Sessão Clínica da Cardiologia
Unidade Emergência Coronariana
Clinic Session of Cardiology
Emergency Coronary Unit

Old man with Congestive Heart Failure and repetitive cardiac arrest episodes

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&
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17/08/11
August, 17 / 2011

CASO CLÍNICO/ CLINICAL CASE

•E.A.L., masculino, 64 anos, branco, aposentado

•QP: cansaço

•HDA: paciente com diagnóstico prévio de hipertensão arterial e insuficiência cardíaca congestiva.

•Ex-tabagista,

•Passou a referir dispnéia de início súbito 1 hora antes da admissão, sem relato de dor torácica ou outra queixa. No caminho, 10 minutos antes da chegada ao hospital, apresentou súbita perda da consciência.

•Paciente admitido na sala de emergência em parada cardiorespiratória (ritmo?) e reanimado após 10 minutos de manobras de ressuscitação.

•Fazia uso regular de captopril, anlodipina, carvedilol, espironolactona sinvastatina, furosemida, monocordil.

•Relato por familiar de antecedentes de duas paradas cardiorespiratórias prévias.

E.A.L., male, 64 years old, white, retired

MC: weariness

HDA: patient with a previous hypertension and congestive heart failure diagnosis.

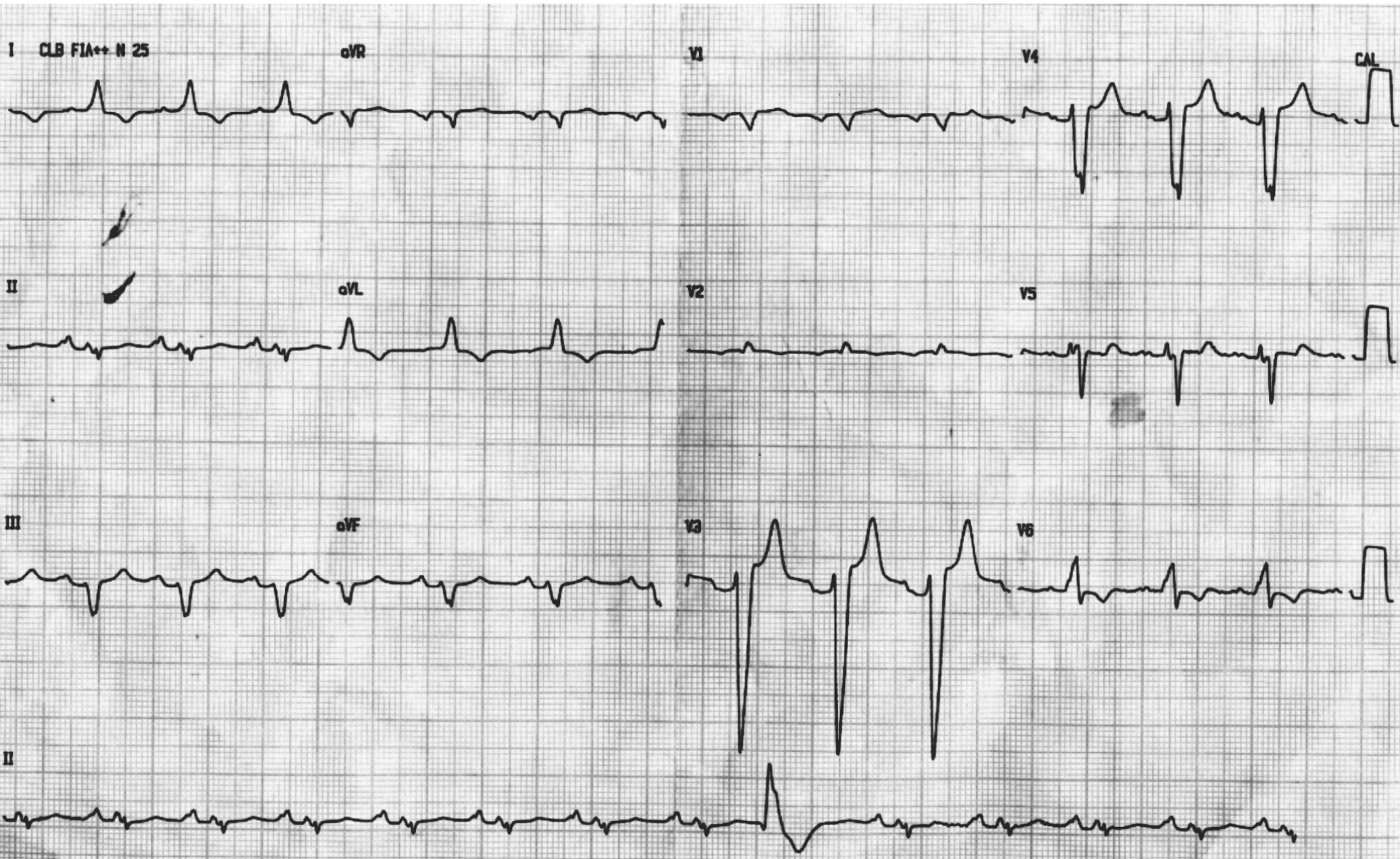
Ex-smoker

Sudden onset of dyspnea 1 hour prior to admission withouth chest pain or other complaint. On the way, 10 minutes before arrival at the hospital, he had sudden loss of consciousness. Patient admitted to the emergency room in cardiopulmonary arrest (rhythm?) And resuscitated after 10 minutes of resuscitation maneuvers.

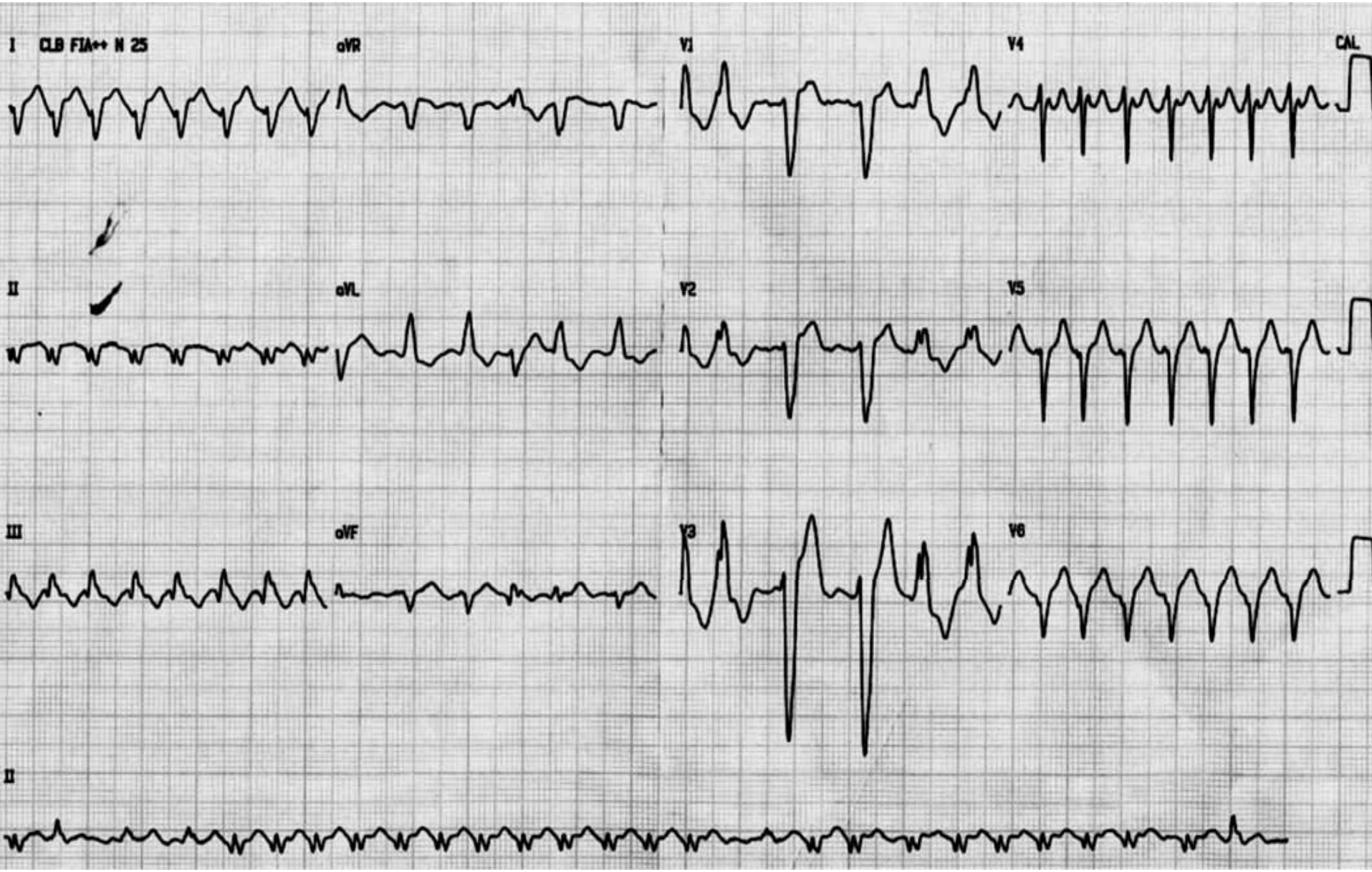
He is using regularly captopril, amlodipine, carvedilol, spironolactone, simvastatin, furosemide, and monocordil.

Report for family history of cardiorespiratory previous sudden cardiac arrest.

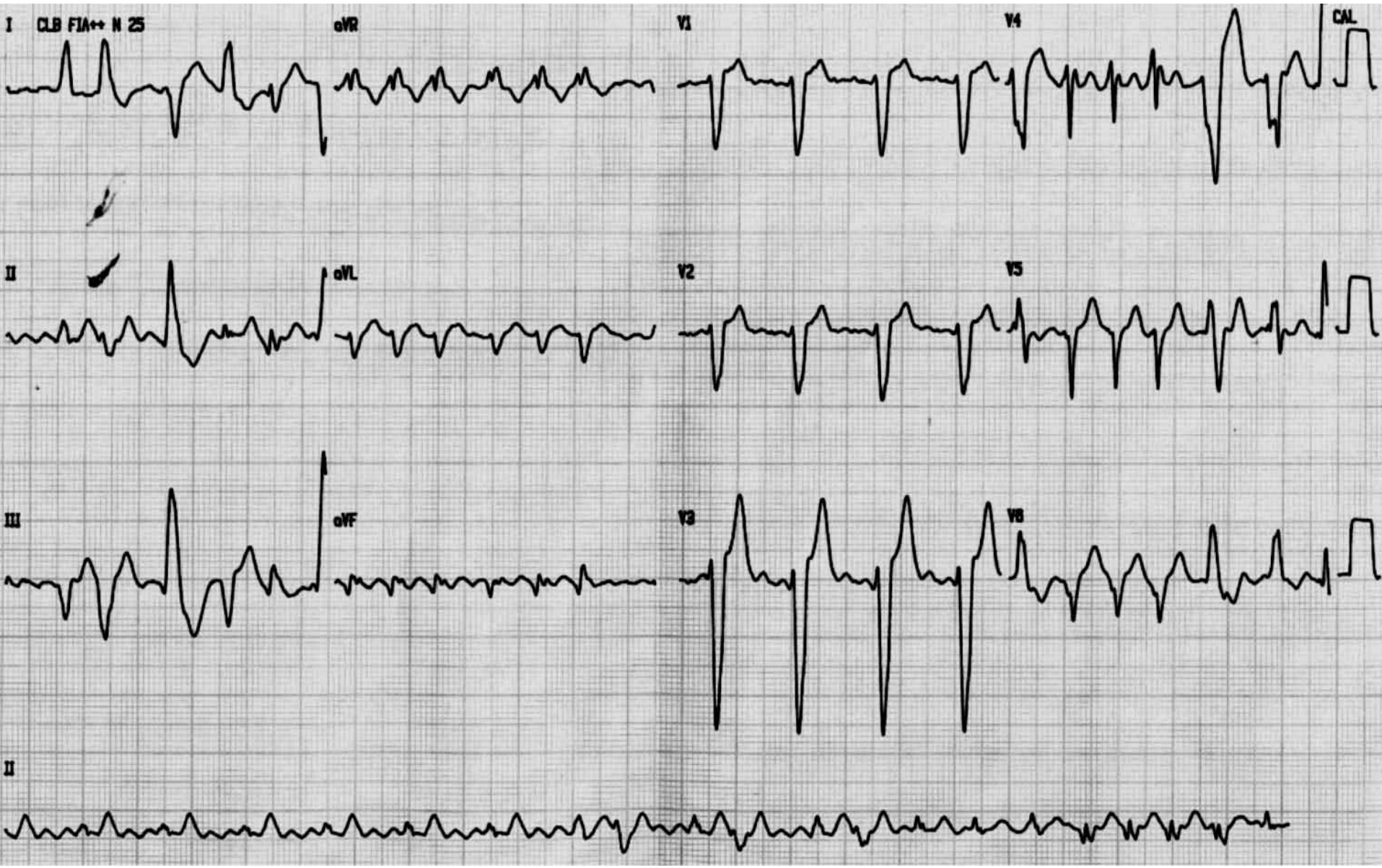
ECG1 August 05/2011
05/08/11 – 18:30



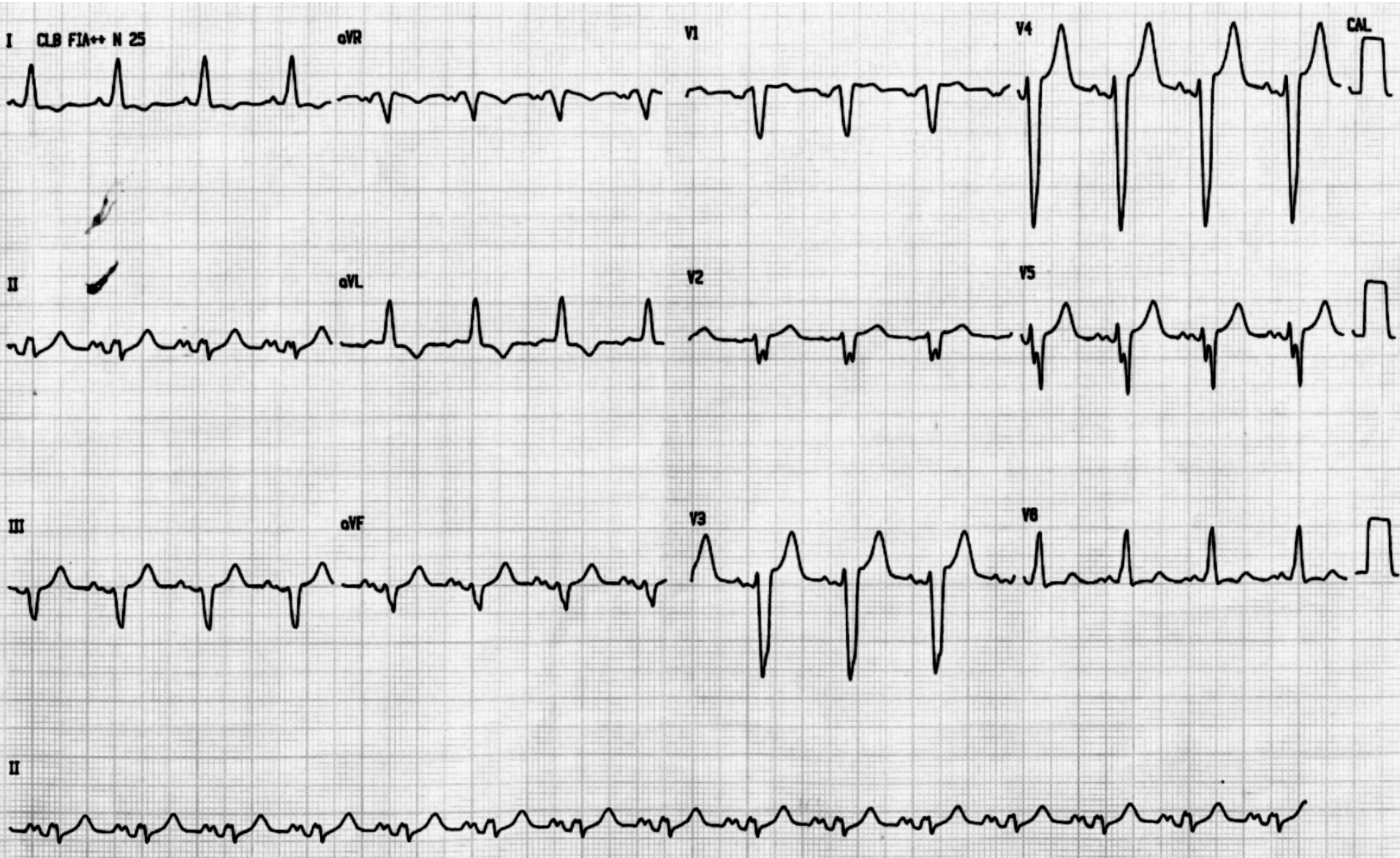
ECG2 August 07/2011 – 19:30



ECG3 August 07/11 21:20h After Amiodarone



ECG5 07/08/11 21:40 after electrical cardioversion 100J



Colleagues opinions

No sabemos cual era el ritmo durante la parada cardiaca, es decir si fue una TV, FV o asistolia. Fue realizado cardioversión???.....,en la secuencia de ECG tiene ritmo sinusal con bloqueo de rama izquierda el dia 5, no se si ese dia fue su ingreso y luego de la parada, mas tarde el dia 7 presenta una TV bien tolerada hemodinamicamente porque le administraron amiodarona, y depues vemos otro ECG con flutter.
Me pareciera que el paciente entra y sale de la TV con flutter repetidamente.
Finalmente le realizan una cardioversion eléctrica electiva por lo que entiendo del caso y sale en sinusal con su bloqueo de ramo izquierdo.
Hay datos de la historia que faltan y son importantes. Me refiero concretamente el estado neurológico. Este dato es muy importante a la hora de decidir la conducta definitiva de este paciente.
No esta demas recordar que toda taquicardia con QRS ancho en paciente con cardiopatia estructural y dilatado es TV hasta que se demuestre lo contrario

Carlos Rodriguez
crartuza@HOTMAIL.COM

We do not know which was the rhythm during cardiac arrest, ie whether it was a VT, FV or asystole? Was performed synchronized electrical Cardioversion?. In the sequence the ECG shows sinus rhythm with LBBB on day 5 do not know if that day was your income and then stop, later day 7 for a TV hemodynamically well tolerated because he was given amiodarone, and after see another ECG flutter. I think the patient enters and leaves the flutter VT repeatedly. Finally was preformed another elective electrical cardioversion from what I understand the case and sinus rhythym with LBBB is observed again.
Important history data are lacking. I refer in particular neurological status. This fact is very important in deciding the ultimate behavior of this patient.
Finally, it is important to remember that all wide QRS tachycardia in patients with structural heart disease is an VT until proven otherwise

Dear friends of the forum: I will try to analyze the ECG by Prof. Andres Ricardo Pérez Riera MD PhD, and Dr. Raimundo Barros Barbosa. This ECG belongs to someone with COPD. Why? Due to the frontal axis shifted to the left and axis of the P wave shifted to the right. The ECG indicates chronic anteroseptal and apical infarction, that has not been reperfused, suggesting septal and apical aneurysm- Deep S wave in V3 indicates remodeling of the posteroseptal area. Also, the high anterior side presents signs of fibrosis, expressed in TIII>TI and inverted T wave in aVL.Positive small wave in V2 indicates V5 remodeling, where previously there was Q/S wave due to high septal infarction. Probably, the patient developed apical VT, originated in the aneurysmic area, which evolved into VT. A good experience I practice, is to investigate the patient hemodynamically, and perform a bypass, if there are other obstructed arteries with infarctectomy, and if the other arteries are free from obstructions, perform infarctectomy. Generally, the signs of heart failure are mitigated or disappeared, as well as ventricular arrhythmias, which threaten life.

Warm regards, Samuel Sclarowsky

Queridos amigos del forum Tatare de analizar el ECG del profe Andres Ricardo Perez Rierra PHD, y del Dr Raimundo Barros Barbosa: Este ECG pertenece a una persona con DPOC. Porque? Por el eje frontal desviado a la izquierda y eje de la onda P desviado a la derecha El ECG indica un infarto anteroseptal y apical crónico, no reperfundido , sugeriendo un aneurisma del septum y apical. Las onda S profunda en v3 indica remodelacion del area posteroseptal. Tambien la cara anterior alta esta con signos de firosis , expresandose en TIII>TI y onda T invertida en aVL. La onda positiva pequeña en V2 indica remdelación de v5 , donde previamente existia una onda Q/S debido a un infarto septal alto Probablementr el paciente desarrollo una TV apical, originado en el area aneurismatica, que evoluciono a FV Una buena experiencia mia es investirgarlo hemodinamicamante, y realizarle by pass, si existen otras arterias obstruidas con infarctetomia, y si las otras arterias estan libres de obstrucciones , realizar infarctetomia generalmente los signos de ICC se atenuan o desaparecen, como asi tambien las arritmias ventriculares ,que amenazan a la vida un fraternal abrazo Samuel

Dear Andrés: This 64 y.o. man with cardiomyopathy possible related to chronic uncontrolled hypertension presented with an exacerbation of heart failure and arrhythmia. He may also have experienced an acute coronary event which precipitated the acute heart failure and arrhythmia. His ECG in May 2011 shows left bundle branch block with a slight left axis deviation and probable left atrial enlargement. The first ECG on 7/20/11 shows mostly a left ventricular tachycardia with QRS axis in the north-west quadrant and a few probable sinus beats appearing in leads V1-3. After Amiodarone he has more supraventricular beats with LBBB morphology, but also runs of NS-VT. The mechanism for the supraventricular beats is uncertain, but I think he is in atrial flutter with 4:1block. If his cardiac enzymes are normal and acute coronary event ruled out, he should be treated with an ICD and biventricular pacing. If this is an acute coronary event further workup is indicated before considering the ICD/pacemaker Rx.
I look forward to the comments of your colleagues.
Regards,
Frank
Frank Yanowitz, MD, FACC
Professor of Medicine
University of Utah School of Medicine
Medical Director, IHC ECG Services

Estimado Andrés:

Este hombre de 64 años con miocardiopatía, posiblemente relacionada con hipertensión crónica no controlada, se presentó con una exacerbación de insuficiencia cardíaca y arritmia. También puede haber experimentado un evento agudo coronario que precipitase la insuficiencia cardíaca aguda y la arritmia.
Su ECG de mayo de 2011 muestra bloqueo de rama izquierda con una ligera desviación del eje hacia la izquierda y probable sobrecarga de la AI.

