVE SEPTAL MYOCARDIAL INFARCTION

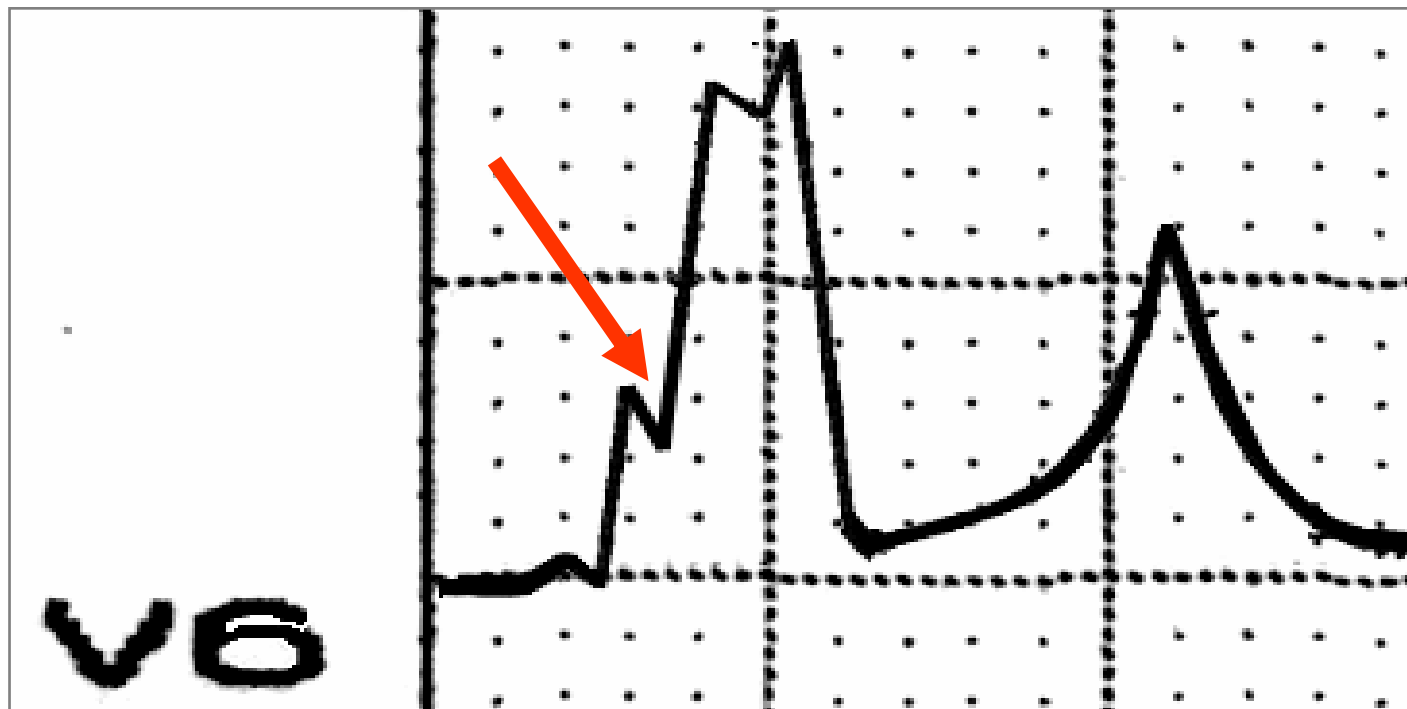


Characterized by a QRS complex observable in V6, formed by initial q wave followed by a positive deflection of low voltage of the "dome and dart" type, and final s wave. It indicates LBBB complicated with extensive anterior and anterolateral MI.

1. Schamroth L. *Electrocardiography Excursions. Vulgaria et Exotica. 50 studies in Electrocardiography Detection. Study 9 – The "Dome and Dark" QRS complex*; pg 23-25. Blackwell Scientific Publication. Oxford, London, Edinburgh, Melbourne; 1975.

The morphology in V6 was called "dome and dart" by Schamroth L. Characteristic of LBBB complicated with extensive anterior or antero-lateral infarction, or sign known in the Anglo-Saxon literature as "the dome and dart QRS complex configuration."

