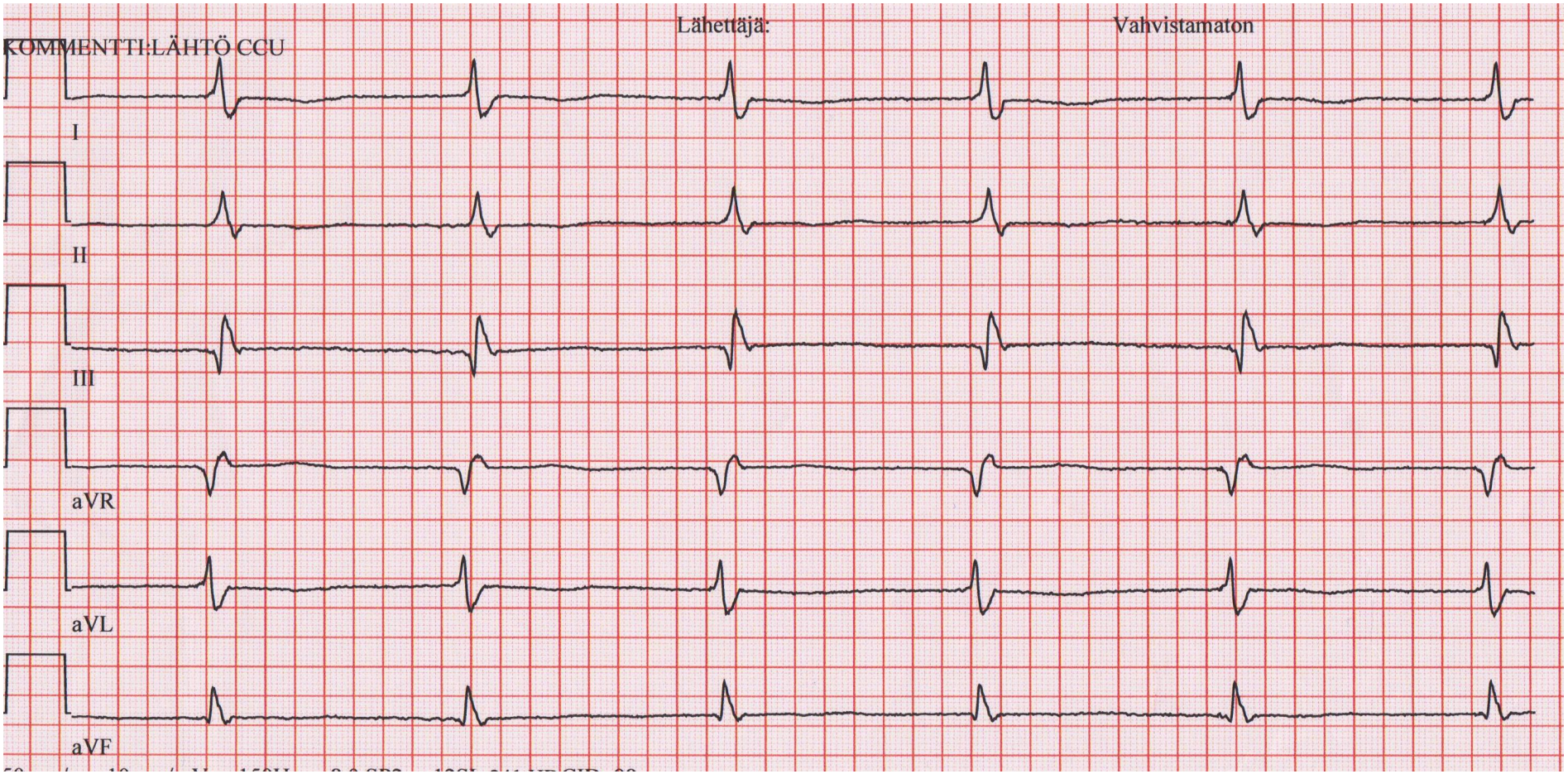
66-year old female

- LBBB since many years
- Now acute septal myocardial infarction with recurrent VT episodes
- Amiodarone infusion started
- In the CCU intermittent rhythm without P waves and narrow QRS alternating with sinus rhythm with LBBB
- No significant changes in heart rate during the two rhythms