El primer ECG del 20 de julio de 2011 muestra principalmente, TV del VI con eje QRS en el cuadrante noroeste y unos pocos latidos sinusales que aparecen en las derivaciones V1-3.

Luego de amiodarona presenta más latidos supraventriculares con morfología de BRI, pero también colgajos de TV no sostenida. El mecanismo de los latidos supraventriculares es incierto, pero creo que está en aleteo auricular con bloqueo 4:1.

Si sus enzimas cardíacas son normales y se descarta evento coronario agudo, debe ser tratado con CDI y estimulación biventricular.

Si es un evento coronario agudo, se indica una mayor investigación antes de considerar el tratamiento con CDI/marcapasos.
Espero los comentarios de los colegas.

Cordialmente,

Frank

Colegas e caros amigos do Fórum: Sei que ê muita petulância por este laudo do primeiro dos ECGs deste caso aqui junto ao do Professor Sclarovsky, mas devo ter coragem de errar aprendendo e aprender errando:
Idoso c/ HAS, ICC, dispnéia súbita há 1 hora e síncope há 10 minutos ECG 1.
Ritmo sinusal irregular, FC = 88 bpm (extra-sístole ventricular isolada em D2 com padrão de BCRE portanto de origem no VD)
Duração: P = 0,12" PR = 0,20" QRS = 0,12"
EIXOS: SÂP = 60º SÂQRS = - 30º SÂT = 120º
Alterações morfológicas:

1. **Onda P larga com entalhes (D2 – V4 a V6) Negativa em V1 e V2**
2. **Infradesnivelamento do segmento PR (D2,D3, aVF, V3 e V4**
3. **Ausência de progressão de R de V1 a V6. *Eletrodo de V2 em possível erro de local.**
4. **Cornell (em presença de BCRE) = 36**

LAUDO:

1. **Bloqueio Completo do Ramo Esquerdo**
2. **Extra-sístole ventricular isolada**
3. **Possíveis zona inferior e anterior inativas**
4. **Infarto atrial**
5. **Sobrecarga Atrial e Ventricular Esquerda**

Colleagues and dear friends,
I know that it is too much vanity to make an analysis of the first ECG about this case right next to Professor Sclarovsky, but I have to be courageous to make mistakes to learn and to learn by making mistakes.
Elderly man with systemic hypertension, CHF, sudden dyspnea 1 hour before and syncope 10 minutes before the first ECG.

Irregular sinus rhythm (isolated premature ventricular contraction in D2 , pattern of CLBBB, so originating in the RV), HR = 88 bpm.

Duration:
P = 0.12” PR = 0.20” QRS = 0.12”

Axes:
SAP = 60º SAQRS = -30º SAT = 120º

Morphological alterations:
1.Wide P wave with notches (D2 – V4 through V6). Negative in V1 and V2
2.PR segment depression (D2, D3, AVF, V3 and V4)
3.Absence of progression of R from V1 through V6. *Electrode of V2 at a possible mistaken location.
4. Cornell (in the presence of CLBBB) = 36
Analysis: Complete left bundle branch block Isolated premature ventricular contraction Possible inactive inferior and anterior areas
Atrial infarction
Left atrial and ventricular enlargement.

Adail

Dear Professor Andres and the rest of the colleagues of the Forum, I go on with the study of ECGs (it seems that Professor Frank Yanowitz got confused with how we write dates, and mentioned an ECG from May that I didn't see:
ECG 2: 7th of August, 2011 – 19h30m: Irregular ventricular rhythm with sinus systoles in couples (AVR, AVL, V1 through V3). HR = 187 Duration: P = impossible to estimate PR = idem RP = idem (dissociation) QRS = 0.12. Axes: SAP = maintained at 60º SAQRS = 120º SAT = -30º Conclusion Ventricular tachycardia that originates in the LV with short episodes of sinus rhythm.
Post-amiodarone ECG3: Alternation of ventricular tachycardia with base rhythm Post-cardioversion ECG4: Return to base rhythm with change in P pattern (m configuration), with PR depression only in DII. 2. CLBBB. 3. Probable inactive anterior and inferior area.
Management: investigate/stratify ischemia and act according to the results. If there is no ischemia, without evaluation of arrhythmia (ICD?), check stage and optimization of therapy for hypertension and heart failure.

Caro Professor Andrés e demais colegas do Fórum; Continuando estudo dos ECGs(Parece que o professor Frank Yanowits confundiu-se com nosso modo de datação e referiu-se a um ECG de maio que não vi):
ECG 2: 07/08/2001- 19h30m Ritmo irregular ventricular c/ sístoles sinusais em duplas (aVR. aVL, V1 aV3) FC = 187. Duração: P = Impossível de calcular PR = idem RP = idem(Dissociação) QRS = 0,12 Eixos: SÂP = Mantido 60º SÂQRS = 120º SÂT = -30º Conclusão: TV de origem no VE c/ curtos episódios de ritmo sinusal
EC3 pós AMIODARONA: Alternância de TV com ritmo de base.
ECG 4 pós cardioversão: Retorno ao ritmo de base com mudança no padrão de P(configuração de m), com infra-desnível de PR apenas em DII. 2. BCRE 3. Provável Zona inativa Ânterior e Inferior. CONDUTA: Investigar/Estratificar isquemia e conduzir-se de acordo com resultados. Se não houver isquemia, aí sim avaliar arritmia(CDI?), ver estágio e otimização da terapeutica de Hipertensão e da Insuficiencia cardíaca.Adail - Bahia - Brasil

Hello and greetings! This is a patient with data of HF and by the tracing, data of CAD. He still has in the first tracing, sinus rhythm with HR of 61X, QRS of 0.12s and LBBB. He presents LV hypertrophy in V3, V4, V5, V6, with inverted T wave, emphasizing the fact of CAD.In DII, in the length of the first tracing, he shows one premature contraction and inverted T wave plus LBBB related to coronary event. From the second tracing, he presents VT with wide QRS in I, V5 and V6. It appears to be fascicular. About aVR, aVF, aVL, V1, V2, V3, V4, he presents ventricular polymorphism by block and affected His bundle fascicle and ventricular hypertrophy. Which artery is affected at this level by use of amiodarone? From V1, V2, V3, V4, V5, V6, ventricular hypertrophy persists with LBBB and fascicular ventricular tachycardia. As therapeutic option, induce other arrhythmias if he already had prior ischemia. In DI, DII, DIII with premature contractions heading to ventricular tachycardia.Right after cardioversion, as conclusion, he presents heart failure with CAD, LBBB, LVH and AV block by use of amiodarone.It is worth mentioning that the previous tracings indicate the degree of involvement of the endothelium with sclerosis. LA and LVH.The management from echo and see EF from the two ventricles and the valves besides the atria. How is the mitral valve in this patient?Continue with electrophysiology and arrhythmology. The sequence and treatment of this patient.Any commentary, my e-mail is:,GMASLIVAR@YAHOO.COM

Dear Malsivar,You are telling me tha the second ECG shows fascicular VT? Don't you think that this type of VT occurs without structural heart disease? Predominantly in 15 to 40-year-old young people.I agree that the event presents a quite narrow QRS duration (<120 ms) and RBBB in V1 with axis shift to the right. Fascicular VT has been classified by its ECG pattern into 3types:

RBBB+LAFB with axis shift to the left and relatively narrow QRS (left posterior fascicular VT). This could be explained by its origin in the left posterior fascicle.

RBBB+LPFB with axis shift to the right, and relatively narrow QRS (could RBBB+itbe this variant?). Left anterior fascicular VT.

RBBB+LSFB. Upper septal fascicular VT. What criteria have you used to express this diagnosis? Could you explain your rationale in a deeper way? Andres

Hola mis saludos.
Se trata de paciente con datos de insuficiencia cardiáca y por el trazo datos de cardiopatía coronaria tiene todavía en el primer trazo un ritmo sinusal con un FC de 61X un QRS de 0.12s y con BRI. Presenta HVI en V3,V4,V5,V6 con inversión de la onda T recalcando el hecho de la cardiopatía coronaria.
En DII largo del primer trazo presenta una extrasístole y la inversión de la onda T mas el BRI relacionado a un envento coronario.
A partir del segundo trazo presenta TV con QRS ancho en DI,V5 y V6 aparenta ser fascicular. con respecto aVR,aVF,aVL,V1,V2,V3,V4, presenta polimorfismo ventricular por el bloqueo y el fascículo del haz de hiz afectado y la hipertrofia ventricular cual de las arterias está afectada a este nivel.
Con el uso de la amidarona desde V1,V2,V3,V4,V5,V6 persiste la HVI con, el BRI y la TV fascicular. como opción terapeútica induce otras arritmias si ya tenía una isquemia previa. en ,DI, DII DIII con extrasístoles rumbo a una TV Justo después de la cardioversión como conclusión presenta una insuficiencia cardiáca con síndrome coronario, BRI, hipertrofia ventricular y bloqueo AV por el uso de la amiodarona.
Cabe resaltar que los trazos previos indican el grado de afección al endotelio con esclerosis hipertrofia de la aurícula izquierda y el ventrículo izquierdo.
El manejo desde un ecocardiograma y ver la fracción de eyección de los dos ventrículos y las válvulas además de las aurículas. cómo quedó la válvula mitral de este paciente.
Continuar con electrofisiología y arritmología la sequencia y tratamiento de este paciente.
Cualquier observación mi correo es

gmaslivar@yahoo.com

Prezado Malsivar: você está dizendo que o Segundo ECG mostra uma TV fascicular? Você não lhe parece que este tipo de TV ocorre sem cardiopatia estrutural? Predominantemente em jovens de 15 a 40 anos.
Concordo que o evento apresenta uma duração do QRS bastante estreita(<120ms) e BRD em V1 com desvio do eixo para direita A TV fascicular há sido classificada por seu padrão ECG em 3 tipos
BRD+ BDASE com desvio do eixo a esquerda e relativamente estreito
BRD+ QRS(left posterior fascicular VT) Esto se explicaria por originar se no fascículo posteroinferior esquerdo.
BDR+ BDPIE com desvio do eixo a direita e relativamente estreito QRS(BDR+ seria esta variante?)Left anterior fascicular VT
BRD+ BDAM Upper septal fascicular VT
Que critérios você há usado para externar este diagnóstico?
Poderia nos explicar teu raciocínio mais aprofundad mente?
Andres

In this patient, to be able to carry out any type of intervention, the way I see it, first it is necessary to know his neurological state. It is very strange for a patient with 2 prior arrests and a third arrest with 10 of reanimation, to have no sequels. It is even more rare if this is an ischemic patient. To be able to survive 3 cardiac arrests with no prior intervention, he must have base heart disease or the history of the patient is confusing.
About the sinus ECG with LBBB, the 5th, the 7th VT and flutter, post electrical cardioversion sinus rhythm, LBBB. Another thing that I wonder about, is that he is admitted in cardiac arrest and after monomorphic VT with hemodynamic stability, amiodarone is given initially. I would like to know more data about the clinical history, because everything is very strange.
Kako

En ese paciente para poder realizar cualquier tipo de intervención primero a mi juicio es necesario saber como esta su estado neurologico, es muy raro que un paciente con 2 paradas previas y una tercera parada con 10 minutos de reanimacion quede sin ningun tipo de secuela, mas raro aun si es un paciente isquemico, para poder sobrevivir a 3 paradas cardiacas sin ningun tipo de intervencion previa debe tener otra cardiopatia de base o la historia del paciente esta confusa.
En cuanto a los ECG sinusal con BRI el 5, el 7 TV y flutter, post CVE RS, BRI. Otra cosa que me llama la atencion es que ingresa en parada cardiaca y luego TV monomorfica con estabilidad hemodinamica, le colocan amiodarona inicialmente, me gustaria saber mas datos de la historia clinica porque esta todo muy raro.

Kako

Dear Kako,
I send you some other information that Raimundinho provided due to your comments.
Andres
Professor Andres,
Some clarifications after reviewing his background:
1-Regrettably, we have no information about reanimation (likely the 2 cardiopulmonary arrests occurred in the ambulance during the trajectory to the hospital of Messejana).
2-There is report of heart arrest in electric activity without pulse? When he arrived to our hospital.
3-He evolved from admittance with post-cardiopulmonary arrest cerebral hypoxia, with mechanic ventilatory assistance required. Subsequently, pneumonia complicated the scenario, as well as renal insufficiency, so he died as a consequence of sepsis after 10 days.

Supplementary tests:
Troponin=0.25 (normal until 0.10); Mg=2.3; K=5; CK-MB=28.63 (normal up to 4.94); urea=61; creatinine=1.8; leukocytosis (20,200 leukocytes).

Coronary angiography: ADA=50% in the middle third; thin RCA with lesion of 80% distal.

Echo: LV=42/21; LA=45; EF=81%; Mass=505; senile mitral valve with discrete reflow; slightly thicker aortic valve with medium gradient of 18 mmHg and discrete reflow; severe concentric LV hypertrophy with significant deficit of relaxation. Normal systolic function.
Note: Echo performed at the border of the bed under vasoactive therapy.
Normal CT of cranium.

Fraternal regards,

Prezado Kako lê envio algunos otros datos que Raimundinho me há fornecido devido a seus comentários.
Andres.

Professor Andrés,alguns esclarecimentos após revisão do prontuário:
1-infelizmente não temos informações sobre a reanimação(as 2 prováveis PCR aconteceram na ambulância durante o trajeto para o hospital de Messejana)

2-há relato de parada cardiaca em AESP? ao chegar no nosso hospital

3-evoluiu à partir da admissão com encefalopatia anóxica pós PCR sendo necessária assistência ventilatória mecânica. Posteriormente complicou com pneumonia e insuficiência renal vindo à falecer em decorrência de sepse após 10 dias

Exames complementares:
Troponina=0,25(Normal até 0,10);Mg=2,3;K=5,3;CK-MB=28,63(normal até 4,94);Uréia=61;Creatinina=1,8;Leucocitose(20.200 leucocitos) Coronariografia: DA=50% no terço médio;CD fina com lesão de 80% distal
ECO: VE=42/21 AE=45; FE=81% MASSA=505;V.mitral senil com discreto refluxo;V. aórtica levemente espessada com gradiente médio de 18mmHg e discreto refluxo;HVE concêntrica severa com deficit importante de relaxamento.Função sistólica normal.Obs:ECO realizado à beira do leito sob terapia vasoativa.
TC de cranio normal.

Abraços

Raimundo

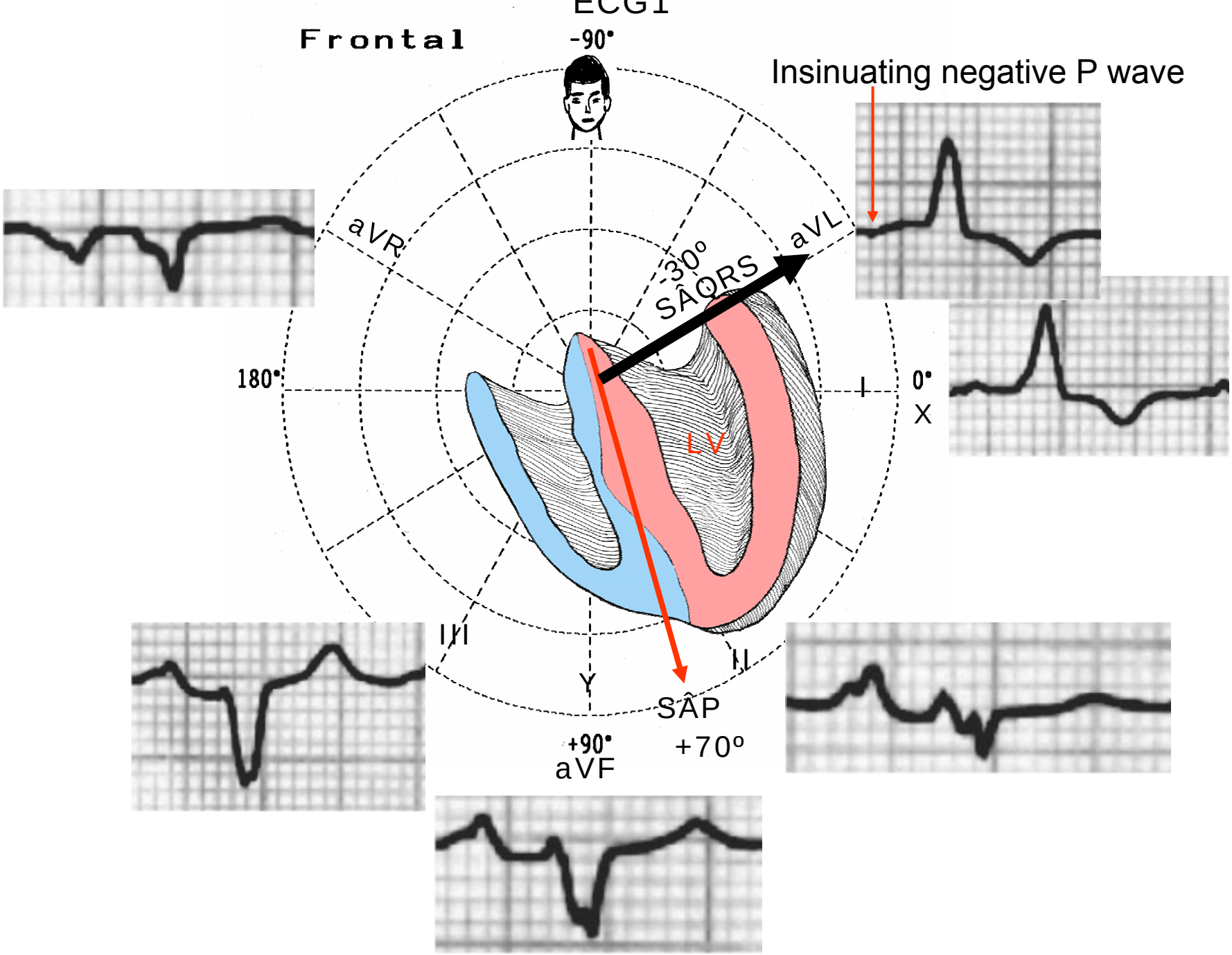
Thank you, Professor, with these data my suspicion about a neurological sequel is corroborated, since I considered it very strange that with 3 cardiorespiratory arrests he would not present anything neurologically. This datum is very important to decide on any type of intervention; regrettably, in this case the patient died.

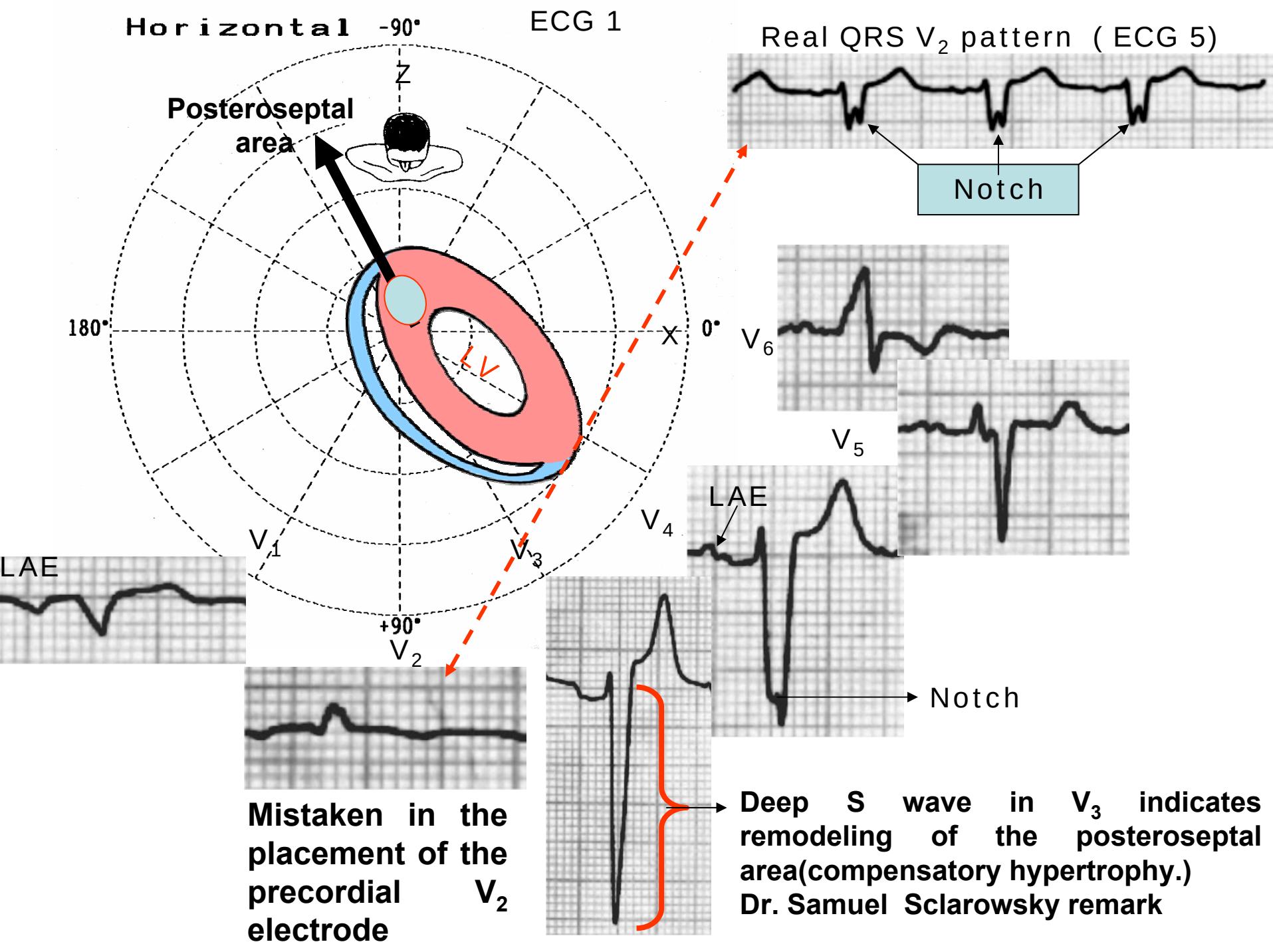
Kako

Gracias Profesor, con estos datos se corrobora mi sospecha que deberia tener alguna secuela neurologica ya que me parecia muy raro que con 3 PCR no presentara nada neurologicamente, ese dato es muy importante para decidir cualquier tipo de intervencion, infelizmente en este caso el paciente murió.