CHAPMAN'S SIGN: LBBB COMPLICATED TO ANTERIOR INFARCTION



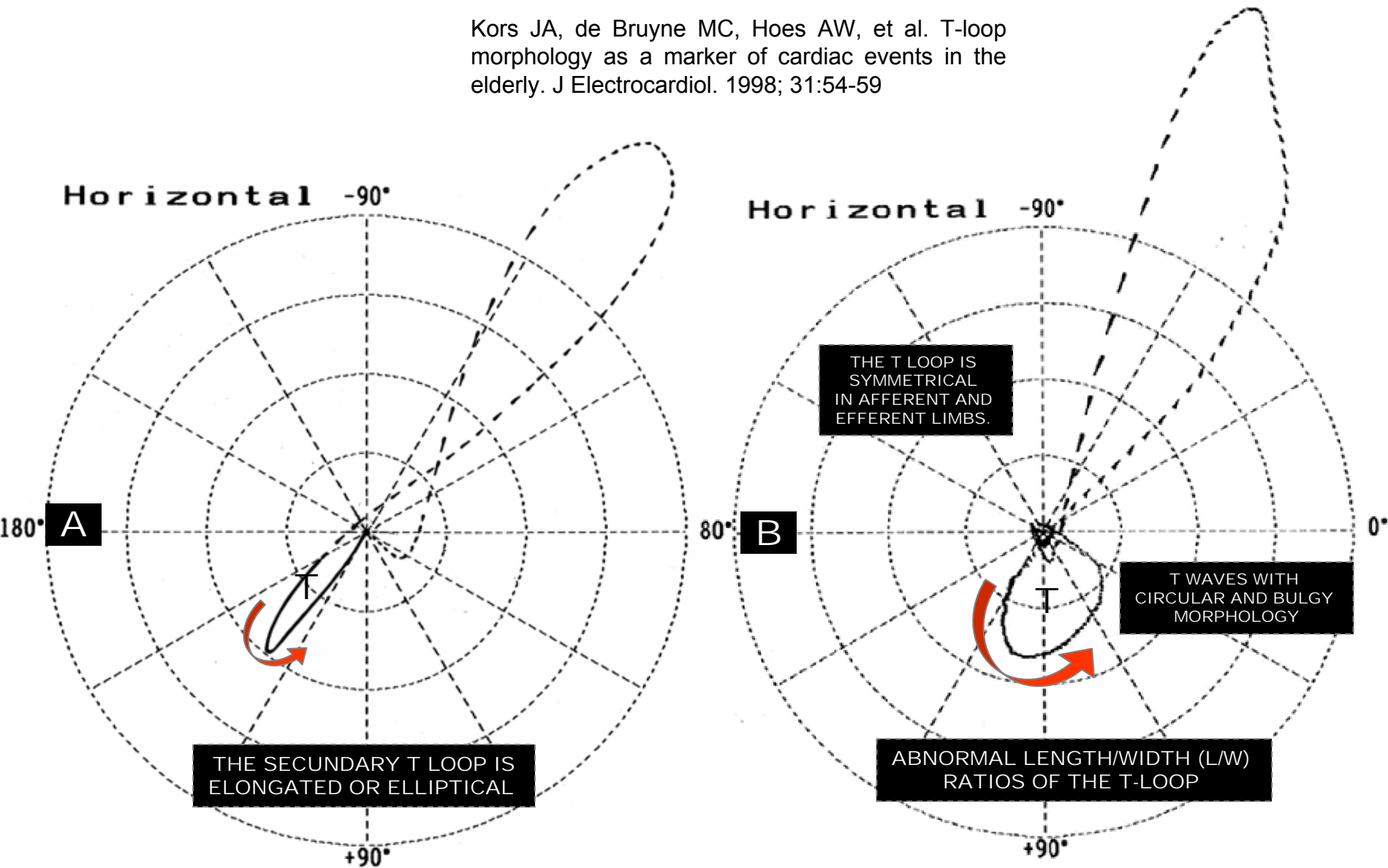
Notch in ascending ramp of R wave in DI, aVL, V₅ and/or V₆.

Chapman's sign of LBBB associated to anterior infarction:
notch in the ascending ramp of R wave in the left leads.

1. Chapman MG, Pearce MI. Electrocardiographic diagnosis of myocardial infarction in the presence of leftbundle-branch block. *Circulation*. 1957; 16: 558-571

NORMAL (A) AND ISCHEMIC (B) T LOOP ON HP

Kors JA, de Bruyne MC, Hoes AW, et al. T-loop morphology as a marker of cardiac events in the elderly. J Electrocardiol. 1998; 31:54-59



MAIN EPIDEMIOLOGIC FEATURES IN LBBB ASSOCIATED WITH ACS

- ✓ Patients with LBBB comprise 5-9% of all patients with AMI¹.
- ✓ The presence of ST elevation of at least 1mm in the limb leads or 2mm in the chest leads on the ECG².
- ✓ A new LBBB occurs in 2-3% of patients with AMI². A new LBBB is an independent predictor of all major adverse cardiovascular outcomes during long-term follow-up³.
- ✓ LBBB, Ventricular Paced Rhythm and Left Ventricular Hypertrophy reduce the ability of the ECG to detect Acute Coronary Syndrome change and AMI⁴.
- ✓ The development of new BBB during AMI is associated with a poor immediate and long term prognosis. This may be related to larger infarcts rather than the conduction defect itself⁵.

- 1) Gunnarsson G, Eriksson P, Dellborg M. Continuous ST-segment monitoring of patients with left bundle branch block and suspicion of acute myocardial infarction. J Intern Med. 2004; 255:571-578.
- 2) Lie KI, Wellens HJ, Schuilenberg RM, Bundle branch block and myocardial infarction. In Wellens HJJ, Lie K, Janse MJ, eds. The Conduction System of the Heart: Structure, Function and Clinical Applications. Leiden: Stenfert Kroese, 1976: 662-672.
- 3) Stephenson K, Skali H, McMurray JJ, et al. Long-term outcomes of left bundle branch block in high-risk survivors of acute myocardial infarction: The VALIANT experience. Heart Rhythm. 2007; 4:308-313.
- 4) Brady WJ, Chan TC, Pollack M. Electrocardiographic manifestations: patterns that confound the EKG diagnosis of acute myocardial infarction-left bundle branch block, ventricular paced rhythm, and left ventricular hypertrophy. J Emerg Med. 2000; 18: 71-78.
- 5) Hassi M, Kunstmann S, Corbalán R, et al. Intraventricular conduction disorders in acute myocardial infarction: early and late clinical significance Rev Med Chil. 1989; 117:1381-1386

THROMBOLYSIS IN PATIENTS WITH A NEW OR RECENT ONSET LBBB

There are two indications for initiating thrombolysis in a patient with cardiac chest pain:

- ✓ The presence of ST elevation of at least 1mm in the limb leads or 2mm in the chest leads on the ECG.
- ✓ A LBBB pattern.
- ✓ There must also be no compelling contraindications to thrombolysis. When meeting a patient for the first time it can be difficult to establish whether the onset of LBBB is new, without a previous ECG, this must be particularly difficult in the pre-hospital setting.
- ✓ Thrombolytic drugs are not without risk and careful questioning must be carried out to ensure that they are given appropriately.

THROMBOLYSIS IN PATIENTS WITH A NEW OR RECENT ONSET LBBB

- Fibrinolytic therapy is recommended for patients who have chest pain and LBBB. However, the presence of baseline ECG abnormalities makes early accurate identification of AMI difficult. Additionally, nearly 50% of AMI patients with LBBB present without chest pain.
- LBBB patients with AMI who present without chest pain are less likely to receive optimal therapy and are at increased risk of death¹.
- Prompt recognition and treatment of this high-risk subgroup should improve survival.
- The clinical history is not effective at distinguishing LBBB patients with AMI among patients who appeared to be candidates for acute reperfusion therapy².