09:30 a.m. Note: 50 mm/sec



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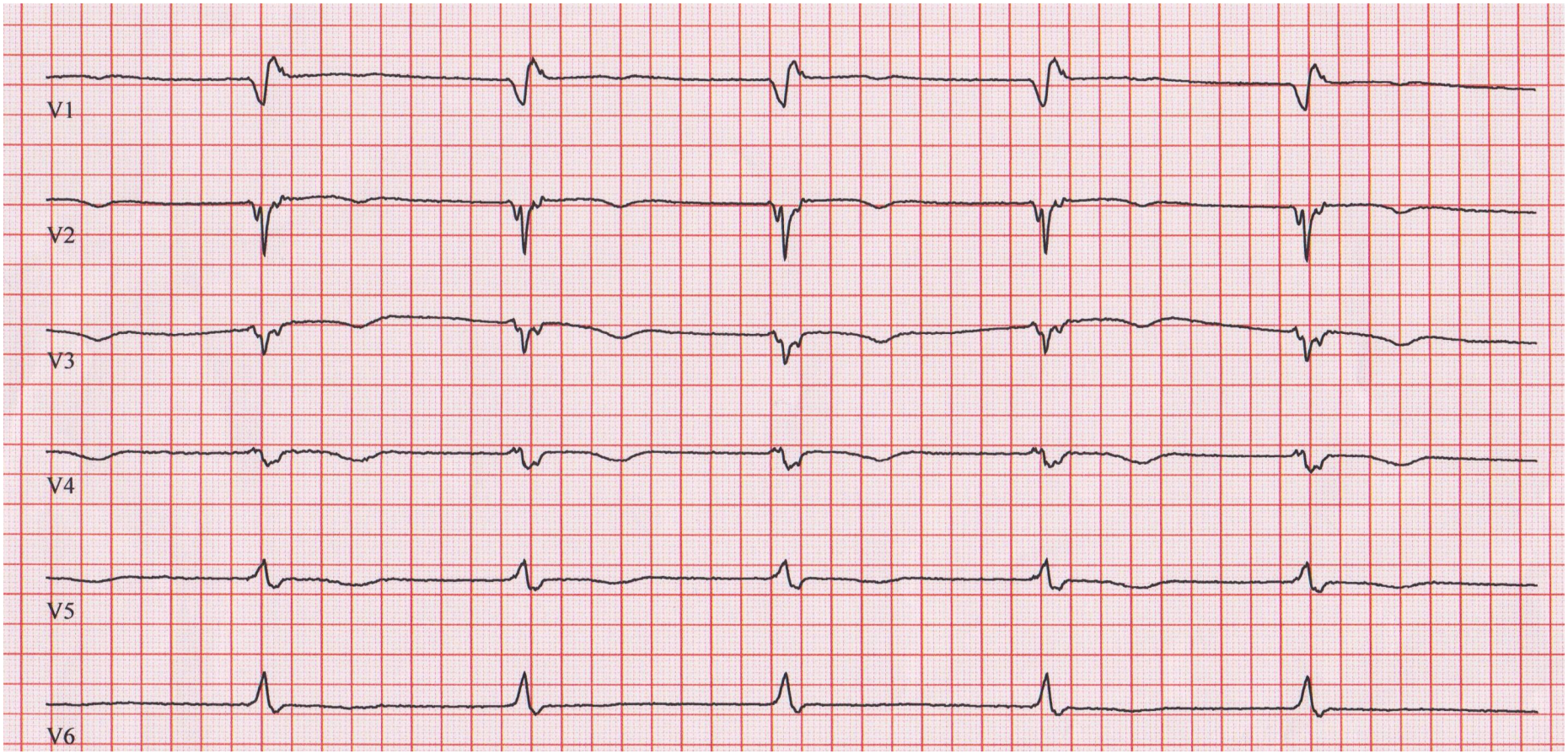