Ours final commentaries and diagnosis hypothesis

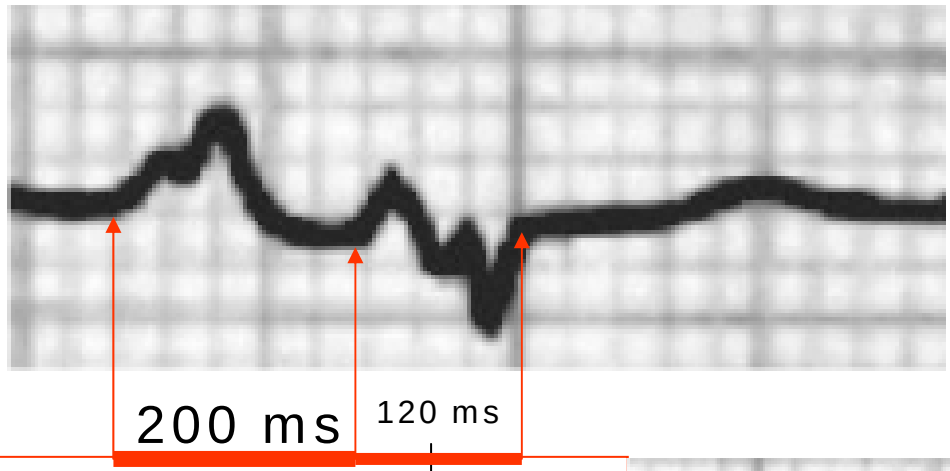
ECG1


Frontal
-90°
Insinuating negative P wave
aVR
-30°
SÂQRS
aVL
180°
0°
I
X
LV
III
II
Y
SÂP
+90°
aVF
+70°


Horizontal
-90°
ECG 1
Z
Posteroseptal area
180°
X
0°
LV
+90°
V1
V2
V3
V4
V5
V6
Real QRS V2 pattern (ECG 5)
Notch
LAE
LAE
Notch
Mistaken in the placement of the precordial V2 electrode
Deep S wave in V3 indicates remodeling of the posteroseptal area(compensatory hypertrophy.)
Dr. Samuel Sclarowsky remark

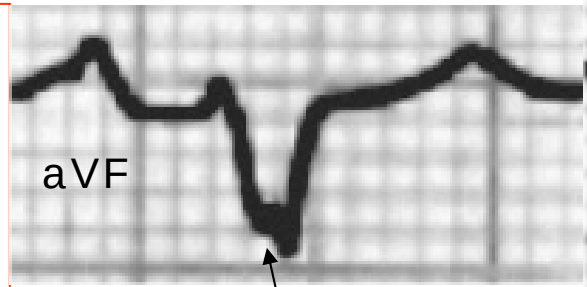
ECG 1

Sinus rhythm
Heart rate: 97bpm
P wave: P duration: 120ms.; P voltage:1.8mm.; P axis: +70º on FP (insinuating negative P wave in aVL) and to back in HP.; P shape: bimodal with the second mode higher related the first one.

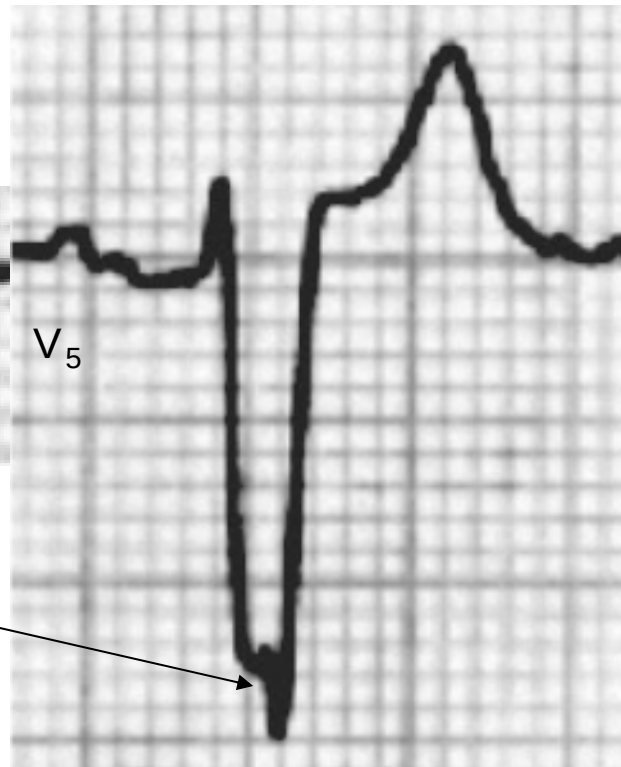


P axis +70º + P duration prolonged + bimodal shape with the second mode higher: biatrial enlargement.

PR interval: 200 ms
QRS axis: -30º
QRS duration: 120ms prolonged
QRS morphology: suggestive of intraventricular Conduction Disturbance associated with myocardial Infarction "nonspecific intraventricular conduction delay" with QRS duration >110ms but does not satisfy the criteria of either LBBB



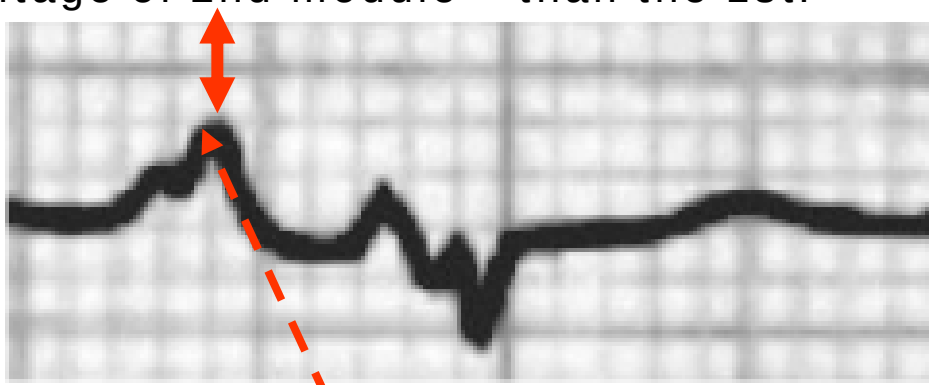
QRS notches

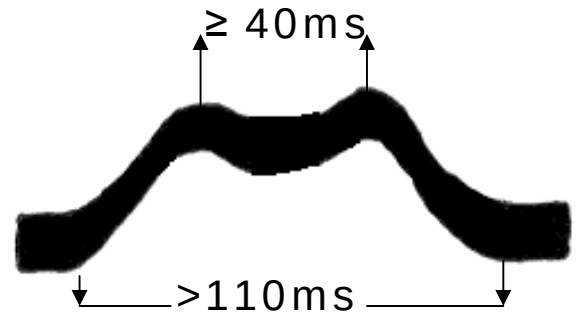


ECG 1 DIAGOSIS

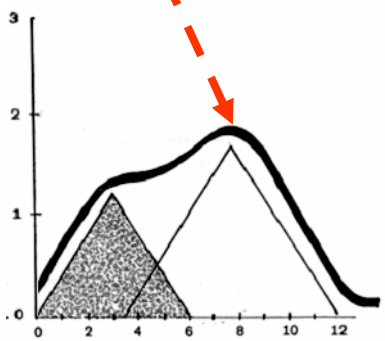
1. **Left Atrial Enlargement (LAE):** P duration prolonged (120ms), abnormal bifid or bimodal P wave. The normal shape of P wave is rounded and monophasic, and there may be small notches (more frequent in V3 and V4) and the distance between these notches should not exceed 30 ms (0.03 s). Notches in P wave with distance between the apexes of ≥ 40 ms (0.04 s) constitutes a sign of left atrial enlargement (LAE) or interatrial block by Bachman's bundle (BB), in charge of activating the left atrium (LA).

Voltage of 2nd module > than the 1st.



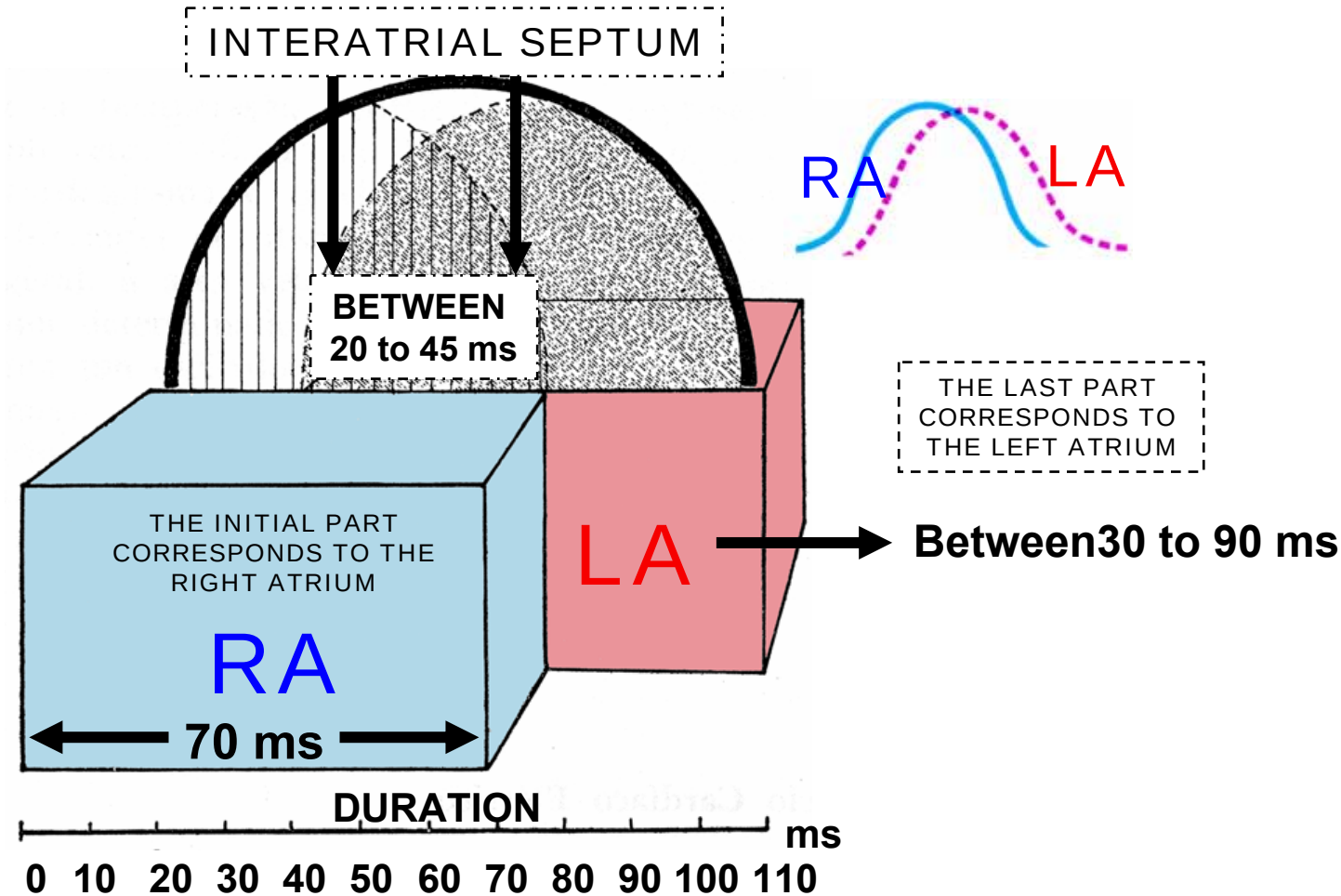


LAE



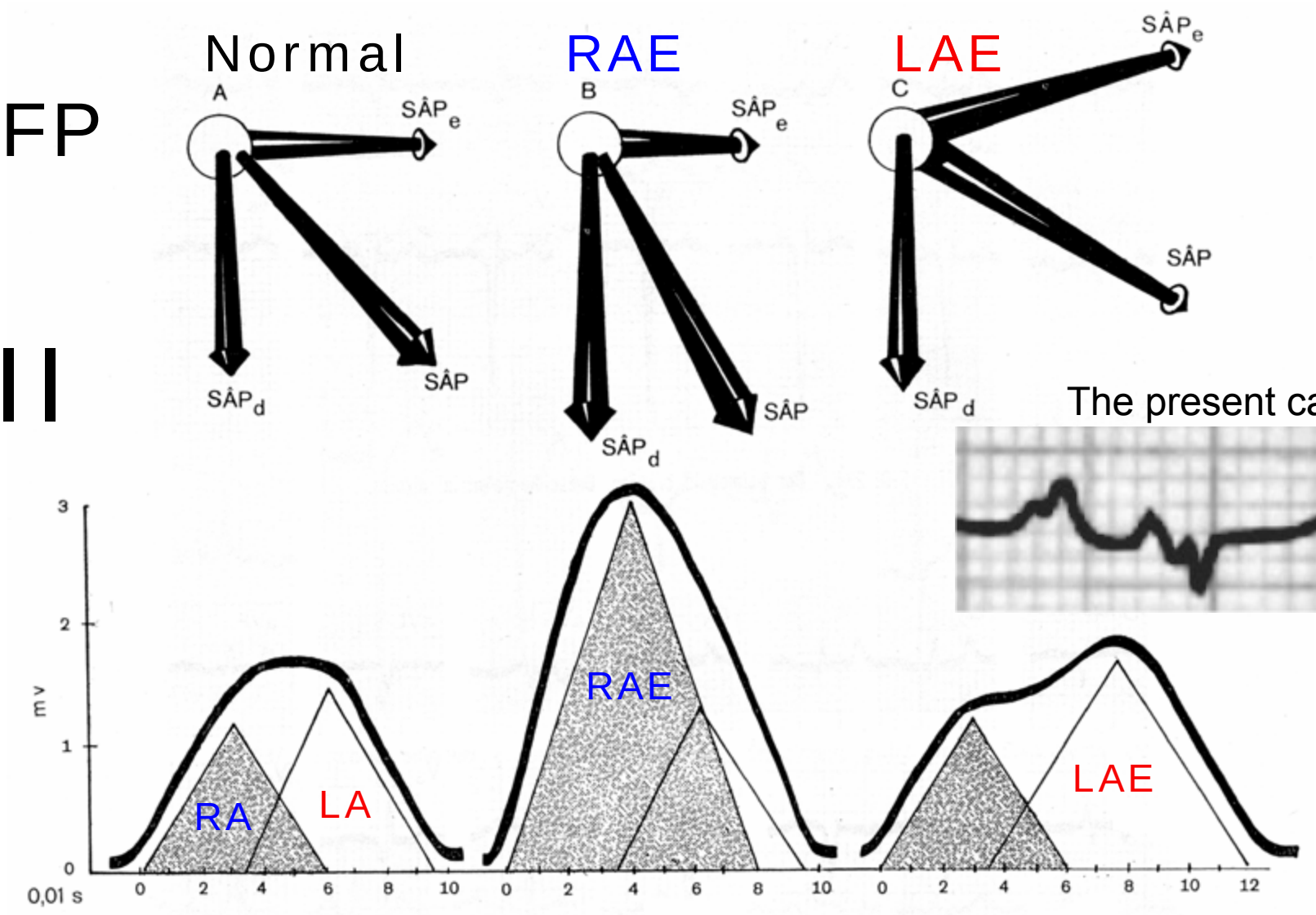
2. Right Atrial Enlargement (BAE): P axis +70º(insinuation negative P wave in aVL) associated with broad bimodal P wave
3. Biatrial Atrial Enlargement: P waves of duration ≥120 ms and bimodal shape with SÂP locate to the right of +65º. We think that it is not necessary that this ECG belongs to someone with COPD because biventricular HF could cause this P axis deviation.

NORMAL ACTIVATION OF BIATRIAL CHAMBER



NORMAL DURATION OF THE P WAVE = 80 to 110 ms

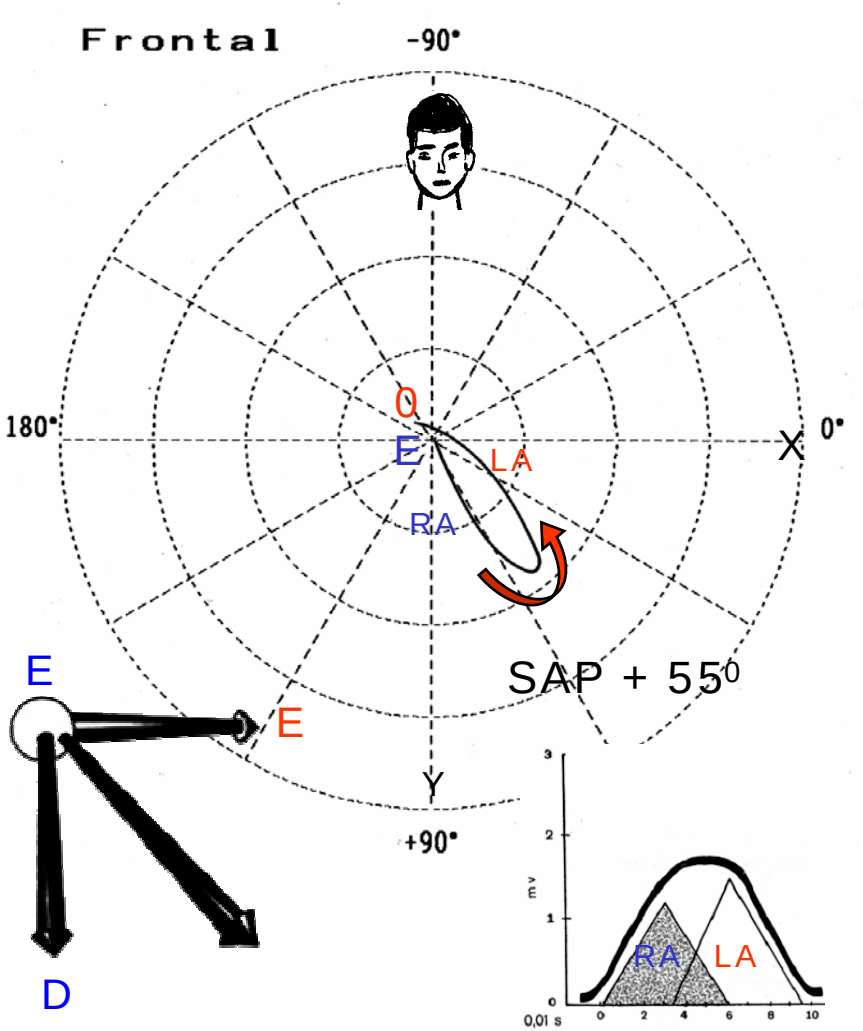
PROFILE OF NORMAL P WAVE IN RAE AND LAE



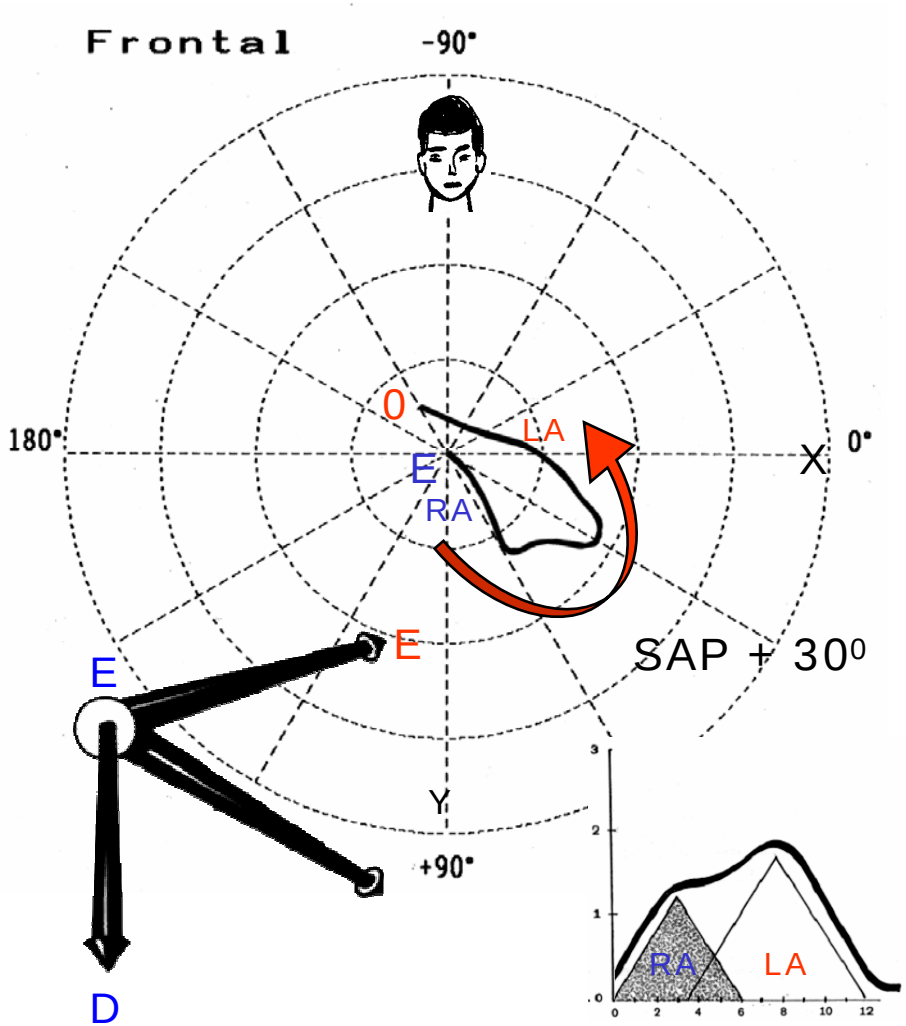
LEFT ATRIAL ENLARGEMENT IN THE FRONTAL PLANE

NOTE: THE FINDINGS IN THE FRONTAL PLANE ARE NOT RELEVANT FOR THE DIAGNOSIS OF LAE.