- 1) Shlipak MG, Go AS, Frederick PD, et al. Treatment and outcomes of left bundle-branch block patients with myocardial infarction who present without chest pain. National Registry of Myocardial Infarction 2 Investigators. J Am Coll Cardiol. 2000;36:706-712.
- 2) Shlipak MG, Go AS, Lyons WL, et al. Clinical Symptoms and Myocardial Infarction in Left Bundle Branch Block Patients. Cardiology 2000;93:100-104

THE USE OF ECGs CRITERIA, BASED ON SIMPLE ST-SEGMENT CHANGES

- Sgarbossa et al. proposed specific ECG criteria for the diagnosis of AMI in the presence of LBBB based on the criteria performance as applied to 131 patients in the GUSTO-1 trial (*Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries*) who had AMI and LBBB in comparison to patients from the Duke database who had LBBB and were clinically stable. The application of the most efficient of the criteria was associated with a high specificity and low sensitivity.
- The base-line ECGs of patients enrolled in the GUSTO-1 T trial who had LBBB and AMI confirmed by enzyme studies were blindly compared with the ECGs of control patients who had chronic coronary artery disease and LBBB.
- Of 26,003 patients, 131 (0.5%) with AMI had LBBB¹.

1. Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. N Engl J Med. 1996; 334:481-487

THE SGARBOSSA'S CRITERIA

- 1) ST-segment elevation of at least 1 mm (≥ 1 mm) in the lead with concordant QRS complex or ST-segment elevation of ≥ 1 mm in the presence of a positive QRS complex a score of 5 points.
- 2) ST-segment depression of at least 1 mm (≥ 1 mm) in lead V1, V2 or V3--a score of 3 points. ST-segment depression of ≥ 1 mm in lead V1, V2 or V3.
- 3) ST-segment elevation of at least 5 mm (≥ 5 mm) in the presence of a negative QRS complex (discordant with QRS complex) a score of 2 points.

Enumeration of the criteria by Sgarbossa et al, for the diagnosis of LBBB associated to AMI.

In Sgarbossa study, the clinical prediction rule score values of these signs were 5; 3; and 2, respectively.

A score ≥ 3 made a diagnosis of AMI with a 90% specificity and a score of 2 with $> 80\%$, specificity.

VALUE OF THE ECG AS GUIDE THERAPY IN PATIENTS WITH LBBB AND SUSPECTED AMI

- The ECG is a poor predictor of MI in a community-based cohort of patients with LBBB and acute cardiopulmonary symptoms.
- The ECG cannot reliably be used to rule out AMI in patients with LBBB¹.
- Patients with LBBB and symptoms of AMI should receive reperfusion therapy if there are no contraindications².
- Acute thrombolytic therapy should be considered for all patients with LBBB who have symptoms consistent with AMI.

- 1) Shlipak MG, Lyons WL, Go AS, et al. Should the electrocardiogram be used to guide therapy for patients with left bundle-branch block and suspected myocardial infarction? JAMA, 1999; 281:714-719.
- 2) Pollack CV, Diercks DB, Roe MT, Peterson ED; American College of Cardiology; American Heart Association. 2004 American College of Cardiology/American Heart Association guidelines for the management of patients with ST-elevation myocardial infarction: implications for emergency department practice. Ann Emerg Med. 2005; 45:363-376.

THE ECG SGARBOSSA CRITERIA SENSITIVITY AND SPECIFICITY. VALUE OF SERUM BIOMARKERS

- The criteria of Sgarbosa are too insensitive to be used as screening (roule out) test to determine which patients with an LBBB do not have an AMI.
- The Sgarbosa criteria are, however, highly specific and can be used reliably as confirmatory test to rule in AMI in patients with LBBB. ECG alone doesn't support the diagnosis of AMI.
- Elevated value of biochemical markers of myocardial necrosis in the presence of LBBB confirms the diagnosis. Currently the best justified strategy is to follow American Heart Association (AHA) and American College of Cardiology (ACC) recommended guidelines to administer thrombolysis to all patients with LBBB presenting with chest pain, particularly if serum biomarkers are elevated¹.

1) Jakuitis A, Statkeviciene A. The importance of left bundle branch block in the diagnosis of acute myocardial infarction Medicina (Kaunas). 2003; 39: 15-20.

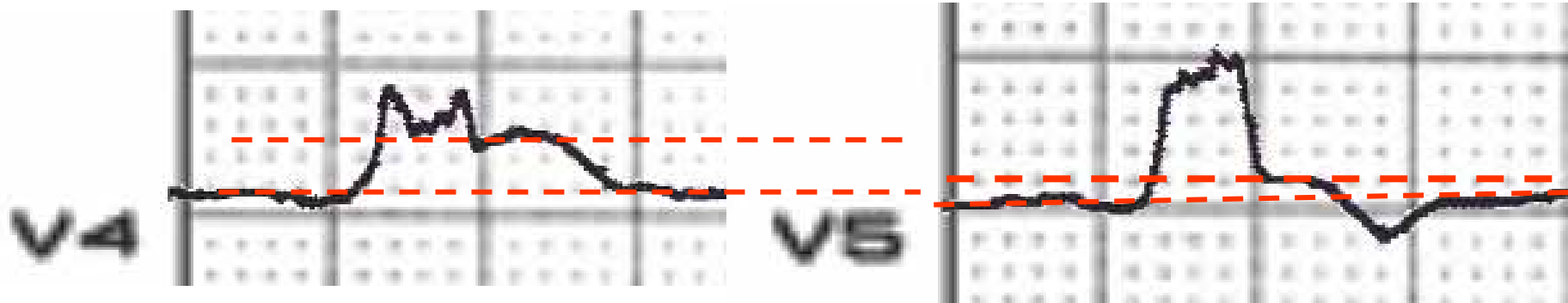
THROMBOLYTIC TREATMENT IN PATIENTS WITH LBBB AND AMI

- **Currently, thrombolytic treatment is under-utilized in patients with LBBB and AMI, and those who are thrombolyzed endure lengthy delays before treatment.**
- **Patients with any of the predictive criteria should be thrombolyzed immediately. When the diagnosis is in doubt, if doubt persists, serial ECGs may show evolving ischemic changes.(1)**
- **Intravenous enoxaparin compared with unfractionated heparin significantly reduced clinical ischaemic outcomes without differences in bleeding and procedural success. Therefore, enoxaparin provided an improvement in net clinical benefit in patients undergoing primary PCI.(2)**