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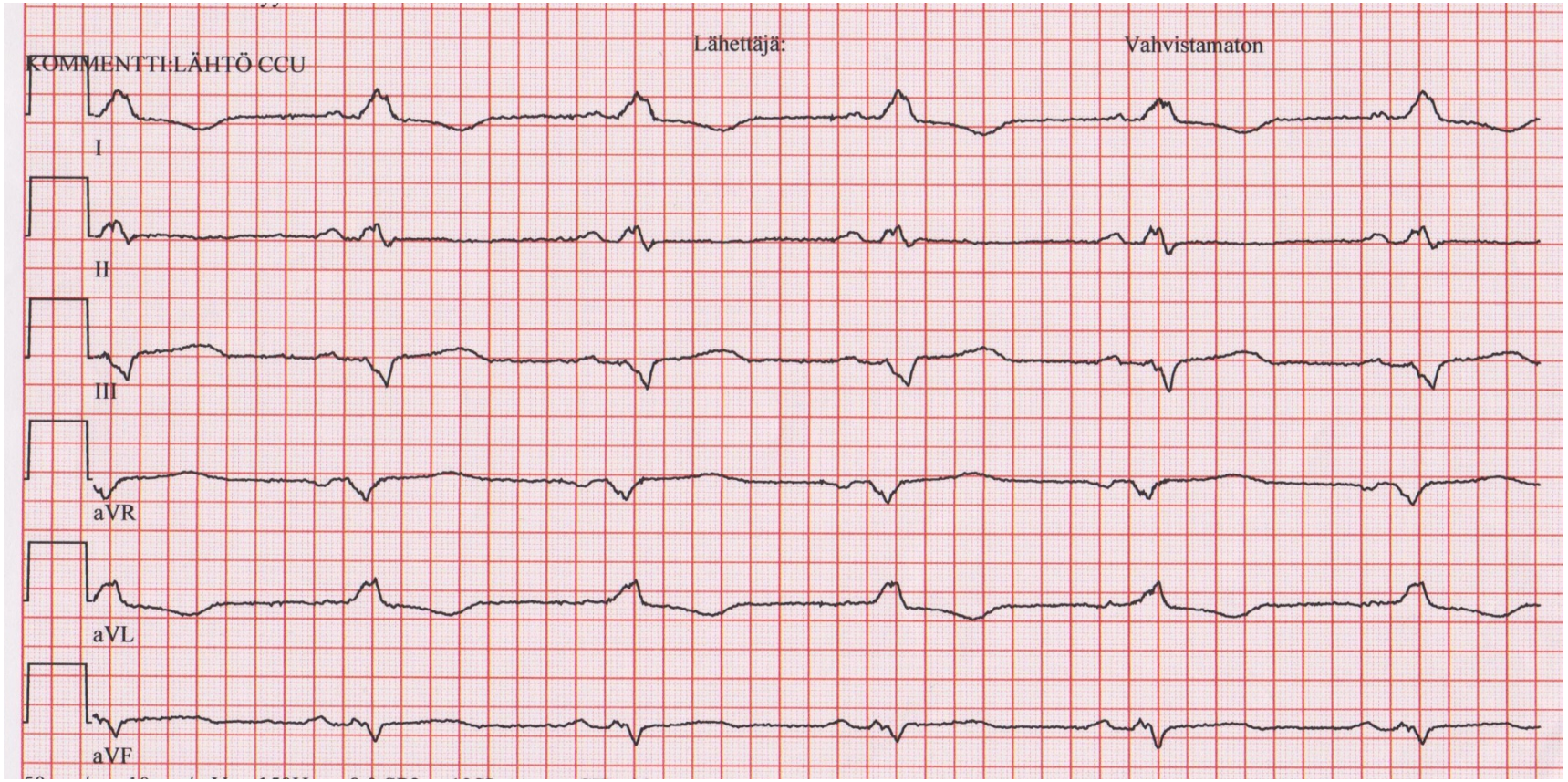
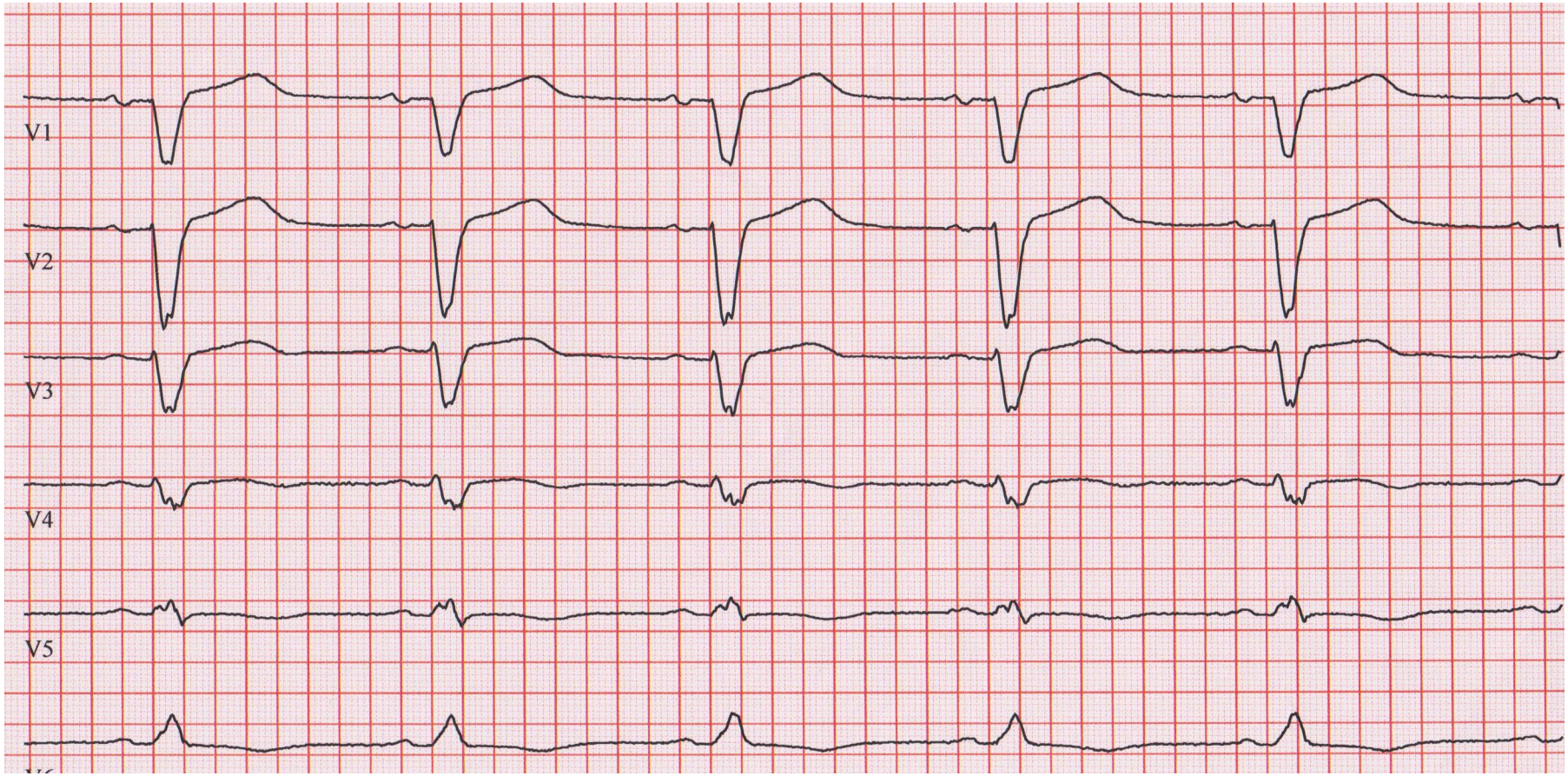