NORMAL P LOOP



P LOOP IN LEFT ATRIAL ENLARGEMENT



Similar advanced interatrial block

On the other hand P axis is located in +65º = biatrial enlargement

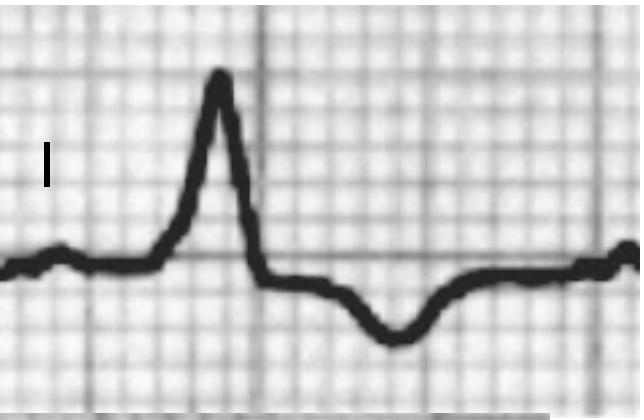
4 Left Ventricular Enlargement Hypertrophy(LVE or LVH) based in

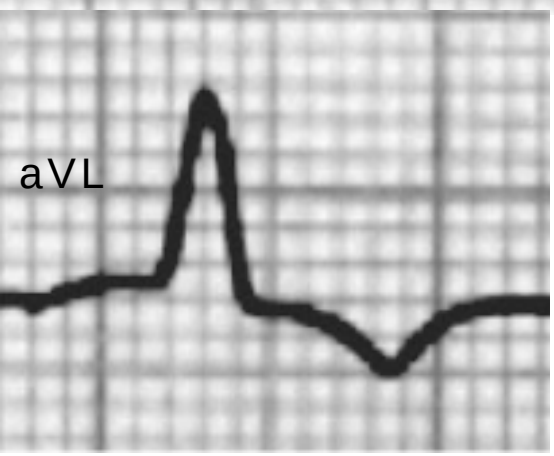
- QRS/T angle broadening
- Tendency to SÂQRS deviation to the left, backward and upward;
- Indirect criteria. notches or complexes in “M” in I and aVL or V3 and V4; Absence of growth of r in precordial leads from V1 to V3, with sudden increase in V6

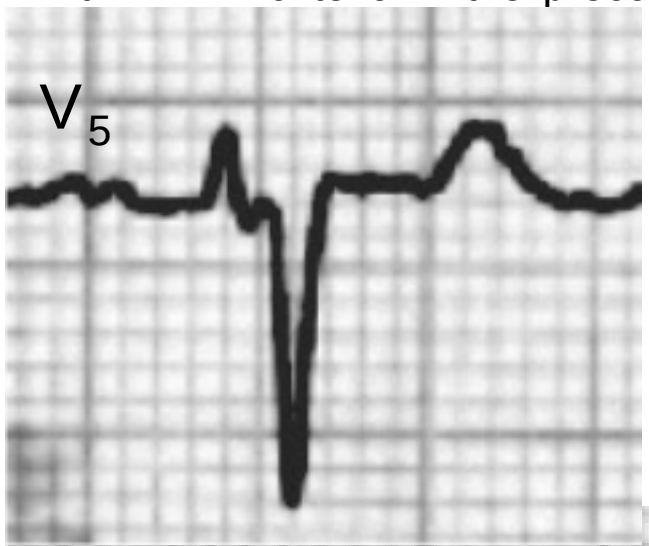
5. Nonspecific or Unspecified intraventricular conduction disturbance?: or “nonspecific intraventricular conduction delay associated with myocardial Infarction ” with QRS duration >110ms but does not satisfy the criteria of either LBBB.(1). ”The term *intraventricular conduction disturbances* refers to abnormalities in the intraventricular propagation of supraventricular impulses that give rise to changes in the shape and/or duration of the QRS complex. These changes in intraventricular conduction may be fixed and present at all heart rates, or they may be intermittent and be tachycardia or bradycardia dependent. They may be caused by structural abnormalities in the His-Purkinje conduction system or ventricular myocardium that result from **necrosis, fibrosis, calcification, infiltrative lesions, or impaired vascular supply**. Alternatively, they may be functional and due to the arrival of a supraventricular impulse during the relative refractory period in a portion of the conducting system, in which case the term *aberrant ventricular conduction* is applied. They may also be due to abnormal AV connections, which bypass the AV node, resulting in ventricular preexcitation”. (2). There are other possibility at the end of this presentation.

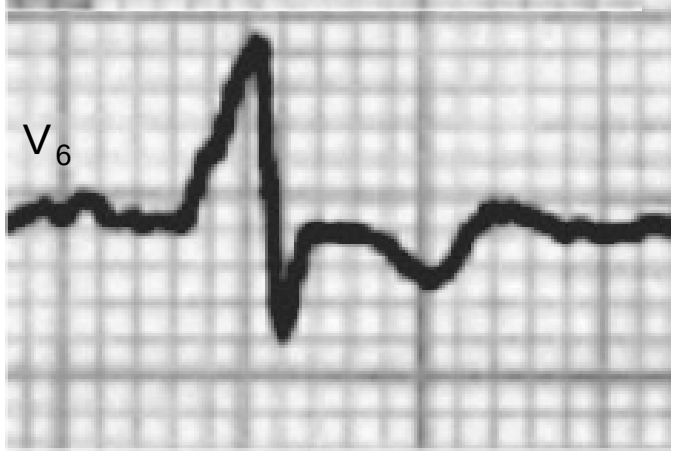
1. **Surawicz B, Knilans TK, In Chou's ELECTROCARDIOGRAPHY IN CLINICAL PRACTICE. Sixth Edition SAUNDERS ELSERVIER, 2008; Chapter 6: pp:120-122.**
2. **Surawicz B, Childers R, Deal BJ, et al; AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology.American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. J Am Coll Cardiol. 2009 Mar 17;53:976-981.**

QRS duration greater than 110 ms in adults, greater than 90 ms in children 8 to 16 years of age, and greater than 80 ms in children less than 8 years of age without criteria for RBBB or LBBB. The definition may also be applied to a pattern with RBBB criteria in the precordial leads and LBBB criteria in the limb leads, and vice versa(1).



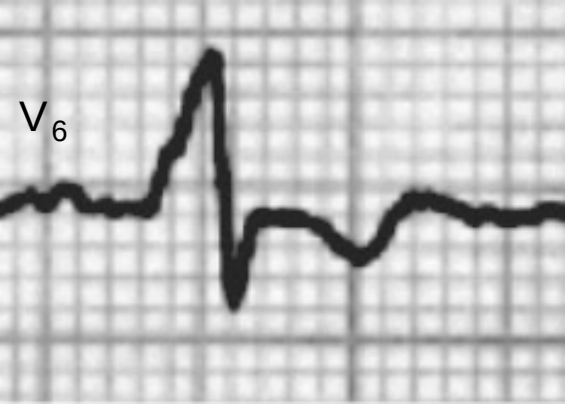




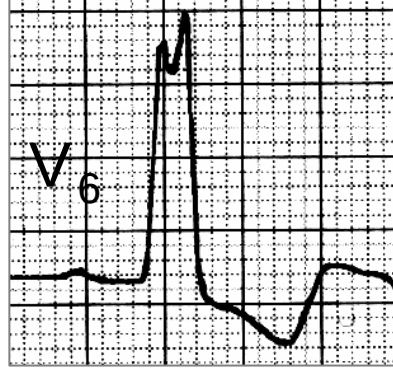


Left leads I, aVL, V_5-V_6 in the present case, however the QRS shape is atypical related a truly LBBB. Rigorous criteria analysis confirm LBBB ECG diagnosis. See next slide.

1. **Surawicz B, Childers R, Deal BJ, et al; AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology.American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. J Am Coll Cardiol. 2009 Mar 17;53:976-981.**



The present case atypical shape



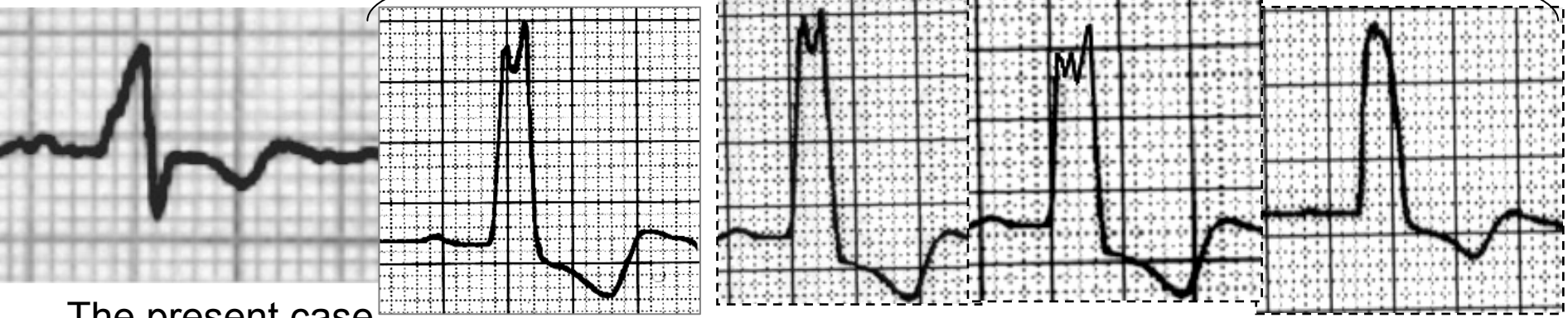
Classical CLBBB Shape Patter in V6

Tully Complete LBBB Criteria

1. ***QRS duration ≥ 120 ms in adults, > 100 ms in children 4 to 16 years of age, and > 90 ms in children less than 4 years of age.***
2. ***Broad notched or slurred R wave in leads I, aVL, V5, and V6 and an occasional RS pattern in V5 and V6 attributed to displaced transition of QRS complex.***
3. ***Absent q waves in leads I, V5, and V6, but in the lead aVL, a narrow q wave may be present in the absence of myocardial pathology.***
4. ***R peak time greater than 60 ms in leads V5 and V6 but normal in leads V1, V2, and V3, when small initial r waves can be discerned in the above leads.***
5. ***ST and T waves usually opposite in direction to QRS.***
6. ***Positive T wave in leads with upright QRS may be normal (positive concordance).***
7. ***Depressed ST segment and/or negative T wave in leads with negative QRS (negative concordance) are abnormal***
8. ***The appearance of LBBB may change the mean QRS axis in the frontal plane to the right, to the left, or to a superior, in some cases in a rate-dependent manner.***

- **Monophasic, broad notched or slurred R wave, recorded slowly in the left leads: I, aVL, V5 and V6.**

Classical shape LBBB pattern in left leads



The present case atypical shape

"Tower" with one notch

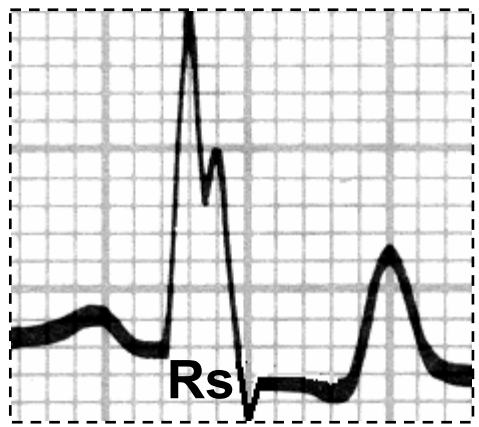
"Tower" with double notch

Plateau

a) There may be initial narrow q in aVL and exceptionally in I, however, never in V_5 and V_6.

b) Occasiona Rs or RS pattern in V_5 and V_6. In this case , it may indicate:

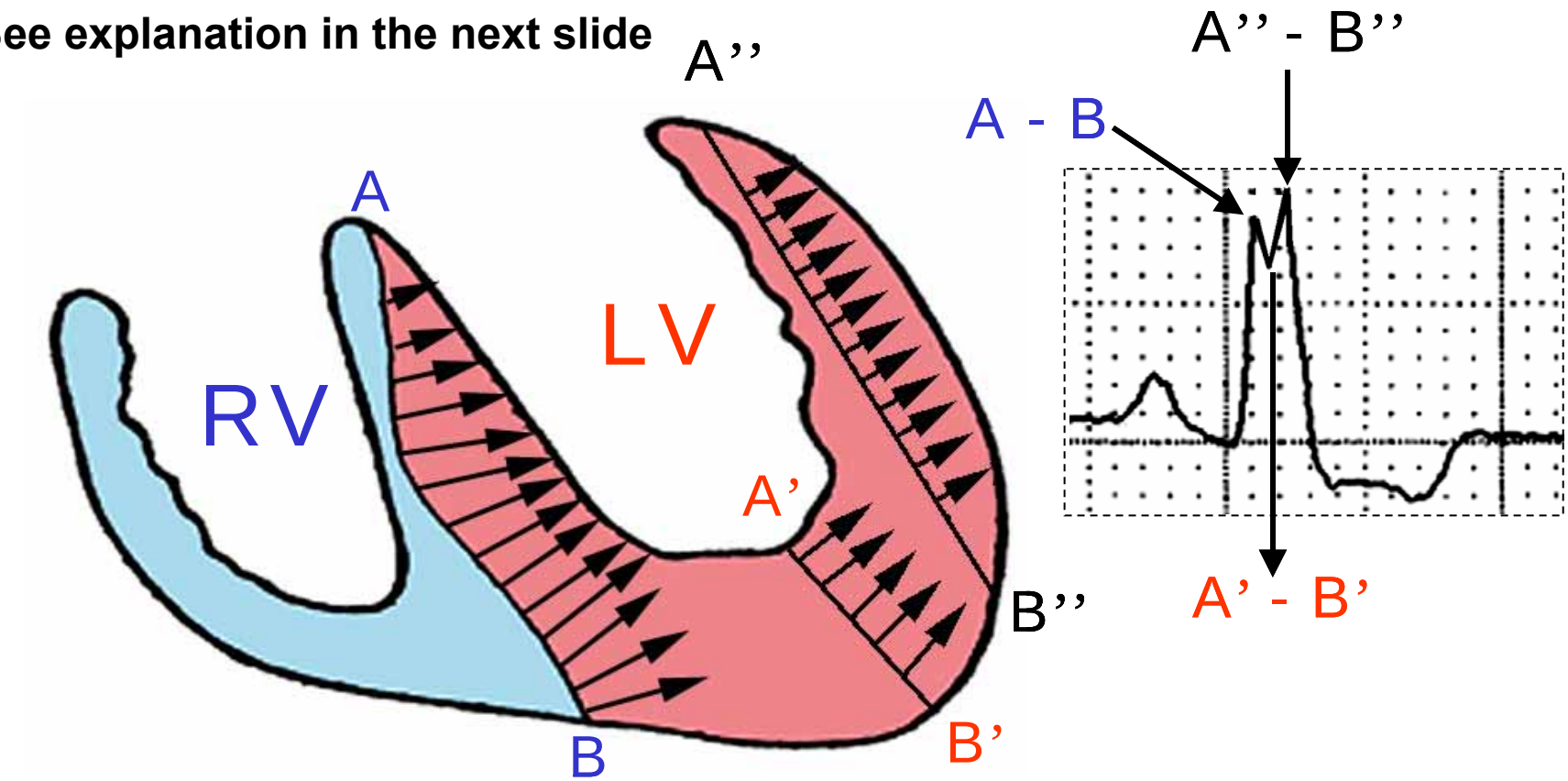
1) Displaced transition of QRS complex to left;
2) Associated right ventricular hypertrophy or RVE;
3) Associated left anterior fascicular block(LAFB);
4) Electrically inactive area of free wall associated to complete LBBB;



Electrocardiographic criteria for Complete LBBB

MONOPHASIC R WAVE OF SLOW RECORDING IN LEFT LEADS DI, aVL, V_5 AND V_6 AND ELECTROPHYSIOLOGICAL EXPLANATION

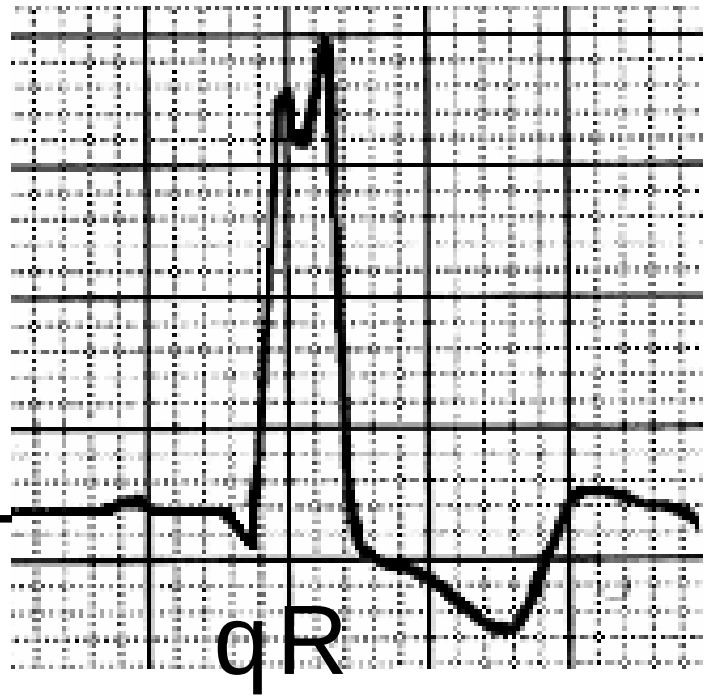
See explanation in the next slide



Outline that explains the frequent notch recorded in the apex of R wave in the left leads. Clearly, the wave front loses intensity when it reaches the ventricular cavity, which explains the notch on R wave in LBBB. 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 wave front width (A'-B') responsible for the notch in the apex of R wave. Next, the wave front reaches the LV free wall increasing again the width of the wave front (A''-B''), responsible for the second apex of R wave.

In the important hypertrophies of the free wall, this second apex presents a higher voltage.

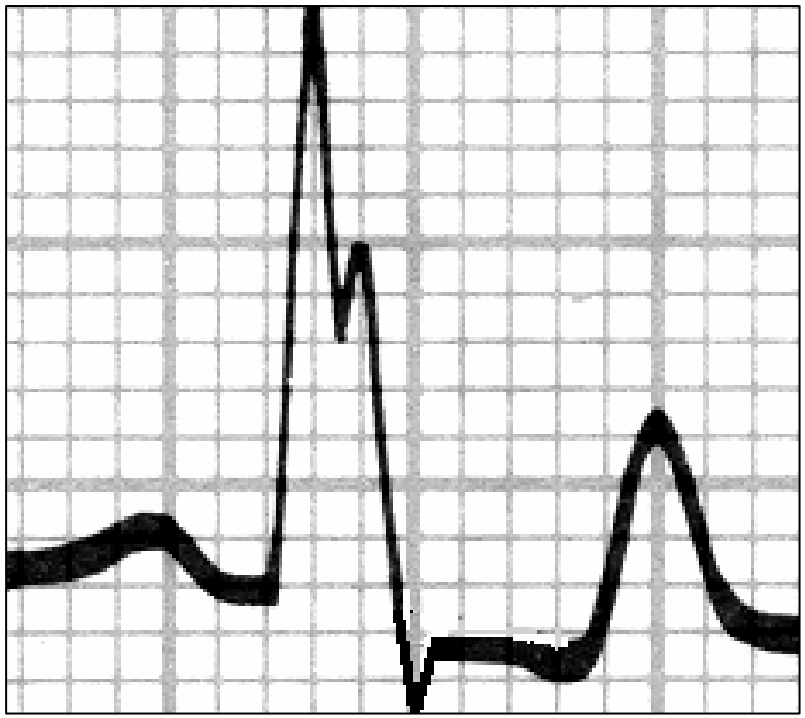
INITIAL q WAVE LEFT LEADS



In complete LBBB is characteristic the absence of initial q waves in left leads I, V5 and V6, but in the lead aVL a narrow q wave may be present in the absence of myocardial pathology/fibrosis/infarction.