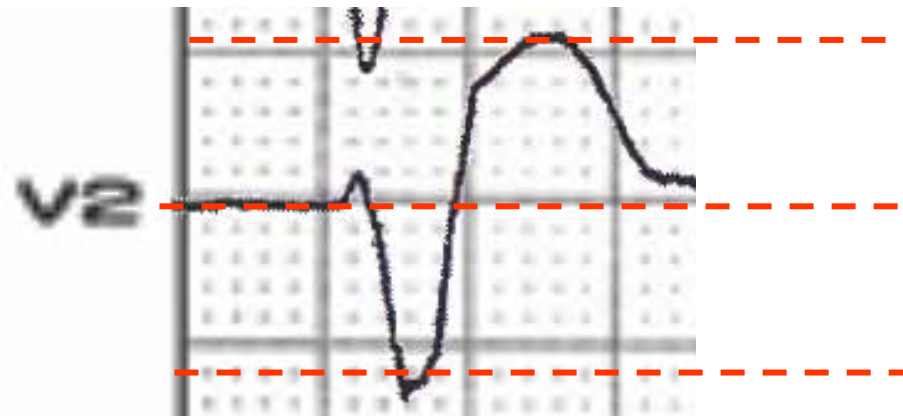
- 1) **Edhouse JA, Sakr M, Angus J, et al. Suspected myocardial infarction and left bundle branch block: electrocardiographic indicators of acute ischaemia. J Accid Emerg Med. 1999; 16:331-335.**
- 2) **Montalescot G, Zeymer U, Silvain J, et al. Intravenous enoxaparin or unfractionated heparin in primary percutaneous coronary intervention for ST-elevation myocardial infarction: the international randomised open-label ATOLL trial. Lancet. 2011 Aug 20;378: 693-703.**

ECG diagnosis

- 1) Complete Left Bundle Branch Block
- 2) ST elevation of at least 1 mm (≥ 1 mm) in the lead with concordant QRS complex or ST-segment elevation of ≥ 1 mm in the presence of a positive QRS complex a score of 5 points.

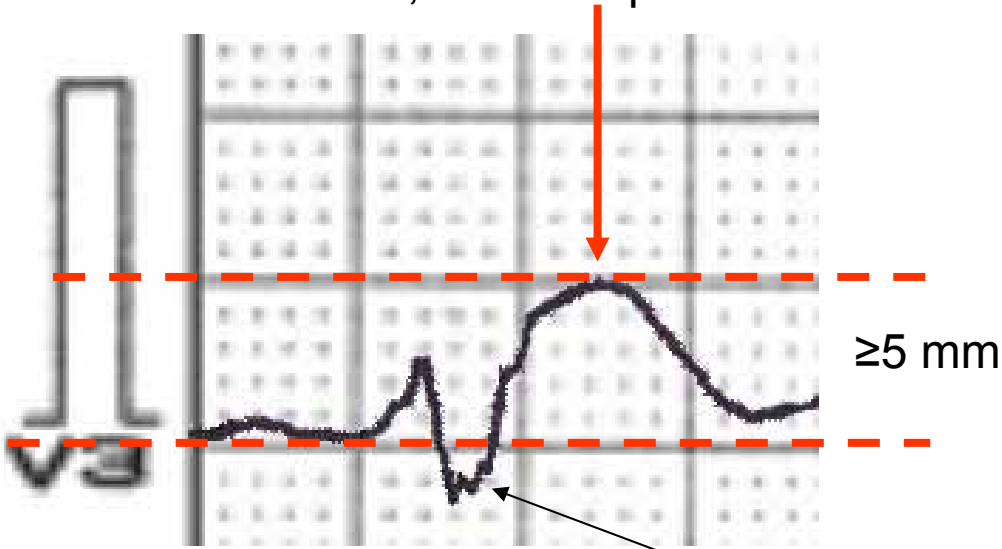


- 3) Diminution of QRS/ST-T ratio in lead V_2 : In uncomplicated LBBB, the ratio of QRS voltage to the ST segment voltage is always greater than 1. Usually 2:1 or 3:1 in V_2 lead¹. During AMI the elevation of ST segment with concomitant eventual reduction in the QRS voltage results in a QRS/ST-T ratio near to 1:1.



1. Schamroth L. The electrocardiology of coronary artery disease. Blackwell Scientific Publications. Oxford-London Edinburgh Melbourne. 1975; pg. 86.