KOMMENTTI:LÄHTÖ CCU
Lähettäjä:
Vahvistamaton
I
II
III
aVR
aVL
aVF

09:31 a.m.



Wonder whether narrow complex rhythm is an idioventricular rhythm coming from basal septum. My comments were not included but thought this was VT from basal septum. The qR pattern in V1 suggests a site close to the Aorto mitral continuity. It is difficult to opine anything about mechanism with out some intervention. Adenosine would a simple start. Adenosine has no effect on reentrant rhythms but in good doses will suppress triggered activity. In terms of decreased BP loss of atrial support is one explanation but also changes in the ventricular activation sequence during VT may affect ventricular filling.

Professor Melvin M.Scheinman,
Department of Cardiac Electrophysiology, University of California San Francisco, San Francisco, California, USA.

Português
Caros colegas

Se o QRS é mais estreito numa arritmia em paciente com bloqueio de ramo esquerdo, a origem do ritmo é ventricular. Neste caso, a morfologia do QRS mais estreito é de bloqueio do ramo direito com imagem nítida de infarto antero-septal. Temos então taquicardia ventricular com frequência cardíaca coincidente com a do ritmo sinusal, morfologia de bloqueio de ramo direito e zona inativa anterior.

Saudações

Jose Claudio Kruse MD Brasil.

English

Dear colleagues

If the QRS is narrower in arrhythmia in patients with left bundle branch block, the source of the rhythm is ventricular. In this case, the narrower QRS morphology is right bundle branch block with clear anteroseptal myocardial infarction. Then we have a ventricular tachycardia with heart rate coincident with sinus rhythm, right bundle branch block morphology and anterior electricaly inactive zone.

Greetings

Jose Claudio Kruse MD Brazil

Spanish Estimado Andrés:

El primer ECG: escape de la unión de 66 latidos por minuto con imagen de BCRD y secuela de infarto anteroseptal. No se observan ondas P y no presenta BCRI.

Segundo ECG: ritmo sinusal (58 latidos por minuto) bloqueo atrioventricular de primer grado, eje eléctrico desviado a la izquierda y BCRI, con melladura de la onda R en DI, aVL, V5 y V6 (signo de Chapman y Pearce) y Melladura > 0.04 seg. de la parte ascendente de la onda S de V3 a V5. (Signo de Cabrera y Friedland) por probable secuela del infarto anteroseptal.

Mi interpretación es que a pesar de pequeñas variaciones de la FC se produce el fenómeno de ritmo de escape de la unión con conducción supranormal por lo que transitoriamente desaparece el BCRI. Por la mejora transitoria de conducción de la rama izquierda probablemente asociada a la isquemia miocárdica.

Porque no presenta onda P en el primer trazado no creo que sea un paro sinusal por isquemia, sino un bloqueo sinusal por la utilización de amiodarona.

Tengo un interrogante solamente, como interpretaron TV si conocían que la paciente presentaba un BCRI, porque la infusión de amiodarona y no de lidocaina en el contexto presentado?

Un cordial saludo

Martin Ibarrola MD Buenos Aires Argentina

English Dear Andres:

The first ECG: Junctional rhythm of 66 beats per minute with CRBBB pattern and anteroseptal MI (sequel.) No P waves are observed and no LBBB. No P wave are observed in the first ECG. I do not think it's a sinus arrest consequence of ischemia, but a sinus node blockage by the use of amiodarone.