The pure monophasic R wave is characteristic in the left leads (I, aVL, V5 and V6). Since the aVL lead is higher, it can rarely show qR pattern in absence of complications.

OCCASIONAL Rs OR RS PATTERN IN V_5 & V_6

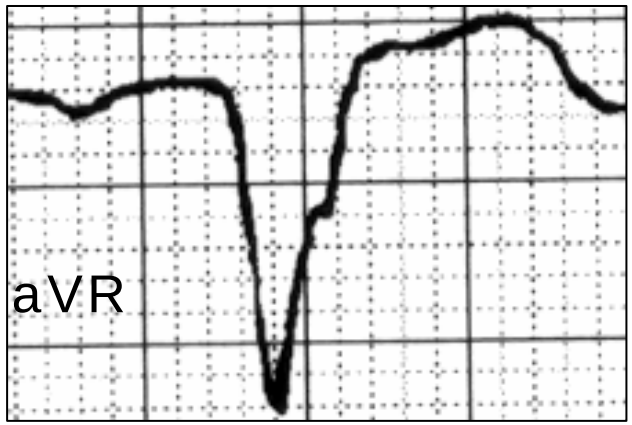


The presence of occasional Rs or RS pattern in the left precordial leads (V5-V6) may indicate:

1) Displaced transitional of QRS complex (transitional recording for LVE or LVH,
2) Association with LAFB,
3) Association with right ventricular enlargement (LVE) or hypertrophy (LVH)
4) Association with electrically inactive area in the LV free wall.

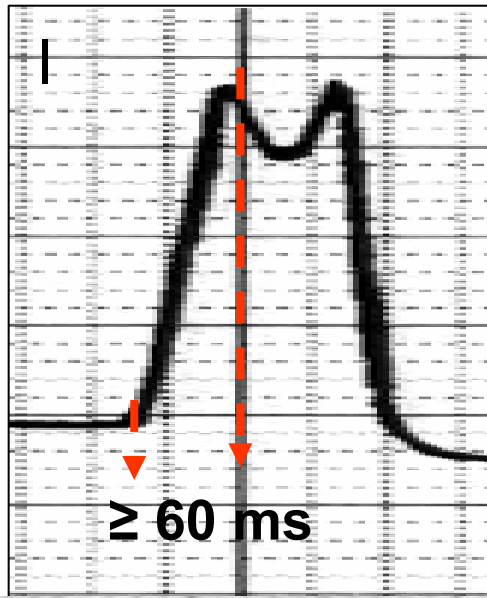
The left leads may show Rs pattern in absence of another associated problem. The presence of Rs pattern in the left precordial leads may indicate: transitional recording due to LVE, association with LAFB, RVE, association with electrically inactive area in LV free wall.

QRS complex of the QS type almost constant in aVR.

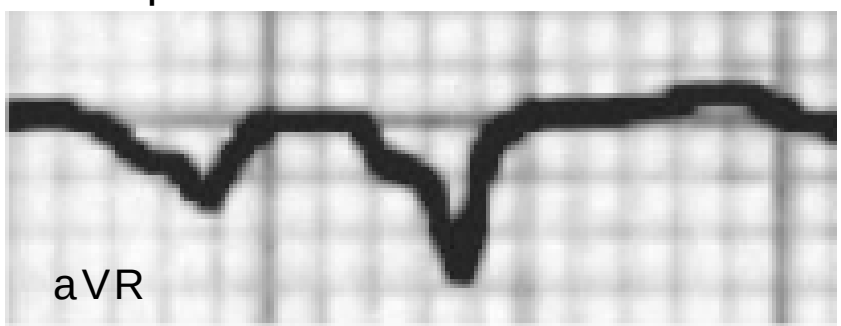


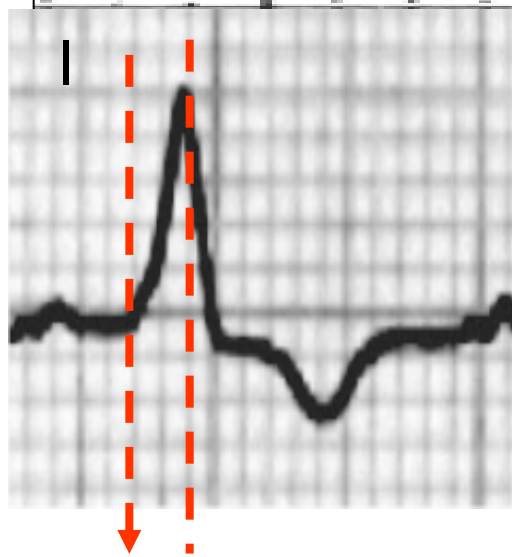
QS

R-peak time or Intrinsicoid deflection in I and V5-V6 ≥ 60 ms but normal in V1-V2 and V3, when small initial r waves can be discerned in the above leads.



The present case

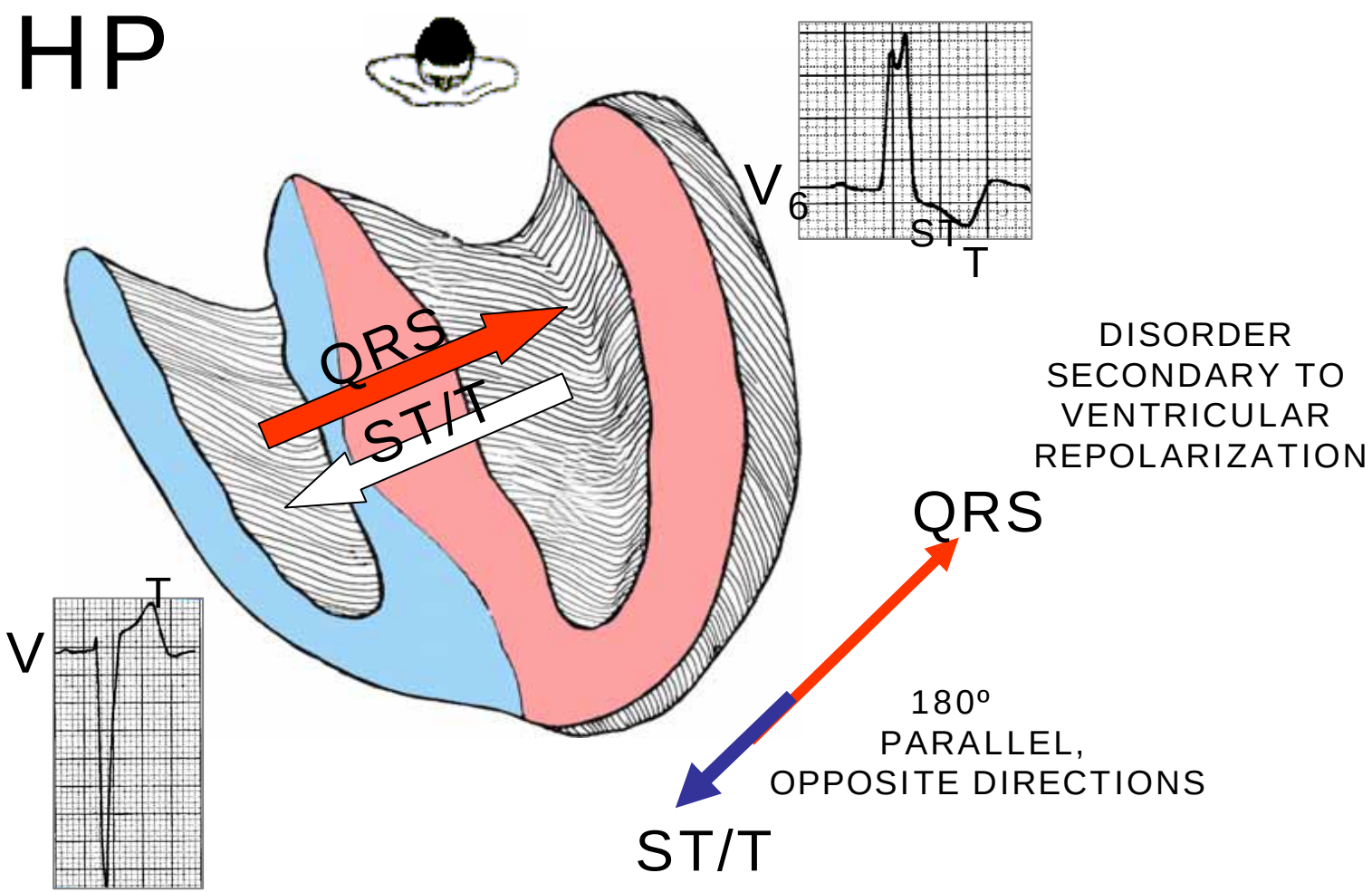




R-peak time = 80ms

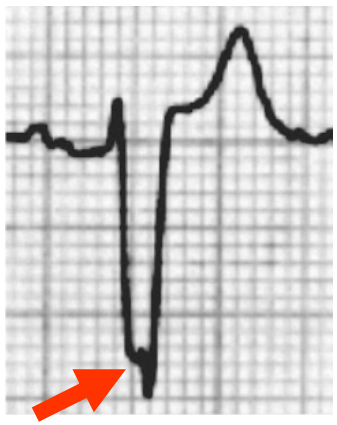
Morphology in aVR and increase of intrinsicoid deflection (≥50 ms) in DI and V6.

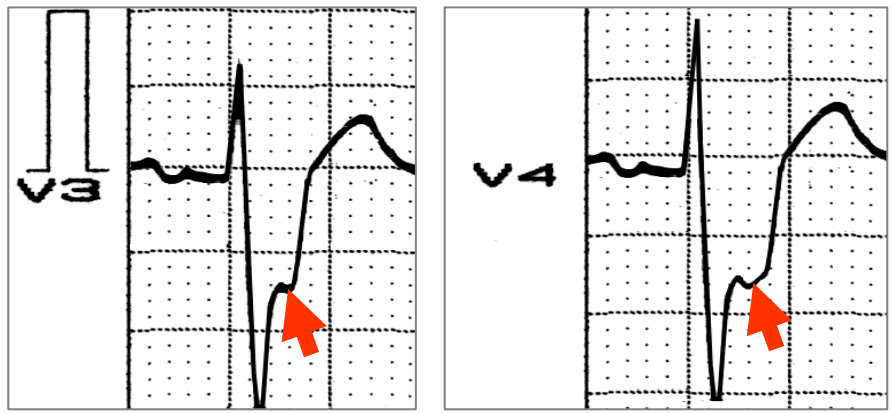
VENTRICULAR REPOLARIZATION IN COMPLETE LBBB



Outline representing ventricular repolarization in CLBBB not complicated. Secondary alteration of ventricular repolarization is observed with QRS/ST-T angle near the 180degree.

6.Notches inside of QRS complexes: notches inside of prolonged QRS complexes are indicative of myocardial scar, and markers of conduction abnormality and mayor tendency to reentrant arrhythmias. Notching of a QRS complex with normal QRS duration should not be diagnosed as an intraventricular conduction disturbance. In most such cases, the notch can be explained by a nearly perpendicular projection of the depolarization vector inscribed at that time.(1). 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. (2;3)





The present case has not the Cabrera sign because the notch is in the descending ramp of S wave

7. **Isolate Premature ventricular contraction** (strip prolonged in II lead below)

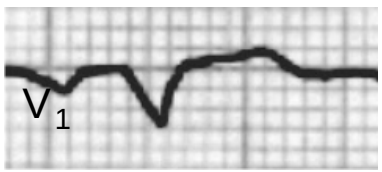
1. **Surawicz B, Knilans TK, In Chou's ELECTROCARDIOGRAPHY IN CLINICAL PRACTICE. Sixth Edition SAUNDERS ELSERVIER, 2008; Chapter 6: pp:120-122.**
2. **Kindwall KE, Brown JP, Josephson ME.Predictive accuracy of criteria for chronic myocardial infarction in pacing-induced left bundle branch block. Am J Cardiol. 1986; 57:1255-1260.**
3. **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.**

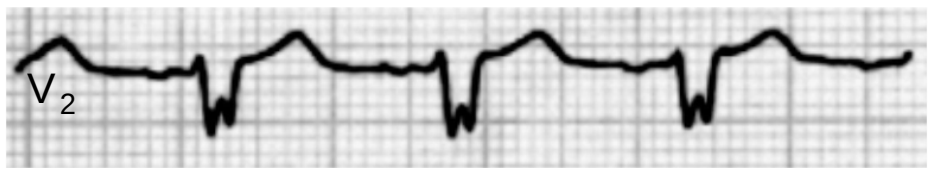
It owns this ECG fragmentation QRS complexes?

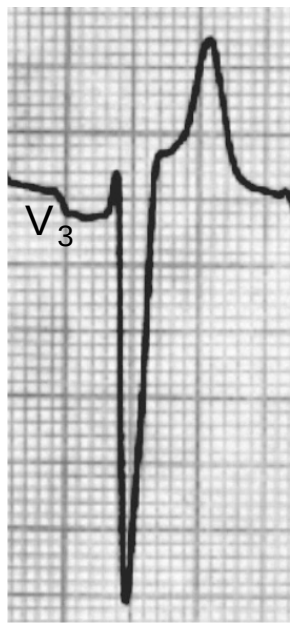
Answer: No. Why? Because fragmented QRS interval (fQRS) is defined as a QRS complex without abnormal Q waves and with the presence of multiple notches in R and S within a **non-wide QRS complex**,(The present case QRSd =12-ms) or when two adjacent leads show RSR' patterns, such as an additional R (R') in the absence of bundle branch block. (1) In others words the presence of a "notch" within a non-wide QRS complex in two adjacent leads.

On the whole both fQRS and notches inside of QRS complexes are indicative of myocardial scar, and markers of conduction abnormality but fQRS may occur in the absence of myocardial scar or heart disease.(2)

8. Possible old necrosis middle of septum? or indirect criteria of Right Atrial Enlargement?

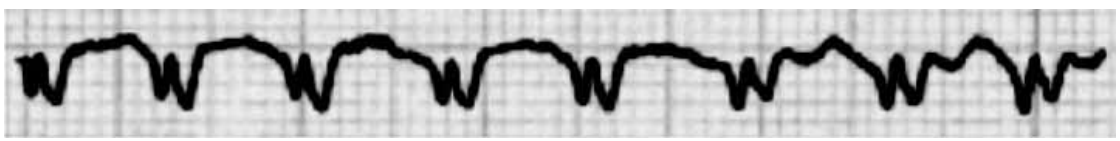






QRS complexes of low voltage in V1-V2 contrasting with QRS complexes of normal voltage or increased in V3: Peñaloza and Tranchesi sign(3). Indirect criteria of Right Atrial Enlargement**.**

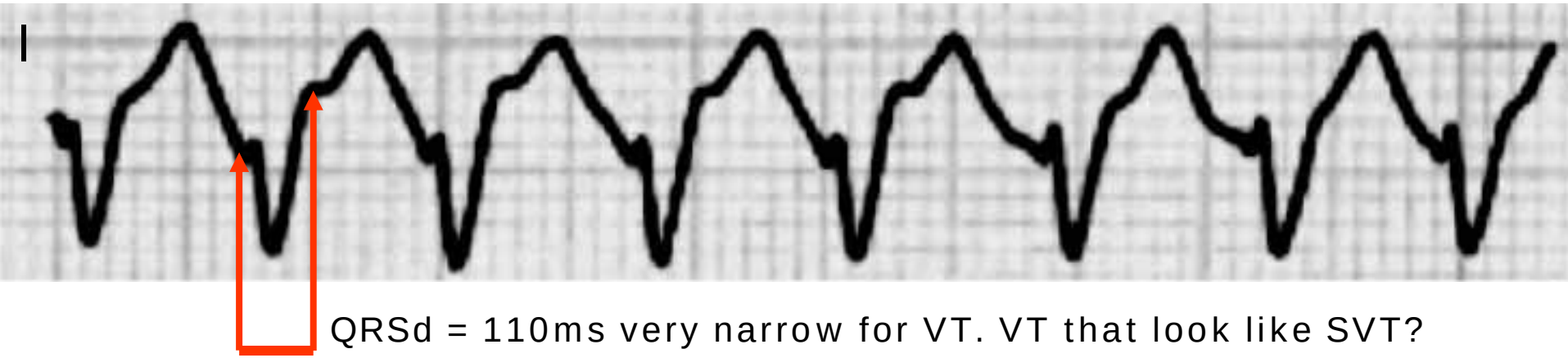
9. Possible inactive area inferior

II 

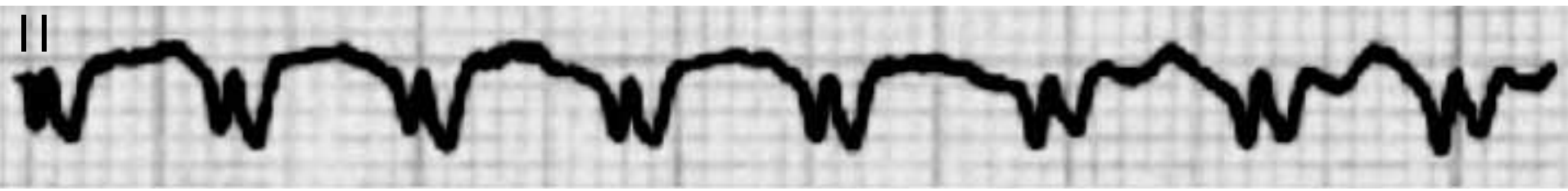
1. **MacAlpin RN. The fragmented QRS: does it really indicate a ventricular abnormality? J Cardiovasc Med (Hagerstown). 2010 Nov; 11: 801-809.**
2. **Morita H, Kusano KF, Miura D, et al. Fragmented QRS as a marker of conduction abnormality and a predictor of prognosis of Brugada syndrome. Circulation. 2008 Oct 21;118:1697-1704.**
3. **PENALOZA D, TRANCHESI J. The three main vectors of the ventricular activation process in the normal human heart. I. Its significance. Am Heart J. 1955 Jan;49:51-67.**

ECG2

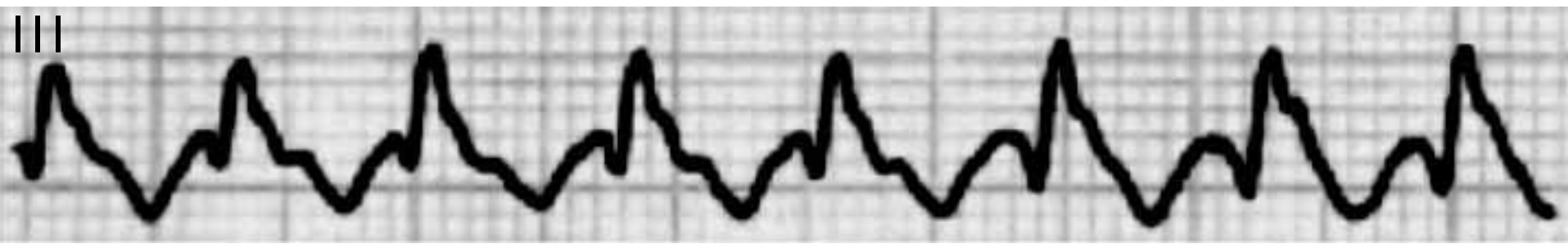
Regular QRS tachycardia HR 187bpm, QRS duration only 110ms



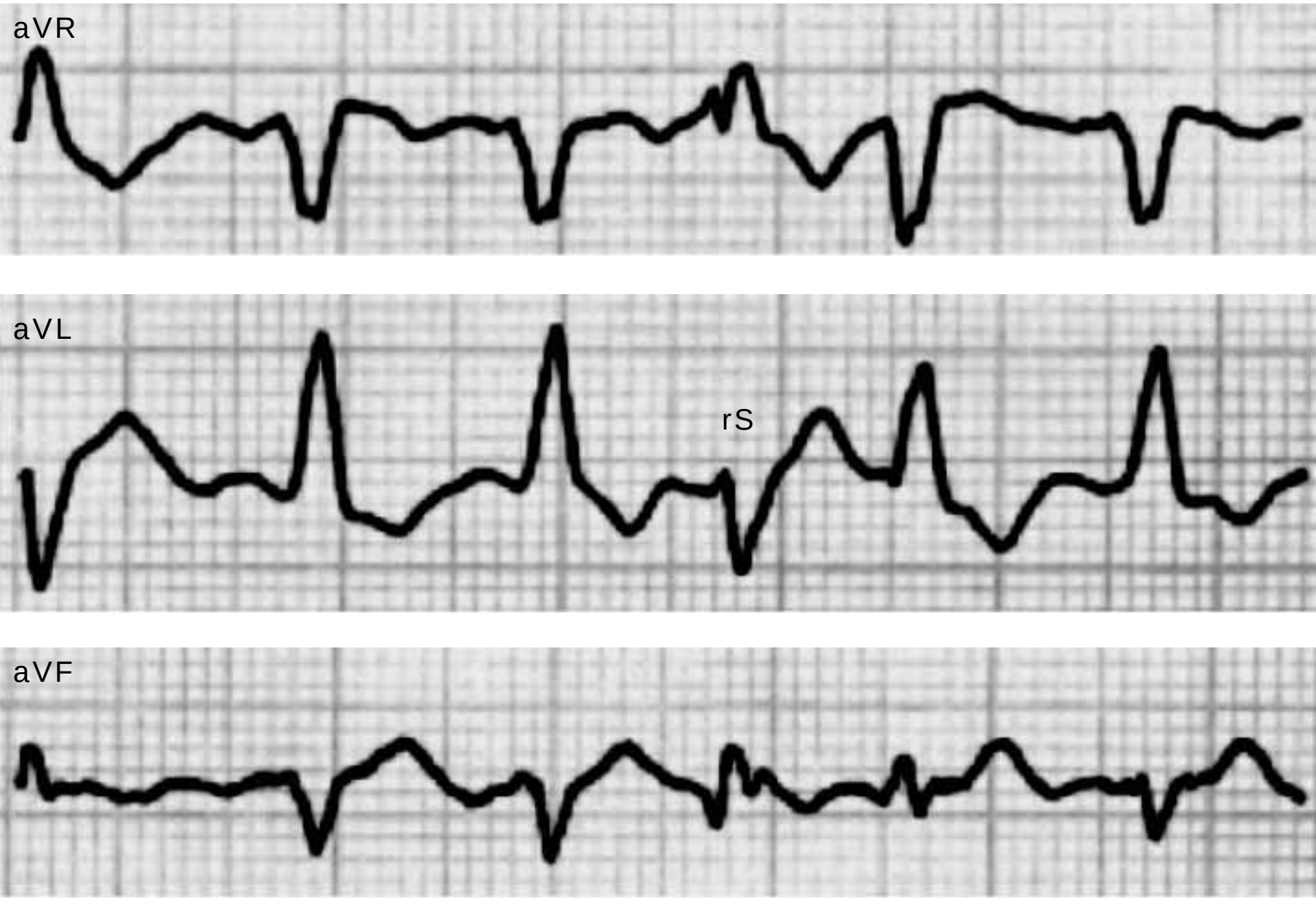
QRSd = 110ms very narrow for VT. VT that look like SVT?