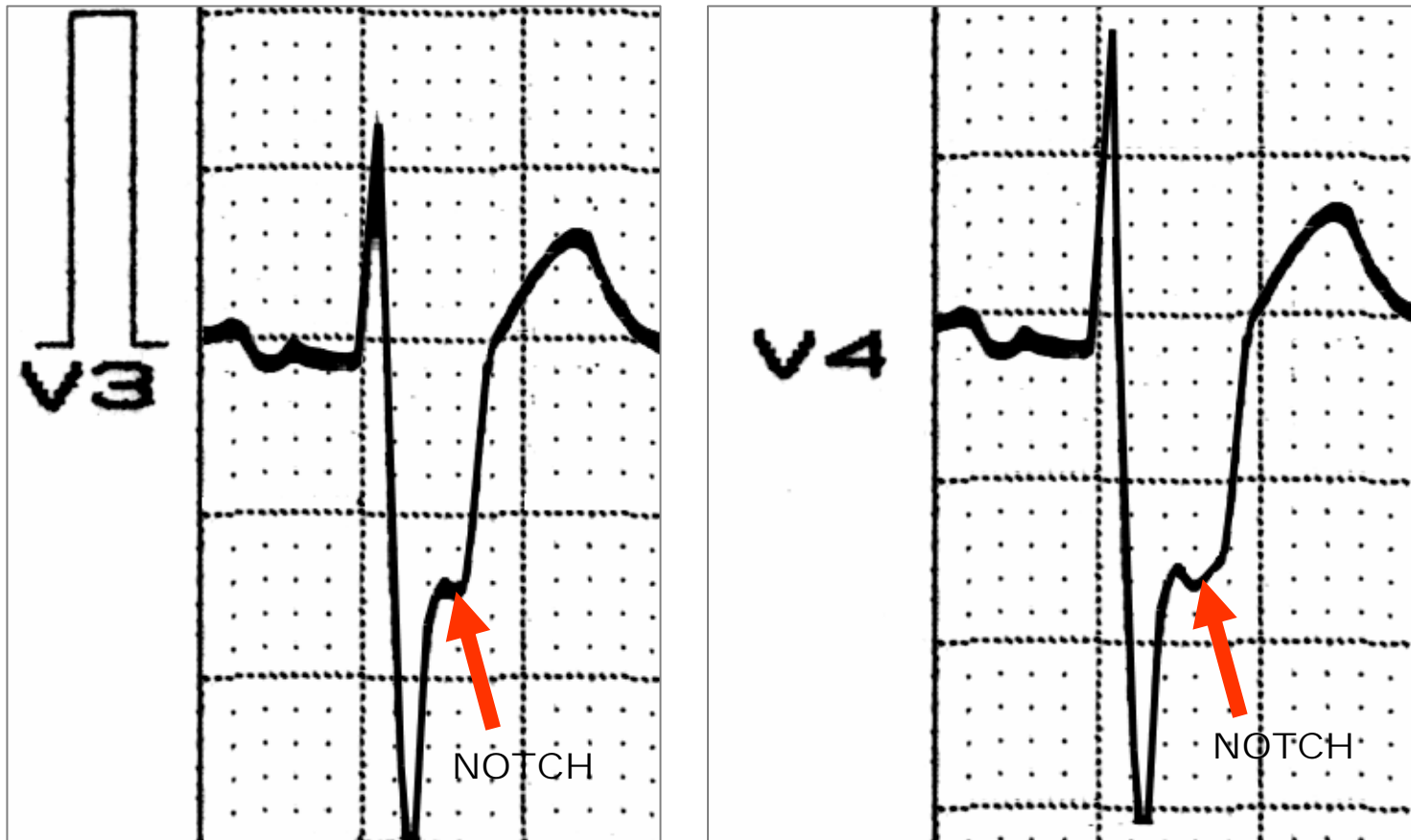
- 4) With AMI the ST segment elevation is exaggerated (≥ 5 mm) in the right precordial leads and becomes coved, convex-upward.



- 5) Notch of 50 ms in the ascending ramp of S wave of V3 and V4. It is seen more often with MI than without (anterior more often than inferior), and the left axis increased its sensitivity: Cabrera sign (see next slide)

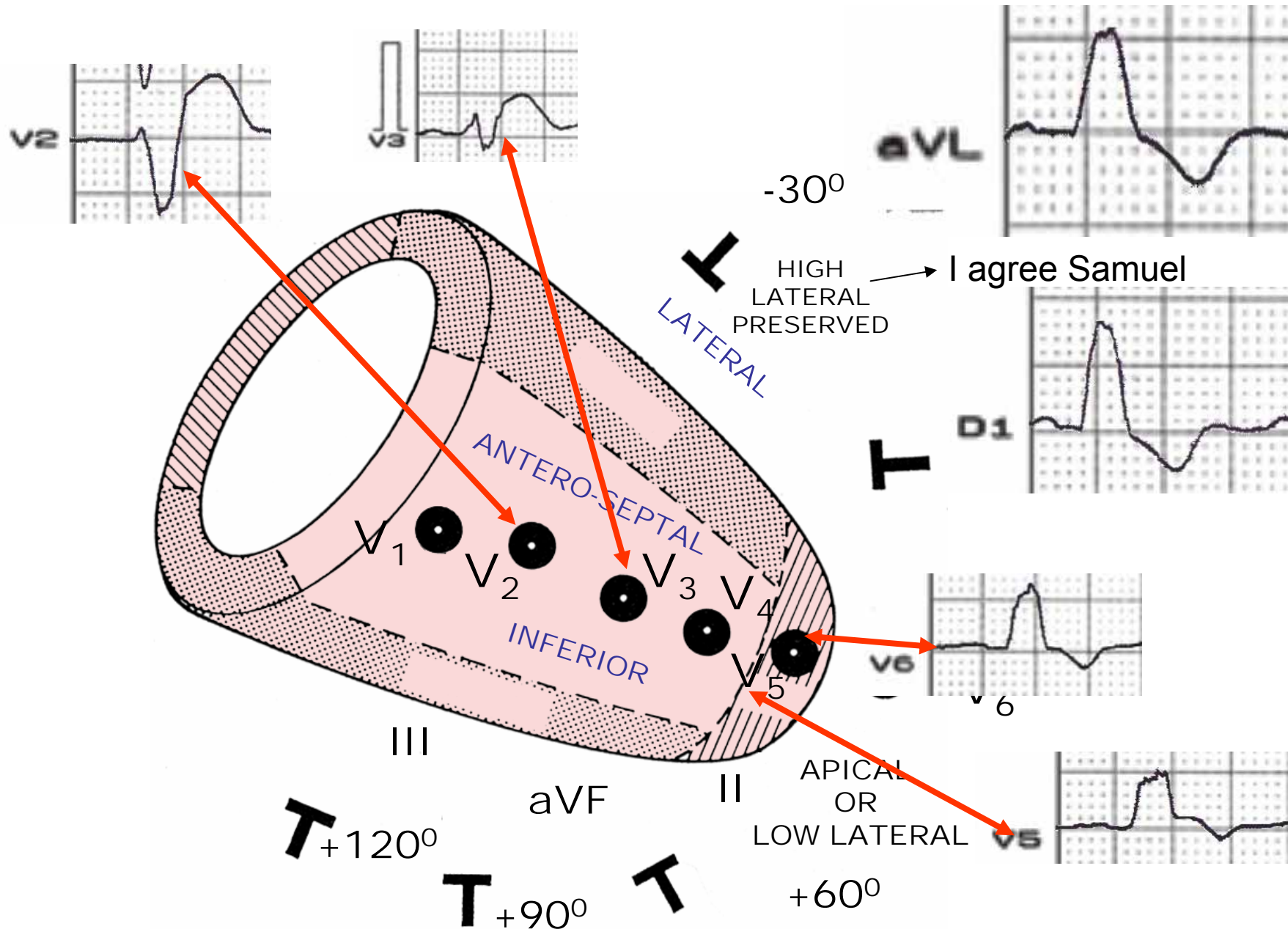
Conclusion: Complete LBBB + acute anteroapical MI

CABRERA'S SIGN: LBBB COMPLICATED WITH ANTERIOR INFARCTION



Notch of 50 ms in the ascending ramp of S wave of V_3 and V_4 . It is seen more often with MI than without (anterior more often than inferior), and the left axis increased its sensitivity^{1,2}.