Second ECG: sinus rhythm (58 bpm) first degree AV block, left axis deviation and LBBB, with notched on R wave in Left leads I, aVL, V5 and V6 (“Chapman and Pearce sign”) and notched > 0.04 sec on the ascending ramp of the S wave from V3 to V5. (“Cabrera and Friedland sign”) probably sequel of the anteroseptal MI

My interpretation is that despite minor variations HR escape junction phenomenon rhythm is consequence of supernormal conduction consequence of transient improvement of conduction on the left bundle branch probably due to myocardial ischemia, so temporarily disappears the LBBB

I have one question only,: what was the interpretation of colleagues in reference to VT if they knew that the patient had previously LBBB?

Why amiodarone infusion and not lidocaine with this presented scenario?

Warm regards

Martin Ibarrola M.D. Buenos Aires Argentina

Portuguese
Andrés Minha interpretação é a seguinte:
Primeiro ECG: Ritmo fascicular originado no ramo E, de substituição (ritmo de escape ventricular) por parada sinusal com FC em torno de 40 sendo o segundo o ECG de ritmo sinusal com SAE e BRE, bradicárdico, frequencia cardiaca em torno de 37 bpm que deve ser o padrão eletrocardiografico basal do paciente que ficou bradicárdico em decorrência do uso de amiodarona.

Claudio Pinho M.D. Campinas São Paulo Brasil

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English
Andrés My interpretation is as follows:
First ECG: fascicular subtitution rhyth originated in left bundle branch, (ventricular escape rhythm) consequence of sinus arrest with heart rate around 40 bpm
Second ECG: sinus bradycardic rhythm, (HR around 37 bpm) left atrial enlargement, and left bundle branch block, that should be the baseline electrocardiographic pattern of the patient who was bradycardic due to amiodarone.

Claudio Pinho M.D. Campinas São Paulo Brazil

Andrés, I will offer a comment but first to my colleagues on the Editorial Board of AGR – this is what Dr. Pérez-Riera does. He is sent these enticing cases and then distributes them to a list serve of physicians all over the world interested in this topic (ECG, VCG, Arrhythmias) soliciting input. A few days later he circulates his analysis. His analysis is comprised of three sections. First is the original unknown. Then the comments from any of the recipients of the original case. This is often published in both English and Portuguese. Finally his analysis which, in virtually every case, is worthy of being a chapter in a text book but commonly is also based on more information that was originally provided. As such the original case is an interesting intellectual exercise which sometimes frustrates me because I always want to take it to the next step, what should be done for the patient.

So my response: When in sinus rhythm, there is indeed the LBBB. We are told that the patient had an acute septal MI with recurrent VT. We are not told the rate of the VT or whether or not it required independent therapy (cardioversion) but the patient is now on an amiodarone infusion so I will presume that it required therapy and that it was rapid. We are not provided an ECG recorded during the earlier episode(s) of VT. The current, non-sinus rhythm is "fast" for a ventricular escape focus so it is probably an accelerated idioventricular rhythm arising from the LV (RBBB morphology) probably on the border of the new infarct that is virtually isorhythmic with the sinus and subtle changes in rate allow it to be expressed. If it was an identical morphology to the symptomatic VT that required therapy and an subset of the original VT rate before amiodarone was started, it might also be continuing VT with an exit block out of the VT focus. Depending on the patient's LV function, even though this rate is not fast, the loss of atrial transport may be hemodynamically compromising. Attached is an arterial pulse tracing from one of my patients who had a VT with 2:1 exit block but the VT rate with the exit block was virtually identical to the sinus rate yet one can see the marked drop in systolic pressure, the pulse pressure and the LV ejection time associated with the loss of atrial transport. This is something that I and others have called Pseudo-pacemaker Syndrome. It is behaving like pacemaker syndrome with the loss of atrial transport as would occur with a VVI pacemaker at a normal rate but in a patient who doesn't have a pacemaker. What to do about this rhythm? if this was very early in the course of the MI, it might quiet down and not require any specific therapy. If it was really the continuing VT with exit block due to the amiodarone and it persists, one has to determine how frequently it is occurring. One could consider VT mapping and ablate the ectopic focus. Another consideration would be to just continue the amiodarone while a third, unless it was occurring very frequently, would be to stop the amiodarone and implantation of an ICD. If it was occurring very frequently, amiodarone would still be necessary but not an ICD unless there was also break-through episodes at a rapid rate. I look forward to the final interpretation and any other clinical information that might be provided.