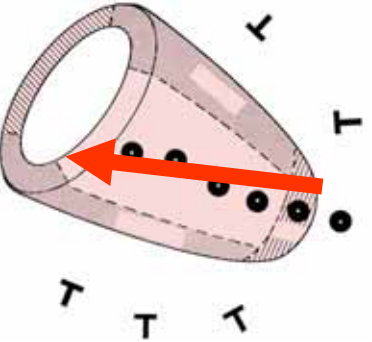


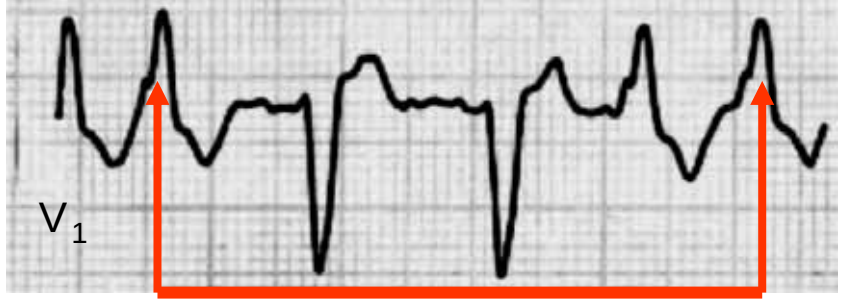
QRS axis with right axis deviation



ECG2



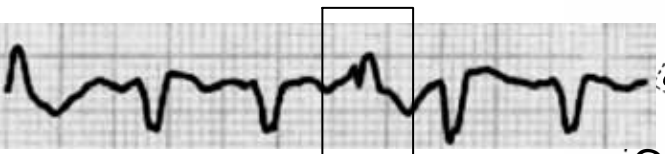


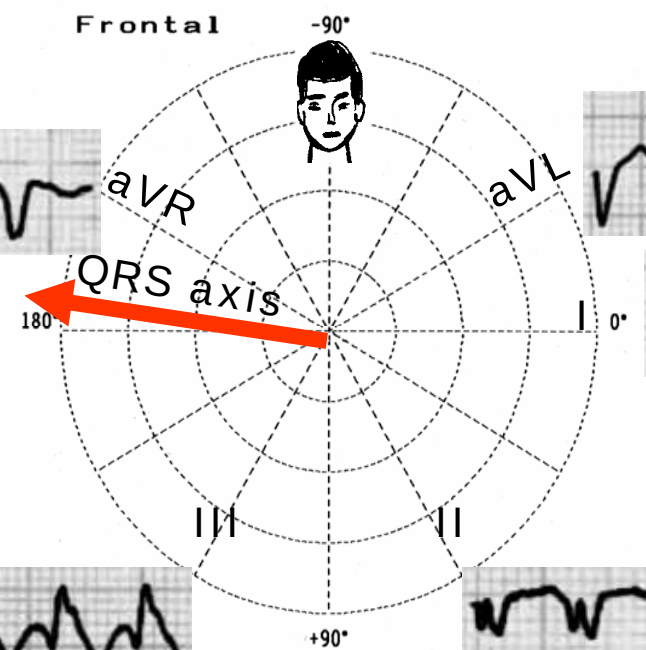


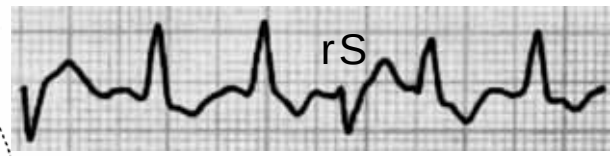
Monophasic positive QRS complex in lead V_1

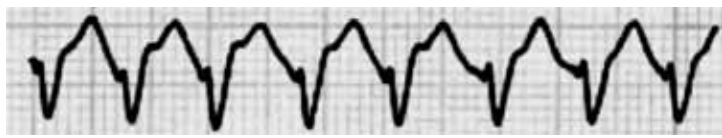
ECG2

LV focus

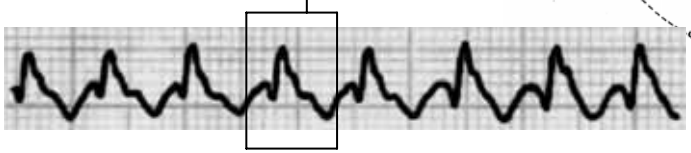


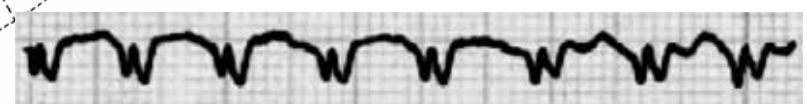






65% of cases of right ventricular ectopy had a negative QRS complex in I

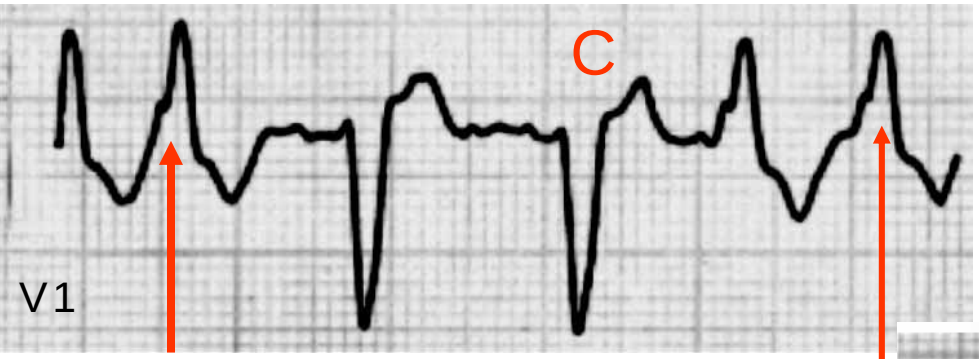




In the V_1 positive broad QRS tachycardia, right or left axis deviation is supportive of VT
There is an erroneous perception that SVT with aberrancy is as common as VT.
In the differential diagnosis of broad QRS tachycardia, hemodynamic status, age, and HR should not be used.

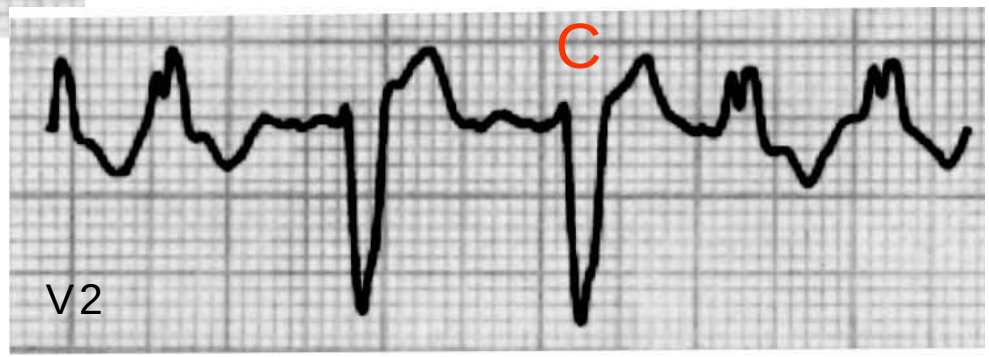
ECG2

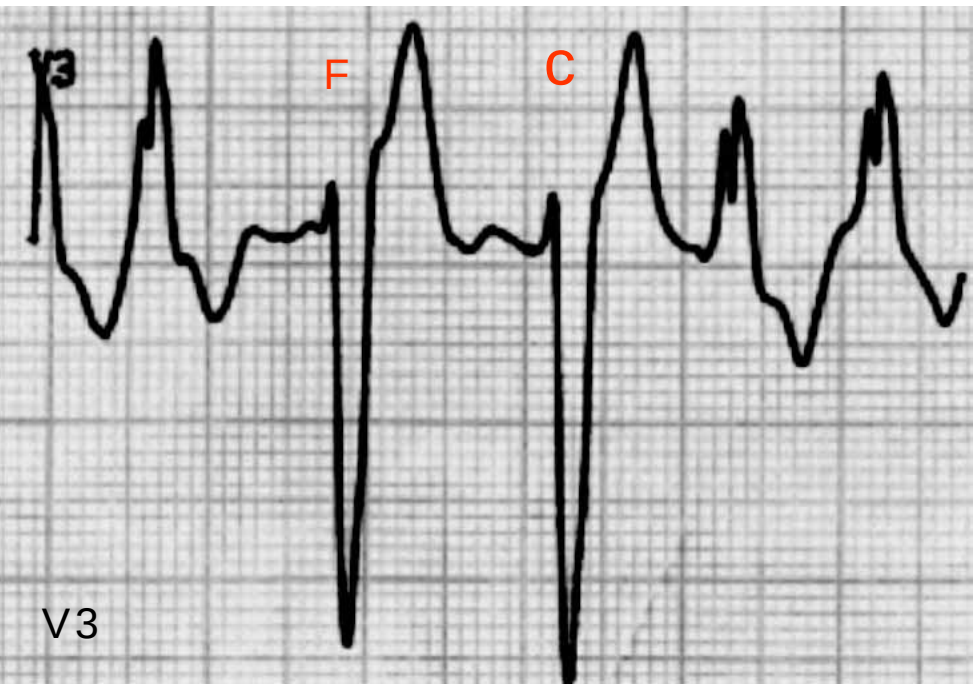
The QRS complexes have a RBBB pattern, QRS axis deviated to right, QRS duration with relatively narrow (110 ms) and so is commonly misdiagnosed as a supraventricular tachycardia (SVT).



Monophasic positive QRS complex in lead V_1

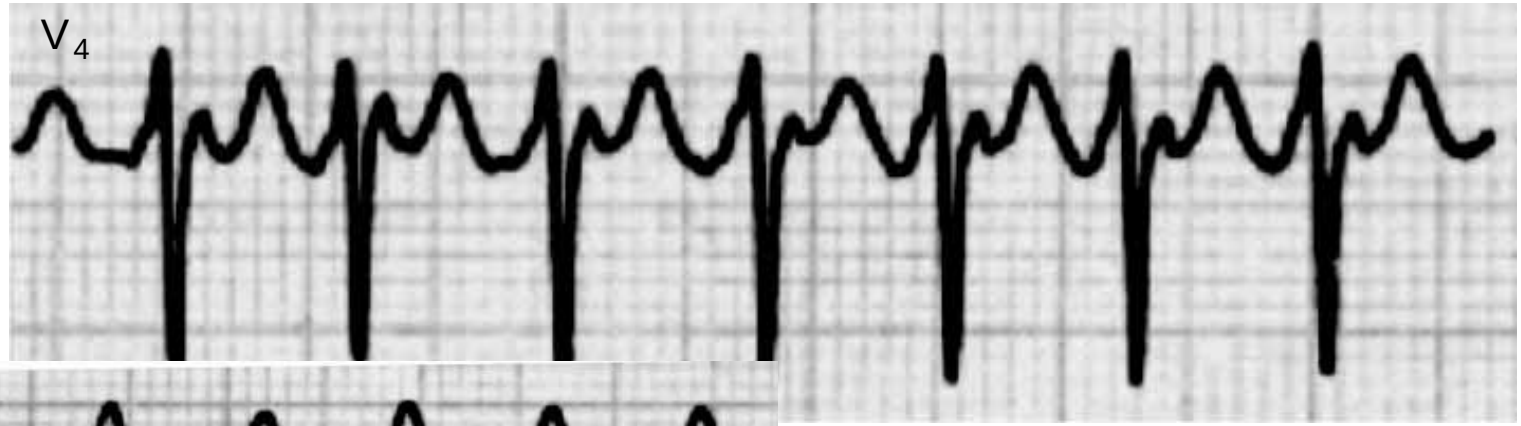
VT

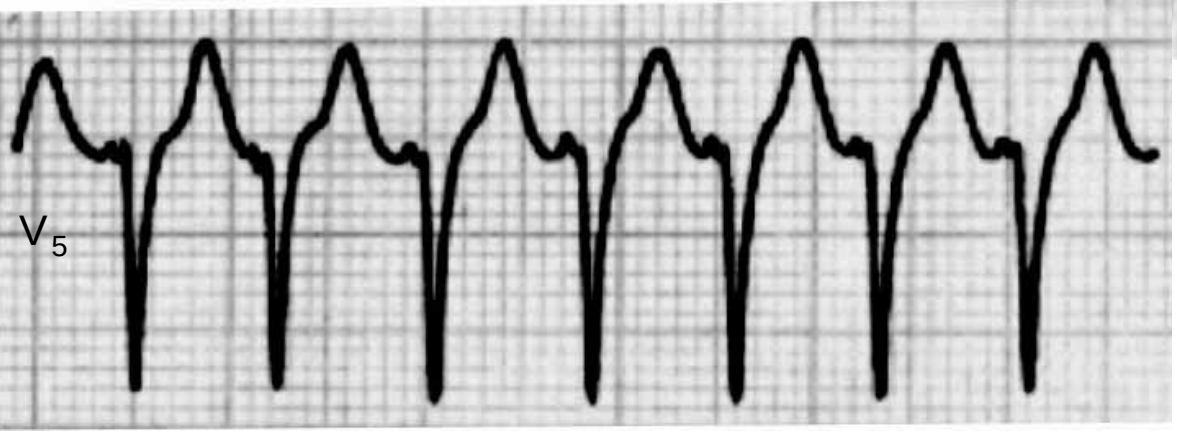




1. C: Capture and fusion (F) beats are ECG signs of dissociation. Fusion and capture beats are strong evidence of VT but is not diagnosis because are also seen in AF with conduction over an accessory pathway.

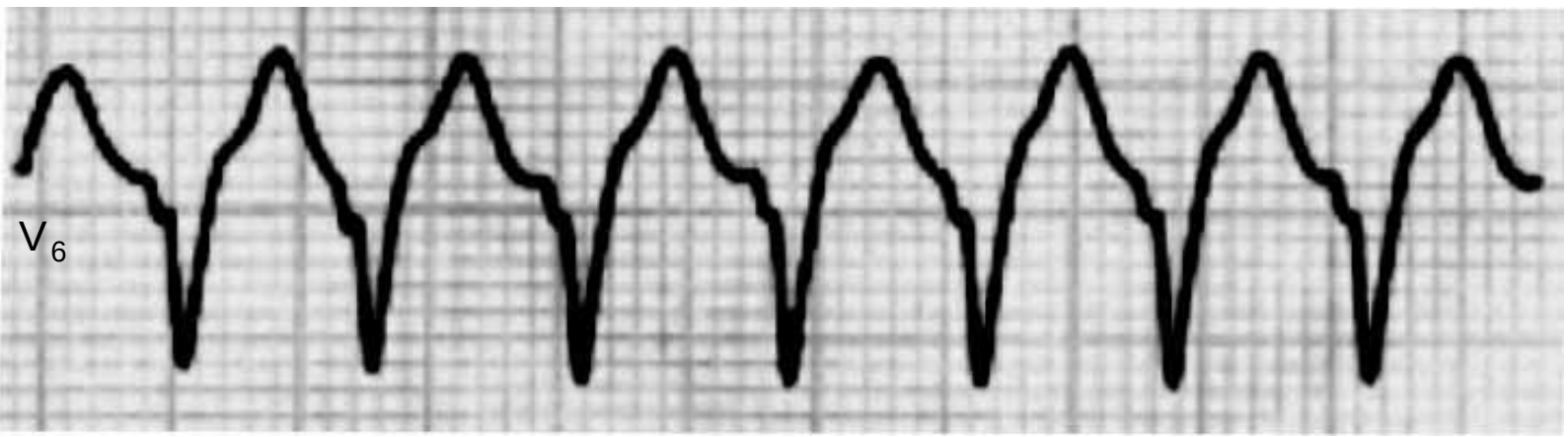
ECG2





R:S ratio in V6<1

Presence of predominantly negative QRS complexes from V4 to V6 are criteria in favor VT



ECG 2 diagnosis:

Fascicular ventricular tachycardia VT that look like SVT

Adenosine-responsive VTs, Belhansen VT or verapamil sensitive

1. Uncommon and not usually associated with underlying structural heart disease.
2. They appear in patients without any indication of heart disease(idiopathic VT)
3. Rarely in patient with dilated cardiomyopathy (bundle branch reentrant VT)
4. Recently was described in association with coronary heart disease(1).
5. Are also observed in patients with digitalis intoxication
6. It originates from the left bundle branch or left posteroinferior fascicle.
7. It produces QRS complexes of relatively short duration (110-140ms) and so is commonly misdiagnosed as a supraventricular tachycardia (SVT). It is a mixed group of VT that look like SVT
8. The QRS complexes have a right bundle branch block pattern.
9. VTs associated with the His-Purkinje system may occur in patients with and without organic heart disease. The former may encounter bundle branch reentrant VT, a macroreentrant VT utilizing the specific conduction system. It frequently occurs in patients with preexisting conduction disturbance such as complete LBBB and may be eliminated by catheter ablation of the right bundle branch. After successful ablation, patient's prognosis depends on the presence or absence of structural heart disease.In patients without structural heart disease, VT with RBBB pattern and superior axis, referred to as idiopathic left VT, is observed. It is a reentrant VT utilizing the posterior left fascicle and the Purkinje network. The two treatment options include antiarrhythmic drug therapy with verapamil or curative RFCA.