- 1) Kindwall KE, Brown JP, Josephson ME. Predictive accuracy of criteria for chronic myocardial infarction in pacing-induced left bundle branch block. *Am J Cardiol.* 1986; 57:1255-1260.
- 2) Cabrera E, Friedland C. Wave of ventricular activation in left bundle branch block with infarct; a new electrocardiographic sign. *Gac Med Mex.* 1953; 83:273-280.



The cardiac cone, its sides and corresponding leads: Antero-septal: V1 to V4; Apical or lowlateral: V5 and V6; High lateral: DI and aVL; Inferior or diaphragmatic: II, DIII and aVF; Dorsal, posterior or postero-basal: V7 and V8.

ACUTE CORONARY SYNDROME OVERVIEW

Acute Coronary Syndrome (ACS) is a continuum of disease that includes non-ST-segment elevation ACS and ST-segment elevation myocardial infarction(MI). refers to a spectrum of clinical presentations ranging from those for ST-segment elevation myocardial infarction (STEMI) to presentations found in non–ST-segment elevation myocardial infarction (NSTEMI) or in unstable angina. In terms of pathology, ACS is almost always associated with rupture of an atherosclerotic plaque and partial or complete thrombosis of the infarct-related artery. In some instances, however, stable coronary artery disease (CAD) may result in ACS in the absence of plaque rupture and thrombosis, when physiologic stress (eg, trauma, blood loss, anemia, infection, tachyarrhythmia) increases demands on the heart. The diagnosis of acute myocardial infarction (AMI) in this setting requires a finding of the typical rise and fall of biochemical markers of myocardial necrosis in addition to at least 1 of the following(1):

1. Ischemic symptoms
2. Development of pathologic Q waves
3. Ischemic ST-segment changes on ECG or in the setting of a coronary intervention.

ACS is usually one of three diseases involving the coronary arteries: ST elevation MI (30%), non ST elevation MI (25%), or unstable angina (38%).(2). ACS should be distinguished from *stable* angina, which develops during exertion and resolves at rest. In contrast with stable angina, unstable angina occurs suddenly, often at rest or with minimal exertion, or at lesser degrees of exertion than the individual's previous angina ("crescendo angina"). New onset angina is also considered unstable angina, since it suggests a new problem in a coronary artery

1. Alpert JS, Thygesen K, Antman E, Bassand JP. Myocardial infarction redefined--a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. *J Am Coll Cardiol.* Sep 2000;36:959-969.
2. Torres M, Moayed S "Evaluation of the acutely dyspneic elderly patient". *Clin. Geriatr. Med.* May 2007; 23 : 307–325.

The terms transmural and nontransmural (subendocardial) myocardial infarction are no longer used because ECG findings in patients with this condition are not closely correlated with pathologic changes in the myocardium. Therefore, a transmural infarct may occur in the absence of Q waves on ECGs, and many Q-wave myocardial infarctions may be subendocardial, as noted on pathologic examination. Because elevation of the ST segment during ACS is correlated with coronary occlusion and because it affects the choice of therapy (urgent reperfusion therapy), ACS-related MI should be designated STEMI or NSTEMI. Attention to the underlying mechanisms of ischemia is important when managing ACS. A simple predictor of demand is rate-pressure product, which can be lowered by β -blockers (eg, metoprolol or atenolol) and pain/stress relievers (eg, morphine), while supply may be improved by oxygen, adequate hematocrit, blood thinners (eg, heparin, IIb/IIIa agents such as abciximab, eptifibatide, tirofiban, or thrombolytics), and/or vasodilators (eg, nitrates, amlodipine). (See Medications.)

In 2010, the American Heart Association (AHA) published new guideline recommendations for the diagnosis and treatment of ACS.

ETIOLOGY

ACS is caused primarily by atherosclerosis. Most cases of ACS occur from disruption of a previously nonsevere lesion (an atherosclerotic lesion that was previously hemodynamically insignificant yet vulnerable to rupture). The vulnerable plaque is typified by a large lipid pool, numerous inflammatory cells, and a thin, fibrous cap. Elevated demand can produce ACS in the presence of a high-grade fixed coronary obstruction, due to increased myocardial oxygen and nutrition requirements, such as those resulting from exertion, emotional stress, or physiologic stress (eg, from dehydration, blood loss, hypotension, infection, thyrotoxicosis, or surgery). ACS without elevation in demand requires a new impairment in supply, typically due to thrombosis and/or plaque hemorrhage.

The major trigger for coronary thrombosis is considered to be plaque rupture caused by the dissolution of the fibrous cap, the dissolution itself being the result of the release of metalloproteinases (collagenases) from activated inflammatory cells. This event is followed by platelet activation and aggregation, activation of the coagulation pathway, and vasoconstriction. This process culminates in coronary intraluminal thrombosis and variable degrees of vascular occlusion. Distal embolization may occur. The severity and duration of coronary arterial obstruction, the volume of myocardium affected, the level of demand on the heart, and the ability of the rest of the heart to compensate are major determinants of a patient's clinical presentation and outcome. (Anemia and hypoxemia can precipitate myocardial ischemia in the absence of severe reduction in coronary artery blood flow.) A syndrome consisting of chest pain, ischemic ST-segment and T-wave changes, elevated levels of biomarkers of myocyte injury, and transient LV apical ballooning (takotsubo syndrome) has been shown to occur in the absence of clinical CAD, after emotional or physical stress. The etiology of this syndrome is not well understood but is thought to relate to a surge of catechol stress hormones and/or high sensitivity to those hormones. Though ACS is usually associated with coronary thrombosis, it can also be associated with cocaine use. Cardiac chest pain can also be precipitated by anemia, bradycardias or tachycardias.