Paul A. Levine MD, FHRS, FACC, CCDS
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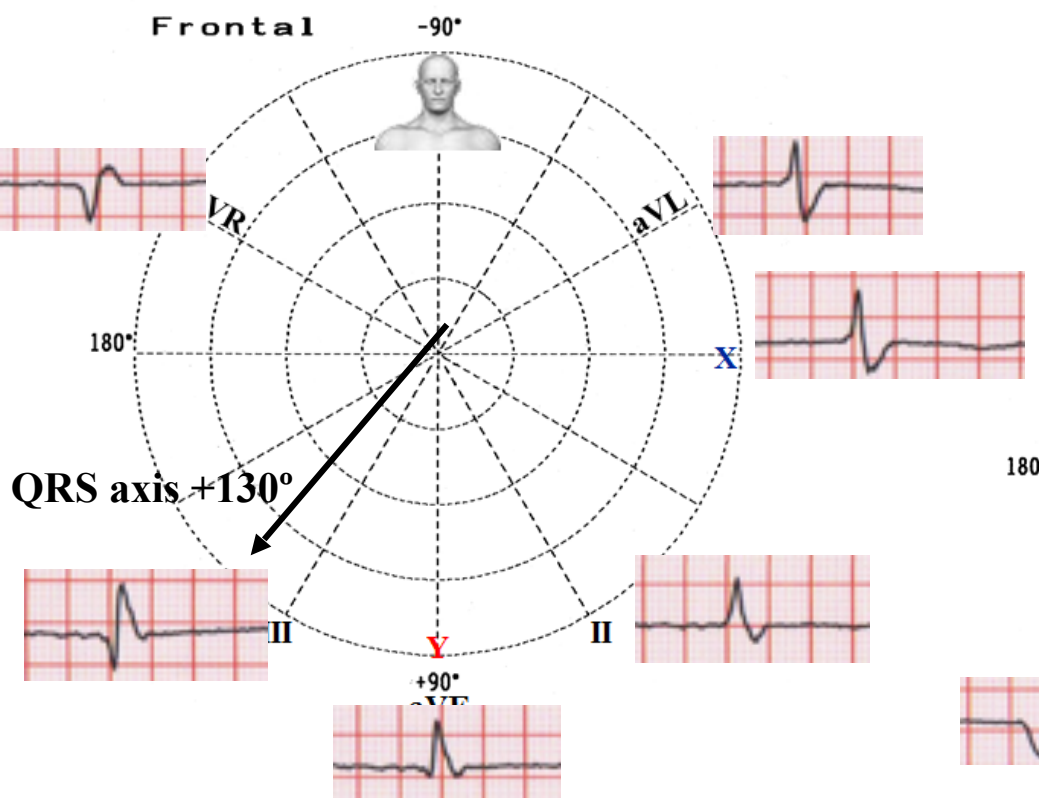
Email: paul91321@gmail.com

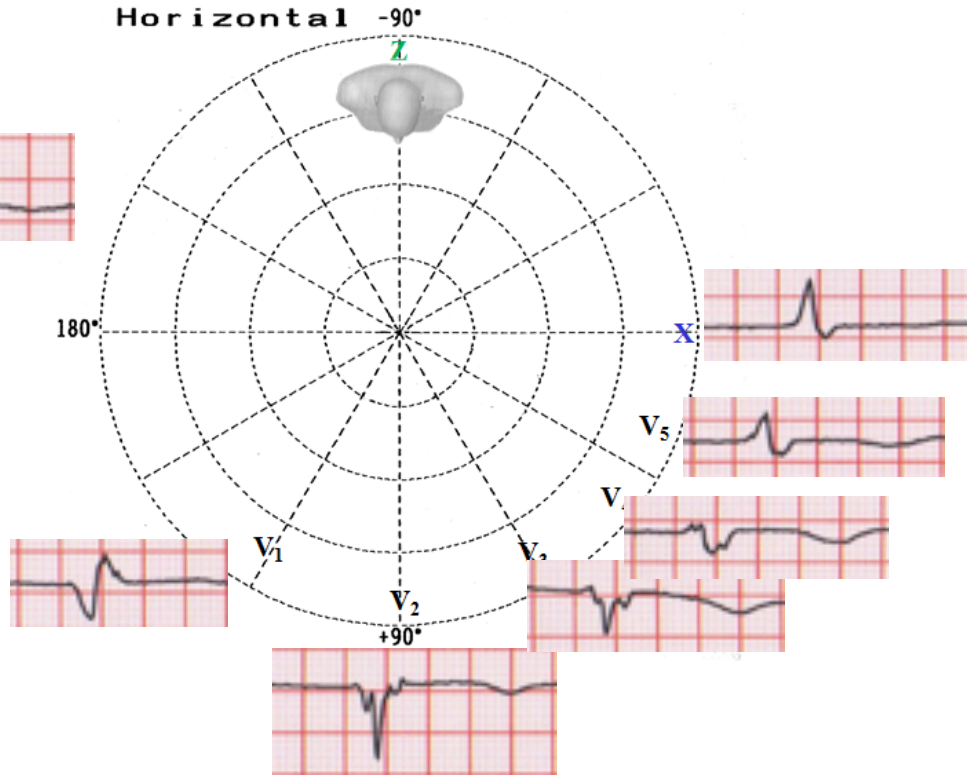
I agree with Dr. Levine's interpretation. Firstly, the paper speed should be mentioned. It would be better to have a 25mm/sec recording. The escape focus of the IVR is likely closer to the left bundle, hence reducing the QRS width. The teaching point here would be that "When there is a wide QRS in sinus rhythm, a less wide QRS in tachycardia or any other rhythm has to be ventricular in origin". This was a classic concept I had learnt from Professor Wellens.