1. **Jane-Wit D, Batsford W, Malm B. Ischemic etiology for adenosine-sensitive fascicular tachycardia. J Electrocardiol. 2011 Mar-Apr;44:217-221.**

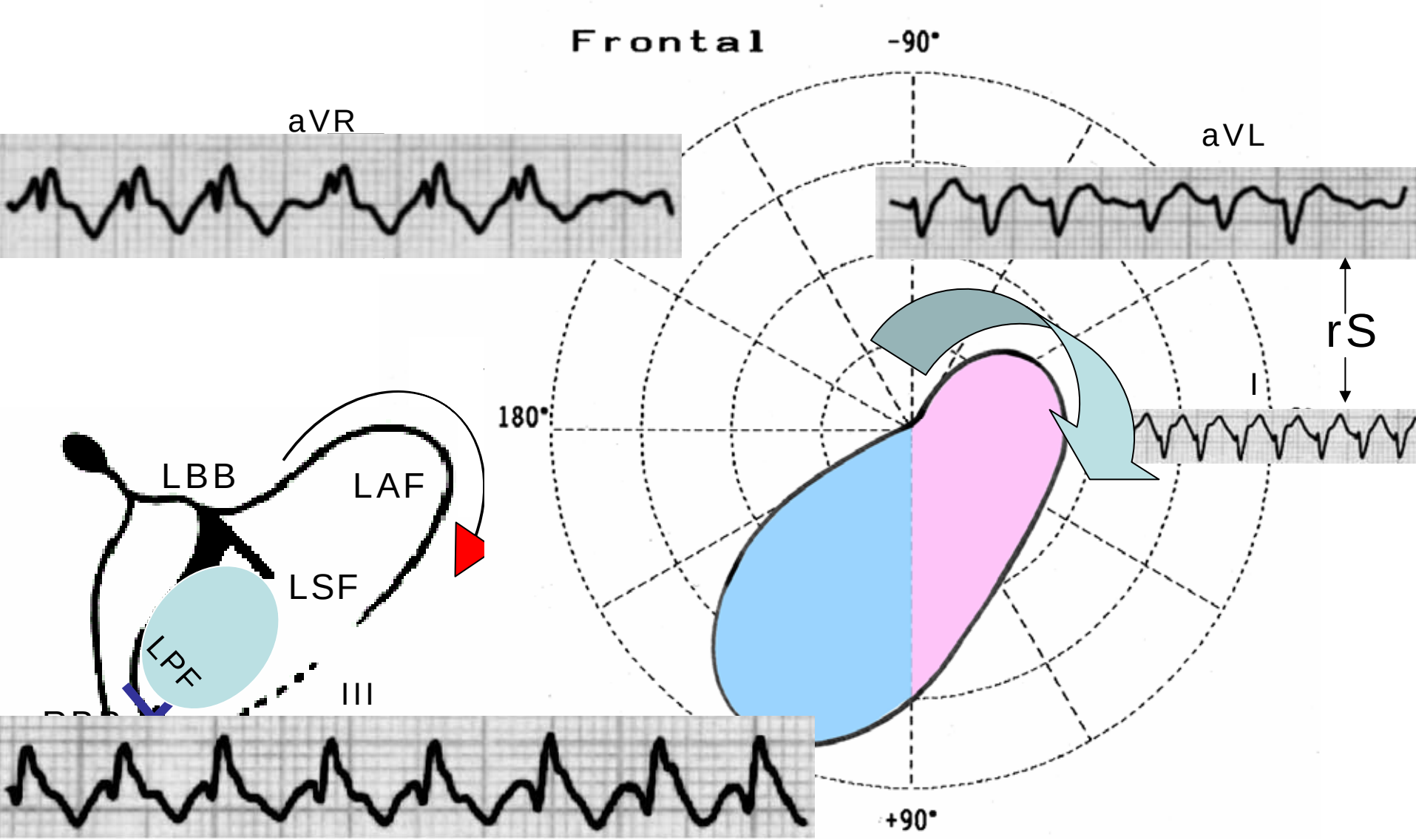
Another form of ventricular arrhythmia originating in the Purkinje network is idiopathic ventricular fibrillation (IVF). Focal triggers from the right and left ventricular Purkinje network induce PVCs inducing IVF. This is amenable to catheter ablation leading to a significant reduction in ICD interventions in sudden cardiac death survivors.(1).

Sustained Fascicular Bundle Branch Reentrant VT: it is a highly malignant for of M-VT that frequently is manifested by palpitations, syncope or SCD. It is associated com severe myocardial disease or with significant disease of the intraventricular conduction system. The ECG features are: 1) ECG base: LBBB or non specific conduction delay consistent with His-Purkinje system disease. If sinus rhythm PR interval frequently prolonged(in this case is border line 200ms) 2) During the event LBBB pattern (98% of cases) or RBBB pattern with right axis deviation(2%).This is the possible variant in this case. Fascicular reentrant VT use the left anterior fascicle of the LBBB anterogradely and the left posterior fascicle retrogradely. and may produce identical QRS morphology during sinus rhythm and VT. In a postinfarct patients with dilated LV and recurrent VT (showing a QRS configuration of RBBB, LPFB-like), endocardial recordings from the His-Purkinje system show that VT is due to interfascicular reentry. Induction of VT occur after progressive retrograde conduction delay on increasing the prematurity of the extrastimulus. The usual target for bundle branch reentry ablation, the right bundle, did not participate in the reentrant circuit. While performing left ventricular endocardial mapping, VT is interrupted when positioning the catheter on the left anterior fascicle, and "reversed" nonsustained BBB reentry occurred with anterograde conduction over the posterior fascicle and retrograde conduction over the anterior fascicle. Ablation of conduction in the anterior fascicle led to cure of the VT.

Interfascicular reentrant VT with RBBB, right-axis QRS configuration can be cured by catheter ablation of anterior fascicle conduction(2).

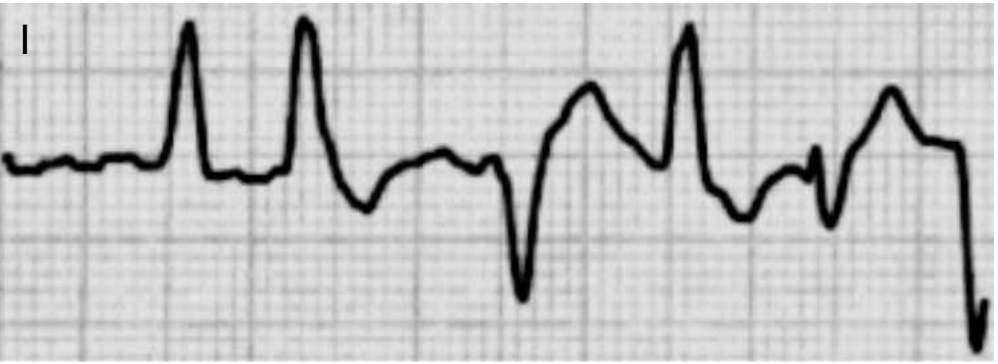
1. **Schmidt B, Chun KR, Kuck KH, et al. Ventricular tachycardias originating in the his-purkinje system. Bundle branch reentrant ventricular tachycardias and fascicular ventricular tachycardias. Herz. 2009 Nov;34:554-560.**
2. **Crijns HJ, Smeets JL, Rodriguez LM, et al. Cure of interfascicular reentrant ventricular tachycardia by ablation of the anterior fascicle of the left bundle branch. J Cardiovasc Electrophysiol. 1995 Jun;6:486-92.**

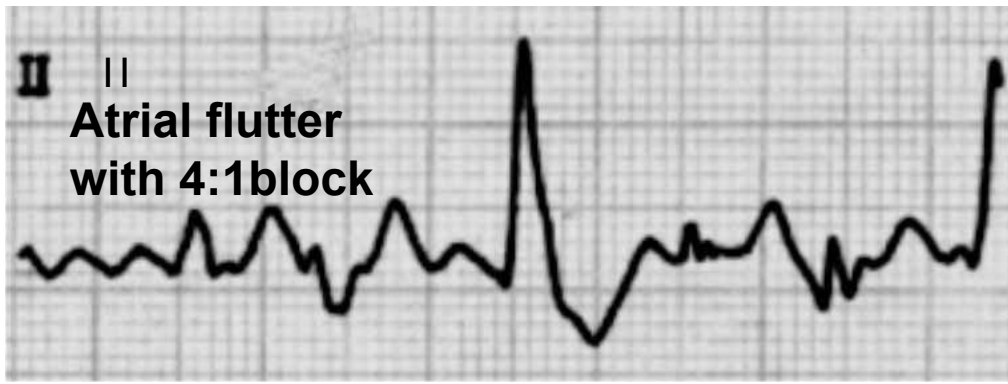
Fascicular Reentrant VT using the left anterior fascicle (LAF) of the LBBB anterogradely and the left posterior fascicle (LPF) retrogradely.