PROGNOSIS

Six-month mortality rates in the Global Registry of Acute Coronary Events (GRACE) were 13% for patients with NSTEMI ACS and 8% for those with unstable angina.

An elevated level of troponin (a type of regulatory protein found in skeletal and cardiac muscle) permits risk stratification of patients with ACS and identifies patients at high risk for adverse cardiac events (ie, MI, death) up to 6 months after the index event.(1, 2)

The PROVE IT-TIMI trial found that after ACS, a J-shaped or U-shaped curve association is observed between BP and the risk of future cardiovascular events.(3)

LeLeiko et al determined that serum choline and free F(2)-isoprostane are also predictors of cardiac events in ACS. The authors evaluated the prognostic value of vascular inflammation and oxidative stress biomarkers in patients with ACS to determine their role in predicting 30-day clinical outcomes. Serum F(2)-isoprostane had an optimal cutoff level of 124.5 pg/mL, and serum choline had a cutoff level of 30.5 $\mu\text{mol/L}$. Choline and F(2)-isoprostane had a positive predictive value of 44% and 57% and a negative predictive value of 89% and 90%, respectively.(4)

1. Antman EM, Tanasijevic MJ, Thompson B, et al. Cardiac-specific troponin I levels to predict the risk of mortality in patients with acute coronary syndromes. *N Engl J Med*. Oct 31 1996;335:1342-1349.
2. Heidenreich PA, Alloggiamento T, Melsop K, et al. The prognostic value of troponin in patients with non-ST elevation acute coronary syndromes: a meta-analysis. *J Am Coll Cardiol*. Aug 2001;38:478-485.
3. Bangalore S, Qin J, Sloan S, et al. What is the optimal blood pressure in patients after acute coronary syndromes?: Relationship of blood pressure and cardiovascular events in the PRavastatin OR atorVastatin Evaluation and Infection Therapy-Thrombolysis In Myocardial Infarction (PROVE IT-TIMI) 22 trial. *Circulation*. Nov 23 2010;122:2142-2151.
4. LeLeiko RM, Vaccari CS, Sola S, Merchant N, Nagamia SH, Thoenes M, et al. Usefulness of elevations in serum choline and free F2)-isoprostane to predict 30-day cardiovascular outcomes in patients with acute coronary syndrome. *Am J Cardiol*. Sep 1 2009;104:638-643.

Testosterone deficiency is common in patients with coronary disease and has a significant negative impact on mortality. Further study is needed to assess the effect of treatment on survival.(1)

A study by Sanchis et al(2) suggests renal dysfunction, dementia, peripheral artery disease, previous heart failure, and previous myocardial infarction are the comorbid conditions that predict mortality in NSTEMI ACS. In patients with comorbid conditions, the highest risk period was in the first weeks after NSTEMI ACS. In-hospital management of patients with comorbid conditions merits further investigation.

In a study that assessed the impact of prehospital time on STEMI outcome, Chughatai et al (3) suggest that “total time to treatment” should be used as a core measure instead of “door-to-balloon time.” This is because on-scene time was the biggest fraction of “pre-hospital time.” The study compared groups with total time to treatment of more than 120 minutes compared with 120 minutes or less and found mortalities were 4 compared with 0 and transfers to a tertiary care facility were 3 compared with 1, respectively.

1. Ma RC, Tong PC. Testosterone levels and cardiovascular disease. *Heart*. Nov 2010;96:1787-1788.
2. Sanchis J, Nunez J, Bodí V, et al. Influence of comorbid conditions on one-year outcomes in non-ST-segment elevation acute coronary syndrome. *Mayo Clin Proc*. Apr 2011;86:291-6.
3. Chughtai H, Ratner D, Pozo M, et al. Prehospital delay and its impact on time to treatment in ST-elevation myocardial infarction. *Am J Emerg Med*. May 2011;29:396-400.

PATIENT EDUCATION

Patient education of risk factors is important, but more attention is needed regarding delays in door-to-balloon time, and one major barrier to improving this delay is patient education regarding his or her symptoms. Lack of recognition of symptoms may cause tremendous delays in seeking medical attention. Educate patients about the dangers of cigarette smoking, a major risk factor for CAD. The risk of recurrent coronary events decreases 50% at 1 year after smoking cessation. Provide all patients who smoke with guidance, education, and support to avoid smoking. Smoking-cessation classes should be offered to help patients avoid smoking after a myocardial infarction. Bupropion increases the likelihood of successful smoking cessation. Diet plays an important role in the development of CAD. Therefore, prior to hospital discharge, a patient who has had a myocardial infarction should be evaluated by a dietitian. Patients should be informed about the benefits of a low-cholesterol, low-salt diet. In addition, educate patients about AHA dietary guidelines regarding a low-fat, low-cholesterol diet. A cardiac rehabilitation program after discharge may reinforce education and enhance compliance. The following mnemonic may be useful in educating patients with CAD regarding treatments and lifestyle changes necessitated by their condition:

A = Aspirin and antianginals

B = β - blockers and blood pressure (BP)

C = Cholesterol and cigarettes

D = Diet and diabetes

E = Exercise and education

For patients being discharged home, emphasize the following:

Timely follow-up with primary care provider

Compliance with discharge medications, specifically aspirin and other medications used to control symptoms

Need to return to the ED for any change in frequency or severity of symptoms.

CAUSES AND RISK FACTORS

Acute coronary syndromes typically result from a disruption of an atherosclerotic plaque in a blood vessel.

Risk factors for ACS therefore are those of atherosclerosis, and include:

1. Increasing age
2. Hypertension
3. Smoking
4. Diabetes
5. High plasma LDL and low HDL
6. Elevated plasma triglycerides
7. Family history and genetic polymorphisms: 9p21, when homozygotic, increases the risk of CHD by 30-40% (McPherson et al, Science, 2007)
8. Infection and inflammation
9. Metabolic syndrome
10. Homocysteine
11. Substance abuse (particularly cocaine abuse)

Less common etiologies include any instance where there is significantly increased demand on the heart coupled with poor myocardial perfusion (i.e. trauma, massive blood loss, anemia, infection, etc.).