Yash Lokhandwala – Mumbai/India

Final comments

ECG-1 (09:30 a.m.): HR = 69 bpm (paper speed = 50 mm/s); QRS duration wide = 124 ms; prolonged QTc = 482 ms; P wave is not identifiable; sustained broad QRS with CRBBB:. Sustained accelerated idioventricular rhythm (AIVR) or slow VT with isorhythmic AV dissociation. This exists when the atrium and ventricles are beating regularly but doing their own thing, just at a very similar rate. Isorhythmic AV dissociation. AV dissociation usually refers to the situation in which the ventricular rate is the same or faster than the atrial rate. When the atrial rate is the same as the ventricular rate but the P wave is not conducting, the rhythm disturbance is known as isorhythmic AV dissociation. When the rates are similar but occasionally the atria conduct to the ventricles, the rhythm is known as interference AV dissociation.





AV dissociation can be a benign phenomenon and can be complete or incomplete. When incomplete, some of the P waves conduct and capture the ventricles (ie, interference AV dissociation), but if they do not, it is complete AV dissociation.
Complete AV dissociation can mimic AV block, but the fact that none of the P waves conduct has more to do with timing of the P waves in relation to the QRS complex rather than the presence of AV block.
A normal cardiac impulse arises from the sinus node and is conducted through the AV junction, the bundle of His, and the bundle branches to the ventricles. The sinus node is the dominant pacemaker because its intrinsic rate is faster than subsidiary pacemakers in the AV junction or in the ventricle. AV dissociation can result from slowing of the dominant pacemaker (sinus node), which allows an escape junctional or ventricular rhythm, or acceleration of a normally slower (subsidiary) pacemaker, such as a junctional site or a ventricular site that activates the ventricles without retrograde atrial capture.

Conditions that can initiate AV dissociation include:
1. Surgical and anesthesia interventions (including intubation)
2.Conditions that increase catecholamine levels (including infusions of inotropes)
3.Drugs that block catecholamines,
4.Sinus node disease,
5.Digoxin toxicity,
6. Myocardial infarction and other structural heart disease,
7.Hyperkalemia,
8.Vagal activation (eg, neurocardiogenic syncope, vomiting),
9.Ventricular tachycardia, or ventricular pacing.
10.Av dissociation can be seen after radiofrequency ablation of the slow pathway responsible for av nodal reentry if some of the vagal fibers are damaged.
11.After exertion, if AV dissociation is present from an escape pacer, it can be a normal phenomenon. Whatever the cause, AV dissociation usually is secondary to some other cause.

In isorhythmic dissociation, the relationship between the P waves and QRS complexes appears to fall into two distinct patterns.

The first type of pattern is characterized by a rhythmic fluctuation of the interval between the P and QRS waves, most often with the P oscillating gradually back and forth across the QRS; that is, with periodically varying P-R and R-P intervals.

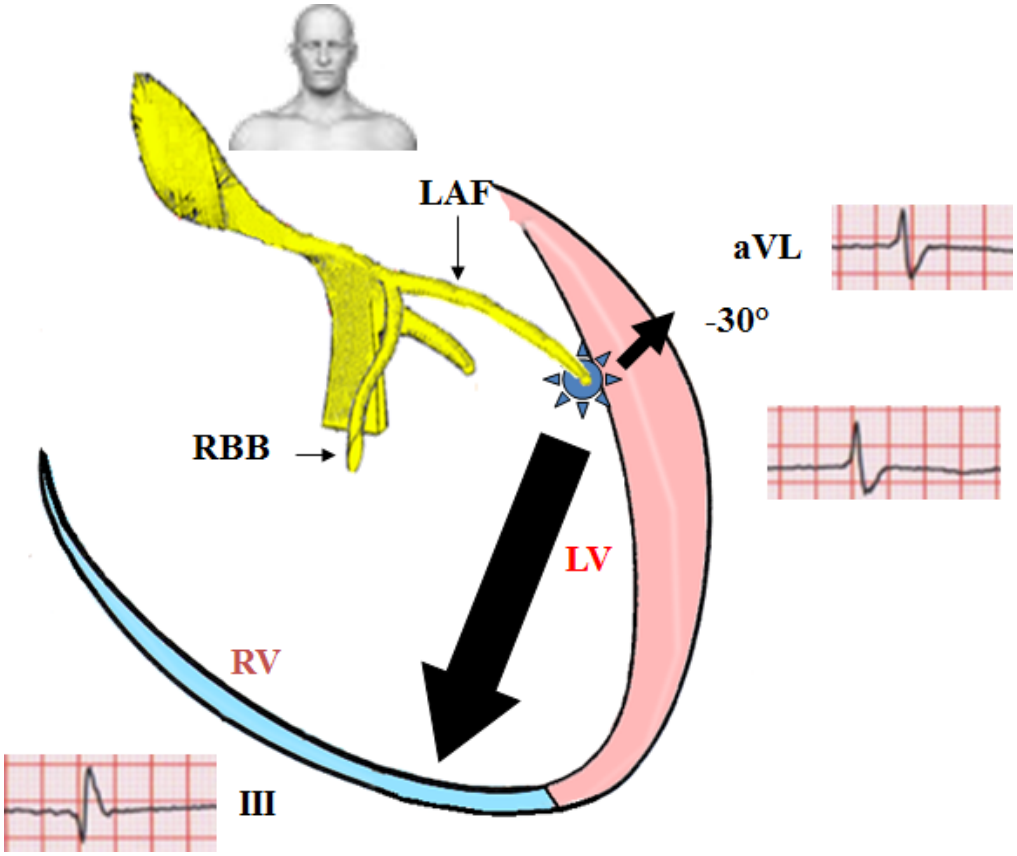
In the second type of electrocardiographic pattern, the P-R or R-P interval did not undergo rhythmic fluctuations, but the P and R waves were in a relatively fixed position with respect to each other. This pattern, has been reported frequently (1;2;3).
It did not appear during synchronization in the experimental animal with complete heart block.