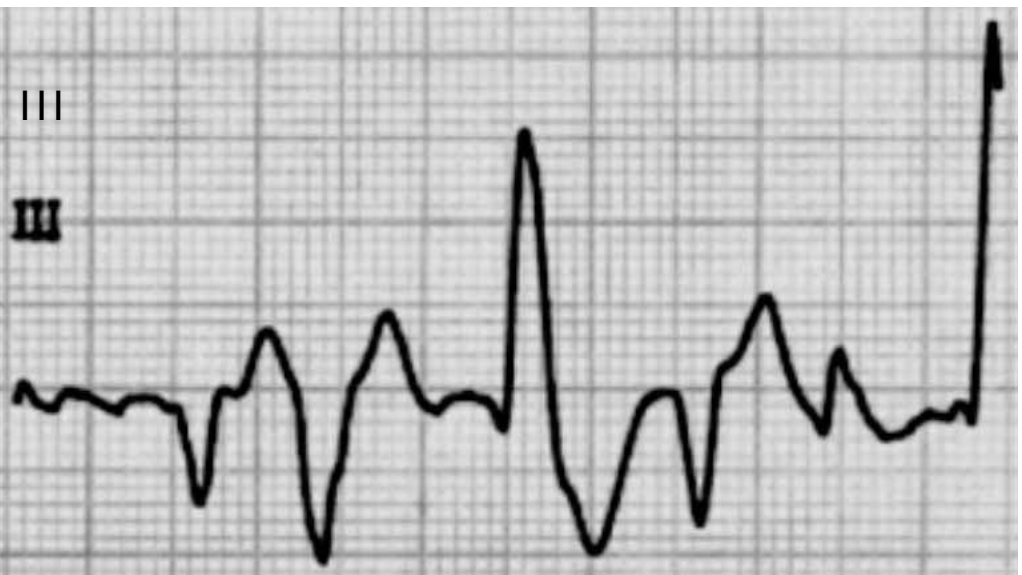


Right QRS axis deviation

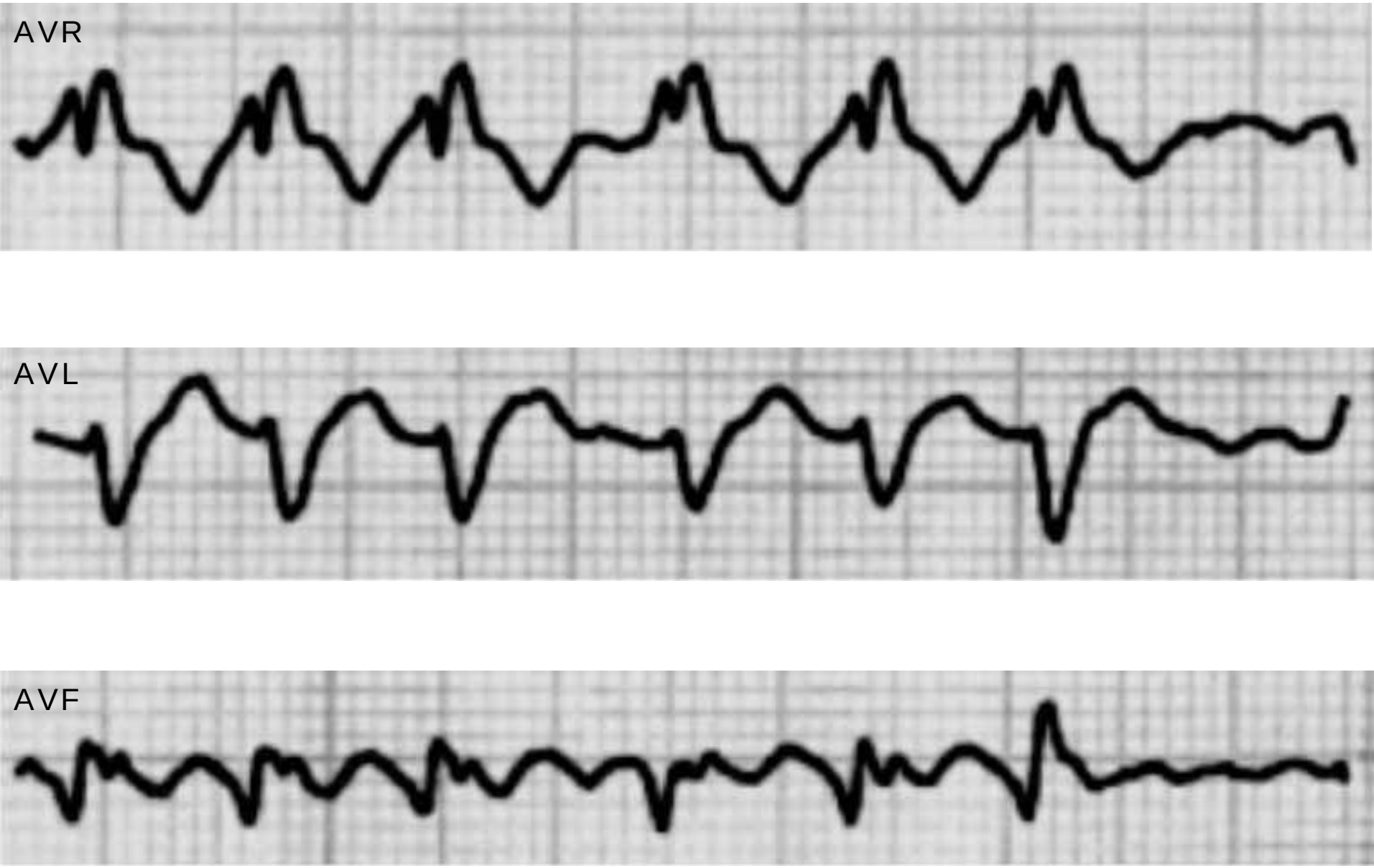
ECG 3



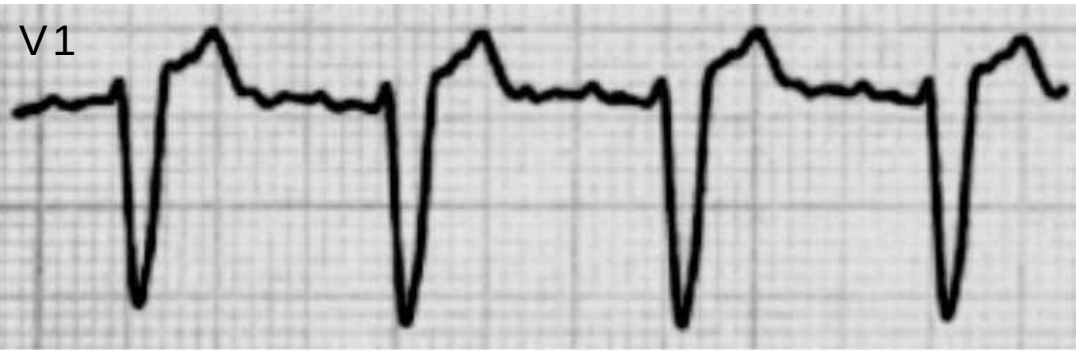


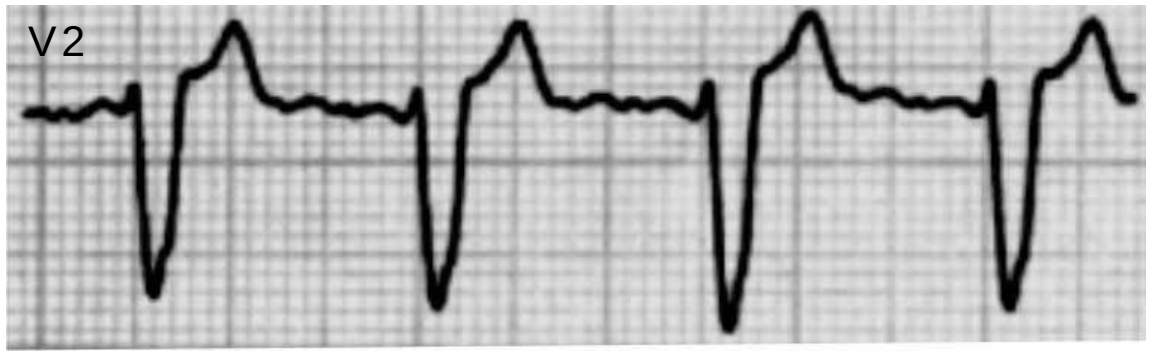


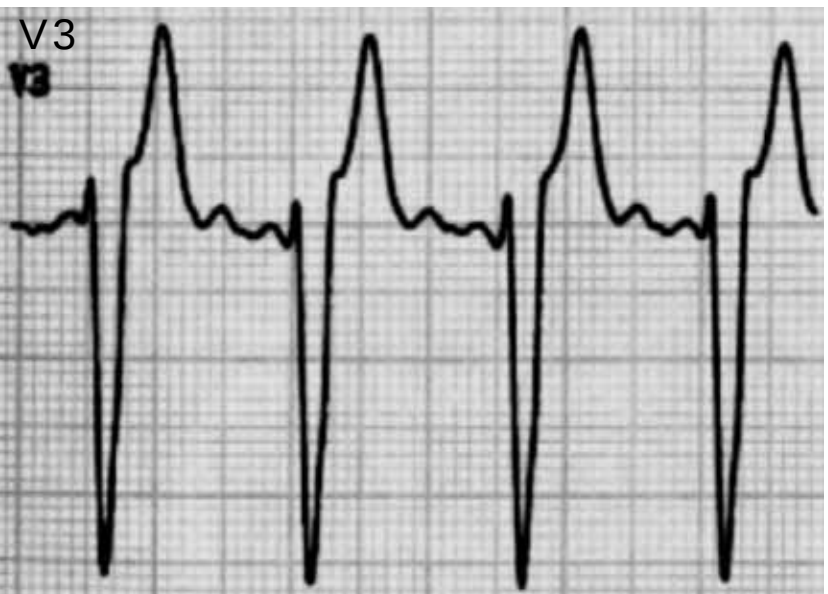
ECG III



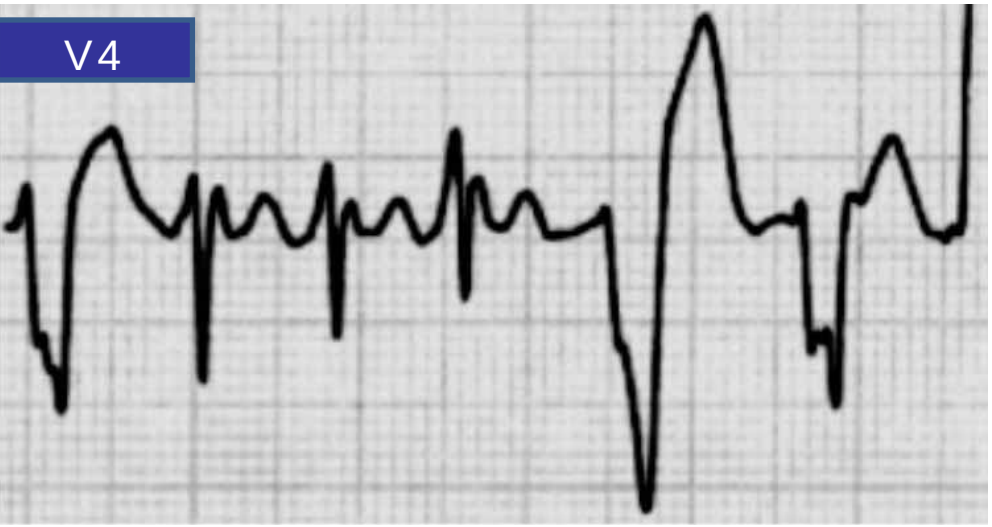
ECG 3

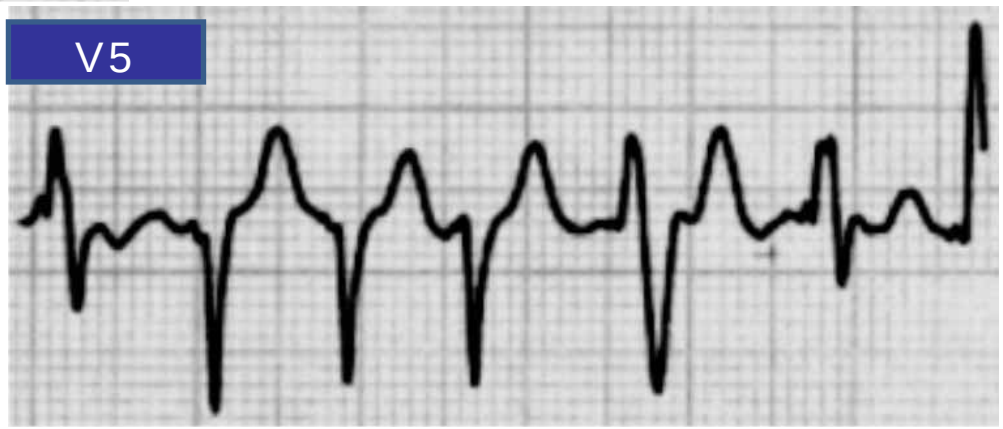


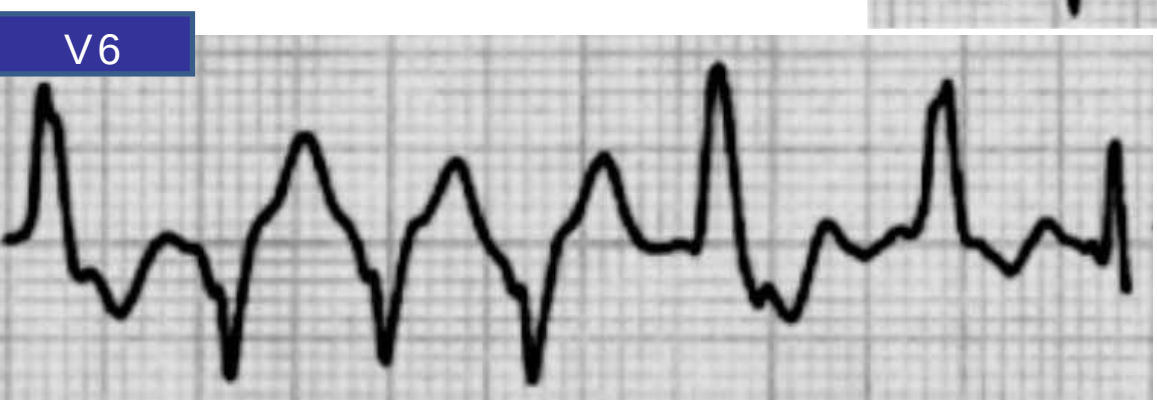




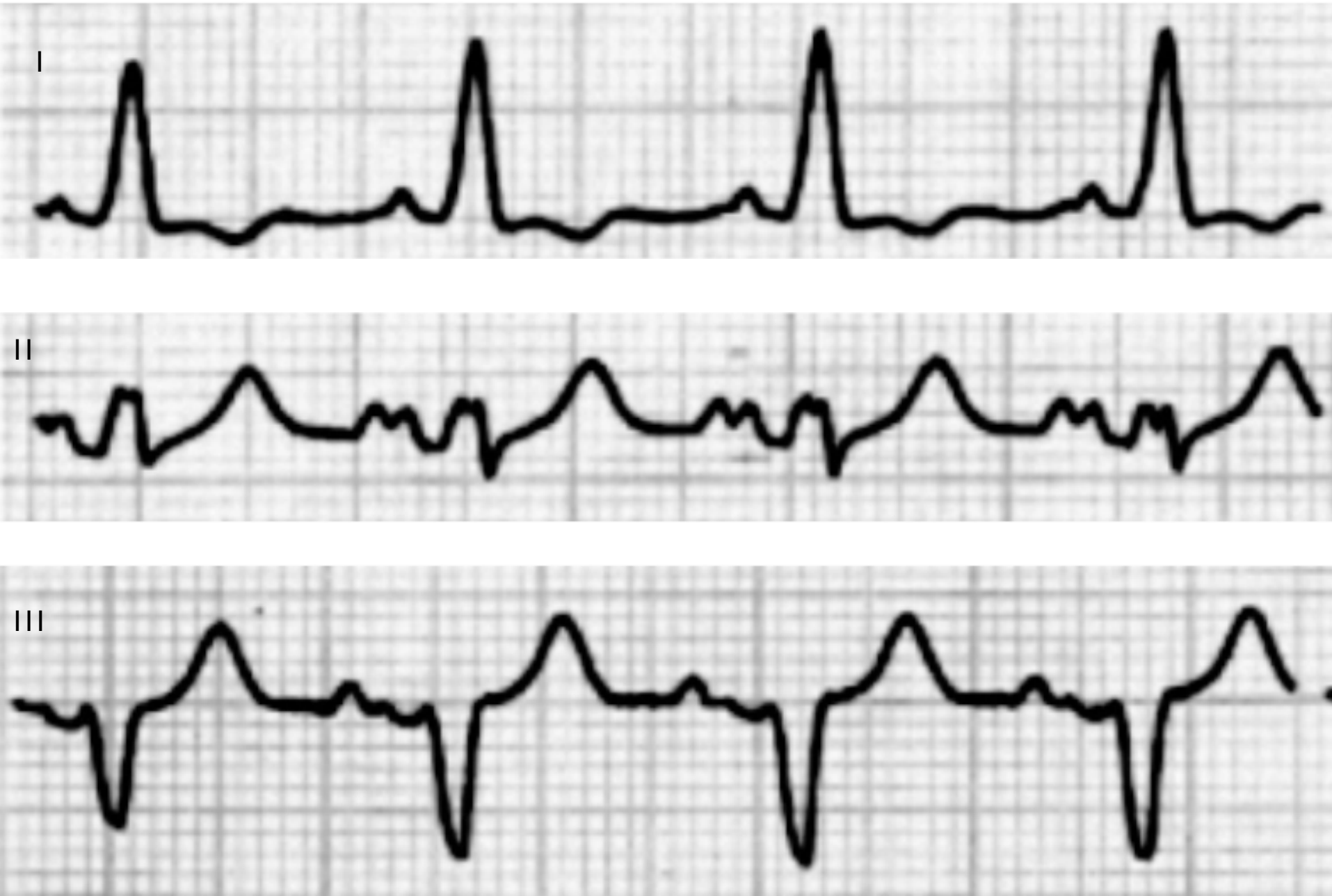
ECG 3



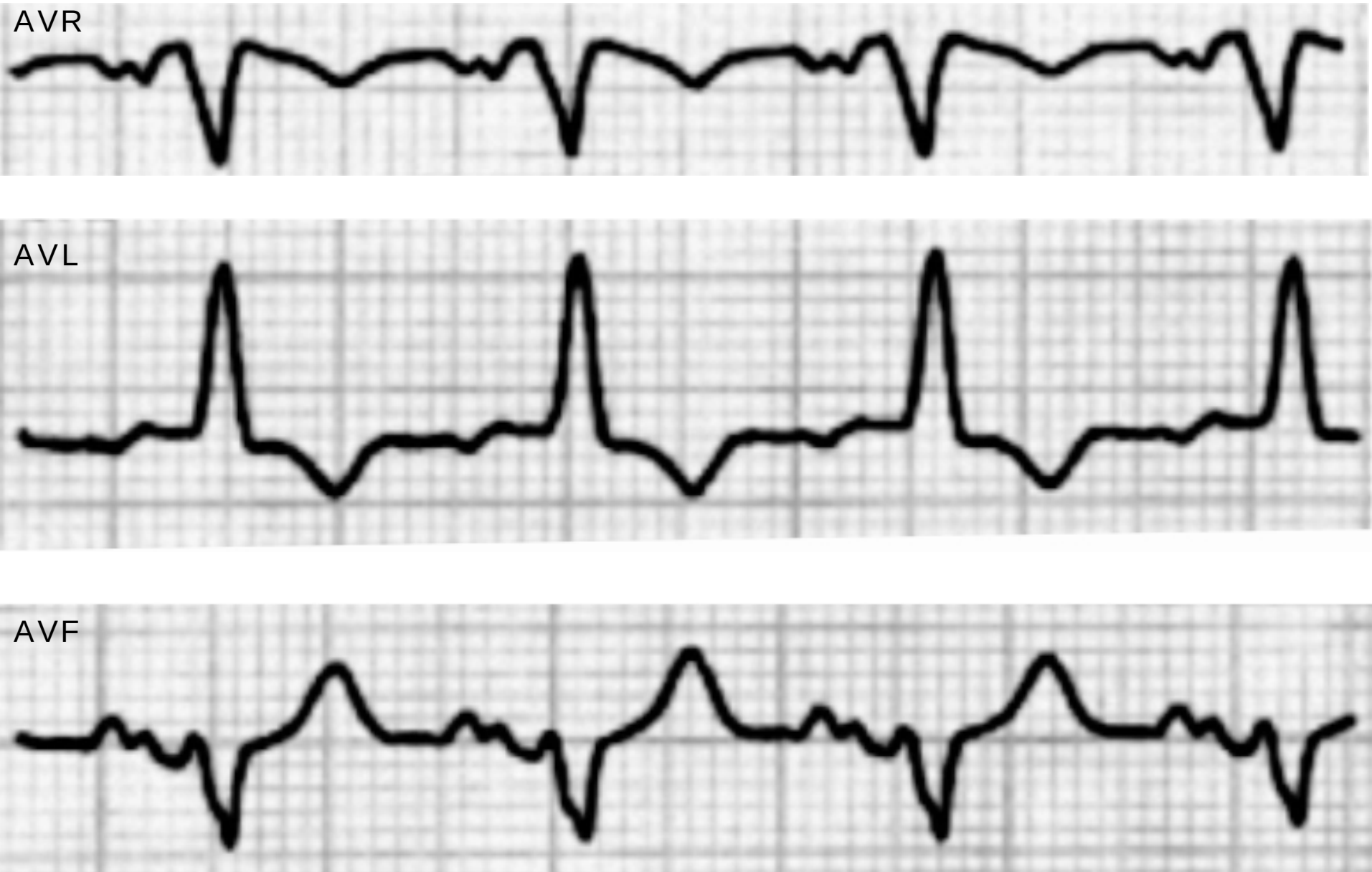




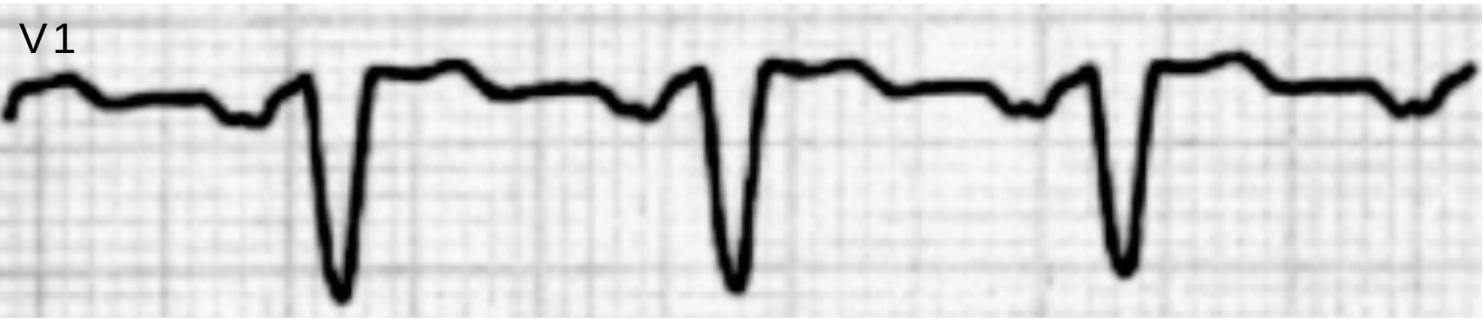
ECG 5

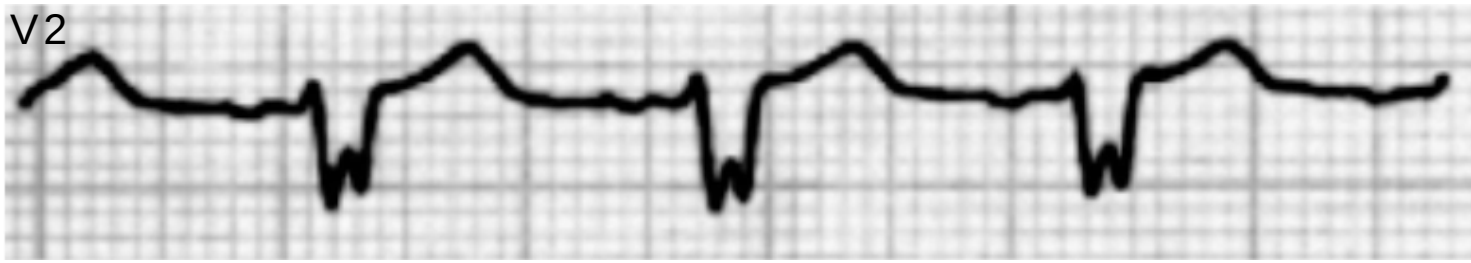


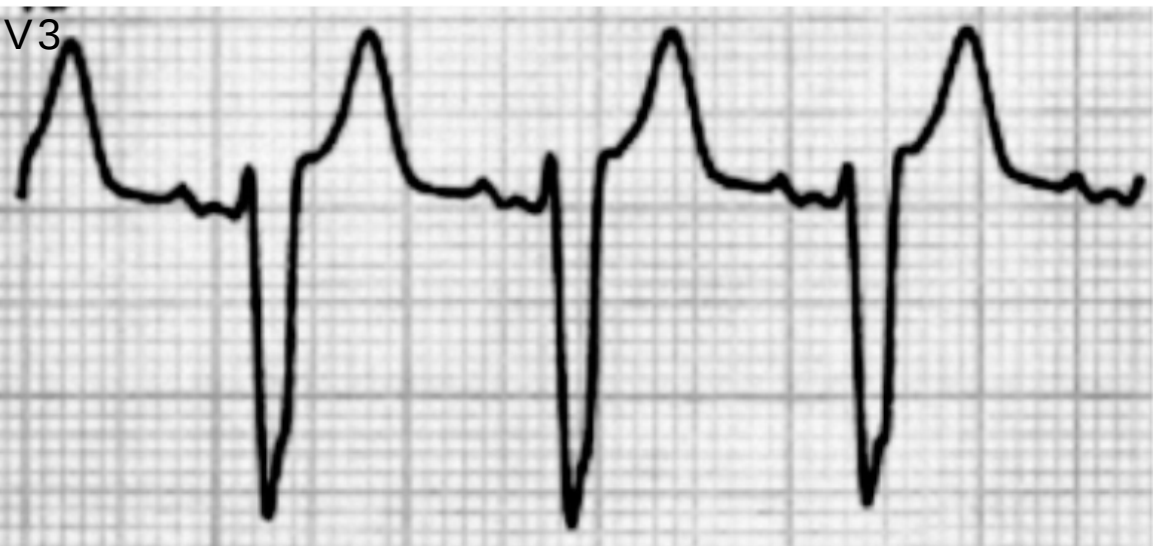
ECG 5



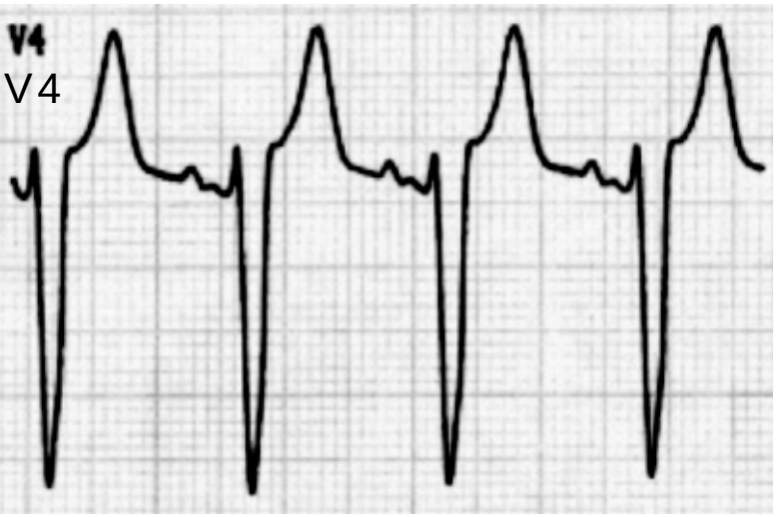
ECG 5

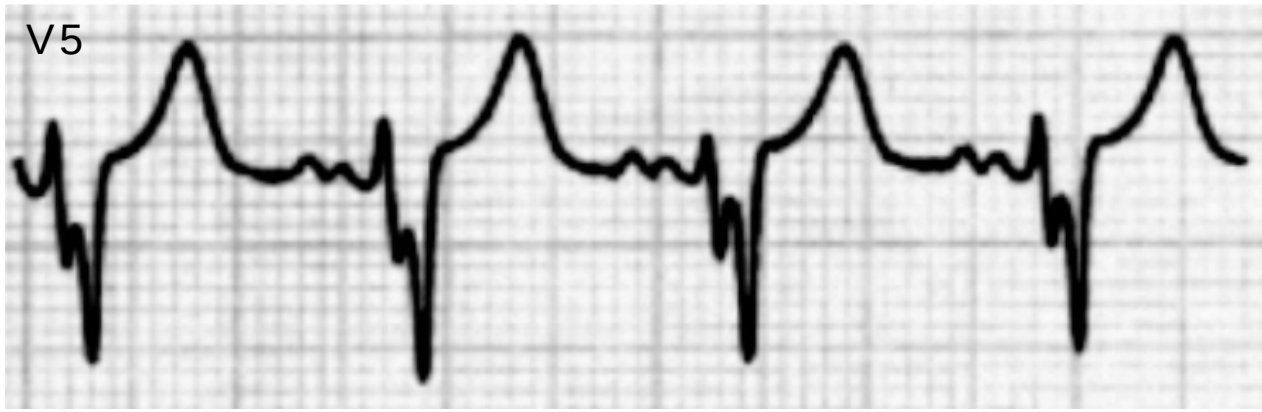


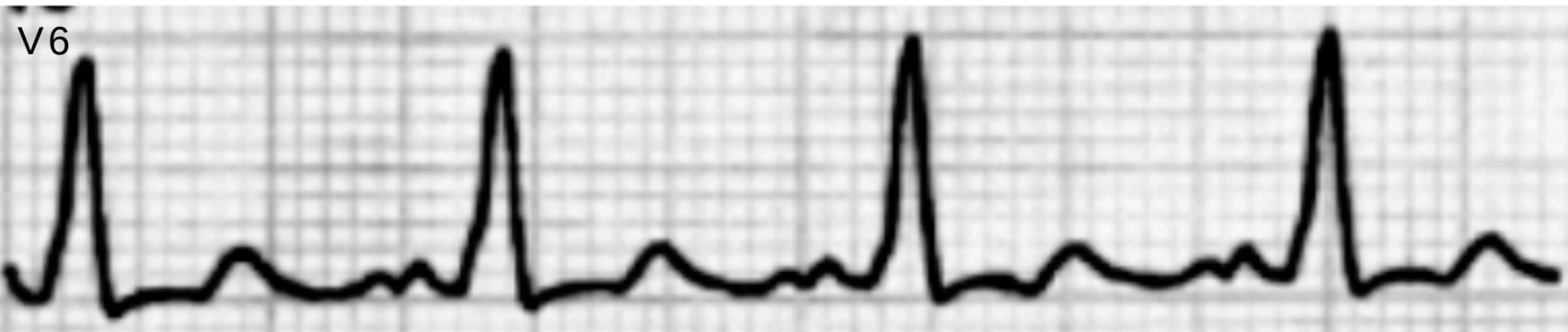


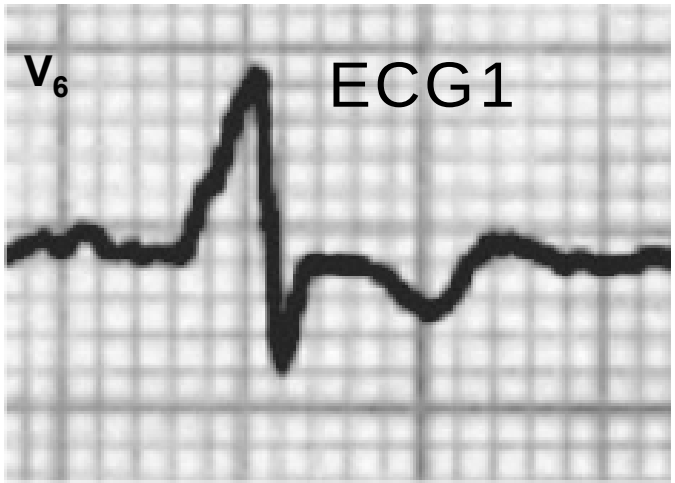


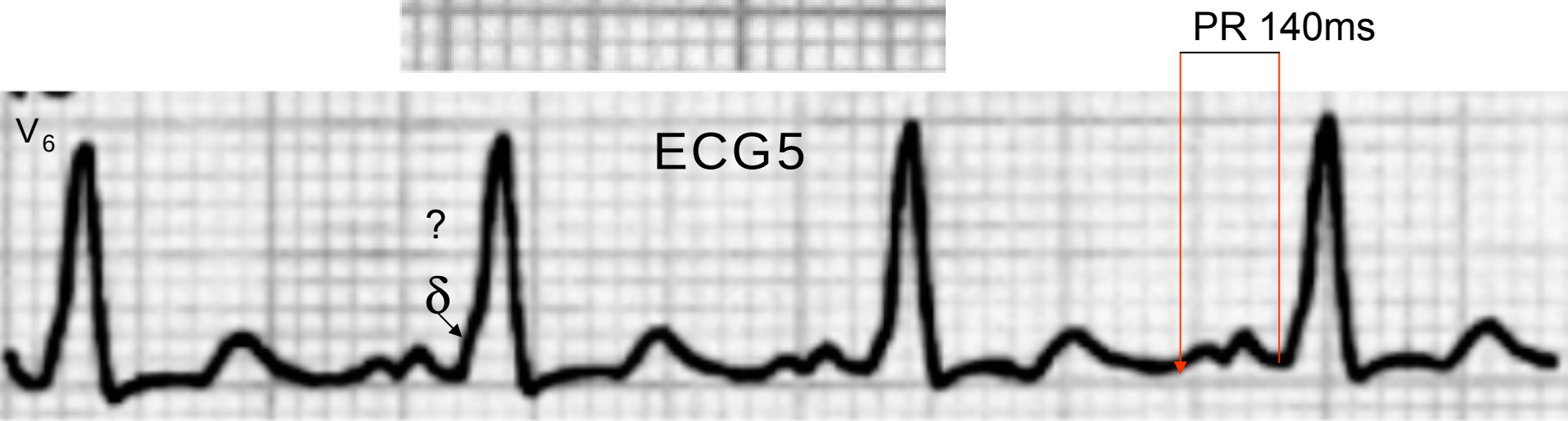
ECG 5











ECGs comparison 1 and 5 shows clearly a mistake in the height of the electrodes position:
ECG1: Rs pattern
ECG 5: R.
Additionally, the V6 lead of ECG 5 suggest Mahaim type ventricular preexcitation: by tracts(*) or by connections(**) A) Tracts: Nodal-fascicular, atrio-fascicular. B) Connections: Nodal-ventricular, fascicular-ventricular, atrioventricular.
(*) Tracts: They are accessory pathways that end in specific conduction tissue.
(**) Connections: They are the accessory pathways that end in the ventricular contractile muscle.

There are accessory pathways (anomalous pathways) that starting from the AV node, or the His bundle branch, or its branches, or even from the atrial muscles, end in the ventricular contractile myocardium (CONNECTIONS) or insert into the conduction-specific tissue fibers (TRACTS), frequently in the right branch of the His bundle.

The fibers that originate in the conduction system after the AV node, present normal PR interval duration interval duration, and if they end in the right septal contractile muscle (nodo-ventricular connections and fasciculoventricular), by slowly activating initially the RV (DELTA δ wave), the QRS pattern becomes similar to Complete LBBB (Mahaim type pre-excitation). I'm not sure.