HISTORY

The severity and duration of coronary artery obstruction, the volume of myocardium affected, the level of demand, and the ability of the rest of the heart to compensate are major determinants of a patient's clinical presentation and outcome. A patient may present to the ED because of a change in pattern or severity of symptoms. Typically, angina is a symptom of myocardial ischemia that appears in circumstances of increased oxygen demand. It is usually described as a sensation of chest pressure or heaviness that is reproduced by activities or conditions that increase myocardial oxygen demand. A new case of angina is more difficult to diagnose because symptoms are often vague and similar to those caused by other conditions (eg, indigestion, anxiety). However, not all patients experience chest pain. They may present with only neck, jaw, ear, arm, or epigastric discomfort. Some patients, including some who are elderly or who have diabetes, present with no pain, complaining only of episodic shortness of breath, severe weakness, light-headedness, diaphoresis, or nausea and vomiting. Elderly persons may also present only with altered mental status. Those with preexisting altered mental status or dementia may have no recollection of recent symptoms and may have no complaints. In addition, evidence exists that women more often have coronary events without typical symptoms, which may explain the frequent failure of clinicians to initially diagnose ACS in women. A summary of patient complaints is as follows: Palpitations; pain, which is usually described as pressure, squeezing, or a burning sensation across the precordium and may radiate to the neck, shoulder, jaw, back, upper abdomen, or either arm; exertional dyspnea that resolves with pain or rest; diaphoresis from sympathetic discharge; nausea from vagal stimulation; decreased exercise tolerance. Stable angina involves episodic pain lasting 5-15 minutes, is provoked by exertion, and is relieved by rest or nitroglycerin. In unstable angina, patients have increased risk for adverse cardiac events, such as MI or death. New-onset exertional angina can occur at rest and is of increasing frequency or duration or is refractory to nitroglycerin. Variant angina (Prinzmetal angina) occurs primarily at rest, is triggered by smoking, and is thought to be due to coronary vasospasm.

PHYSICAL EXAMINATION

Physical examination results are frequently normal. If chest pain is ongoing, the patient will usually lie quietly in bed and may appear anxious, diaphoretic, and pale. Physical findings can vary from normal to any of the following: Hypotension - Indicates ventricular dysfunction due to myocardial ischemia, infarction, or acute valvular dysfunction. Hypertension - May precipitate angina or reflect elevated catecholamine levels due to anxiety or to exogenous sympathomimetic stimulation.; Diaphoresis. Pulmonary edema and other signs of LVHF, Extracardiac vascular disease.; Jugular venous distention.; Cool, clammy skin and diaphoresis in patients with cardiogenic shock. In addition, a third heart sound (S3) may be present, and frequently, a fourth heart sound (S4) exists. The latter is especially prevalent in patients with inferior-wall ischemia and may be heard in patients with ischemia or systolic murmur secondary to mitral regurgitation. A systolic murmur related to dynamic obstruction of the LVOT may also occur. It is caused by hyperdynamic motion of the basal left ventricular myocardium and may be heard in patients with an apical infarct. A new murmur may reflect papillary muscle dysfunction. Rales on pulmonary examination may suggest LV dysfunction or mitral regurgitation. Patients who present to the ED with chest pain who have a low short-term risk of a major adverse cardiac event must be identified to facilitate early discharge in order to avoid lengthy and costly hospital stays.(1) The ASPECT study tested a 2-hour, accelerated diagnostic protocol (ADP) that included the use of a structured pretest probability scoring method, ECG, and a point-of-care biomarker panel that included troponin, creatine kinase MB, and myoglobin levels. The study suggests that ADP can identify patients at low risk for a short-term major adverse cardiac event who may be suitable for early discharge; such an approach could be used to decrease the overall observation periods and admissions for chest pain and has the potential to affect health-service delivery worldwide.

1. Than M, Cullen L, Reid CM, et al. A 2-h diagnostic protocol to assess patients with chest pain symptoms in the Asia-Pacific region (ASPECT): a prospective observational validation study. *Lancet*. Mar 26 2011;377:1077-1084.

COMPLICATIONS

Complications of ischemia include pulmonary edema, while those of myocardial infarction include rupture of the papillary muscle, left ventricular free wall, and ventricular septum.

DIAGNOSTIC CONSIDERATIONS

As many as half of all cases of ACS are clinically silent in that they do not cause the classic symptoms of this syndrome. Consequently, ACS goes unrecognized by the patient. Maintain a high index of suspicion for ACS, especially when evaluating women, patients with diabetes, older patients, patients with dementia, and those with a history of HF.

Although ST-segment and T-wave changes are associated with CAD, alternative causes of these findings are left ventricular aneurysm, pericarditis, Prinzmetal angina, early repolarization, Wolff-Parkinson-White syndrome, and drug therapy (eg, with tricyclic antidepressants, phenothiazines).

Increasing public awareness of the typical and atypical presentations of ACS is of the utmost importance for optimal and timely treatment. Many patients do not recognize that their symptoms are cardiac in origin and therefore may delay seeking medical help.

Guidelines from the European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA) recommend that patients with established CAD call emergency medical services if they have chest pain that does not resolve after they take a sublingual nitroglycerin tablet.

DIFFERENTIAL DIAGNOSIS

1. Anxiety
2. Aortic stenosis
3. Asthma
4. Cardiomyopathy, dilated
5. Esophagitis
6. Gastroenteritis
7. Hypertensive emergencies in emergency medicine
8. Myocarditis
9. Pericarditis and cardiac tamponade
10. Myocardial contusion
11. Aortic dissection
12. Pneumonia
13. Pneumothorax
14. Pleurisy
16. Pulmonary contusion
17. Pulmonary embolism
17. Pulmonary hypertension
18. Boerhaave's syndrome
19. Peptic ulcer disease
20. Esophageal spasm
21. Cholecystitis or biliary colic
22. Gastroesophageal reflux disease
23. Gastritis or esophagitis
24. Mallory-Weiss syndrome
25. Pancreatitis
26. Musculoskeletal pain
27. Herpes zoster

1. **Six AJ, Backus BE, Doevendans PA. Rapid diagnostic protocol for patients with chest pain.Lancet. 2011 Jul 30;378(9789):398-389.**