1.Schott A: Atrioventricular dissociation with and without interference. Progr Cardiovasc Dis 2: 444, 1959 16.

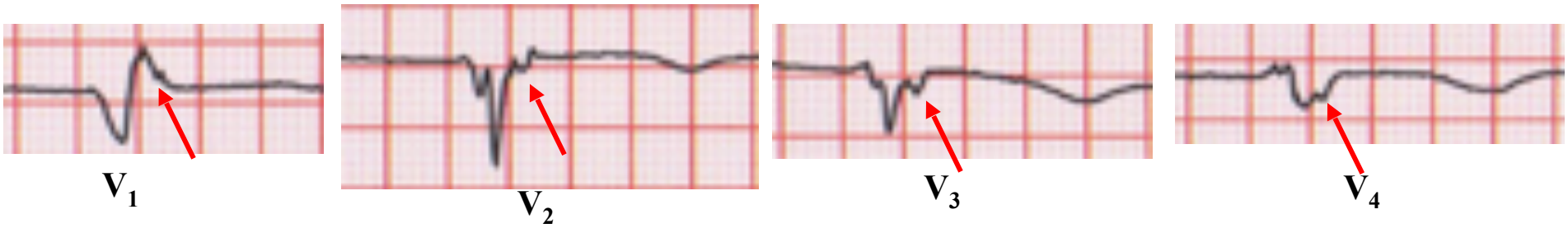
2.Marriott HJL: Interactions between atria and ventricles during interference dissociation and complete A-V block. Amer Heart J 53: 884, 1957

3.Waldo AL, et al: The mechanism of synchronization in isorhythmic A-V dissociation. Circulation 38: 880, 1968

Where is the focus?



PVCs or VTs that originate in the left ventricle. They show CRBBB pattern and duration, with QRS electrical axis with extreme superior shift of the LAFB type or with inferior shift of the LPFB type, depending on whether they originate in the posteroinferior wall or anterosuperior wall respectively. In brief, if the PVC originates in the posteroinferior region of the left ventricle, it will have a CRBBB pattern associated to LAFB, with QRS axis close to -60º, Q waves in I and aVL, V4-V6, and QRS >130 ms. If the PVC originates in the anterosuperior region of the left ventricle, it will have a CRBBB pattern associated to LPFB, with axis in +120º, rS in I and aVL, and QRS duration >130 ms. A third form is the pure CRBBB pattern. In this case the PVC is born in the free wall of the LV or the septum in equidistant points of the septal or the free wall territories of the Purkinje network. Finally, a pattern of CRBBB is possible, with minimal degrees of LAFB or LPFB.



Complete RBBB pattern associated with transmural anterior myocardial infarction.
Red arrows show wide QRS fragmentation (W-f-QRS)

There are several situations that may cause the qR/QR pattern in V1 as follows:

➢Systolic right ventricular hypertrophy (RVH) with strain pattern of repolarization: indicating the presence of supra-systemic right intraventricular pressure;

➢Large increase in right atrium (RA) volume; e.g.: Ebstein's anomaly and severe tricuspid regurgitation or long-standing. The great volumetric increase of the RA gets it closer to the exploring electrode of V_1, registering the initial negative intracavitary potential of the RA. The qR pattern in V1 or V1-V2 (or qRs, QR and Qr) is known in electrocardiology as Sodi-Pallares sign (5) indicative of significant RA dilatation. It is considered an indirect sign of right atrial enlargement;

➢RBBB associated with anterior myocardial infarction. This is the diagnosis in the present case;

➢RBBB with isoelectric initial r wave in V1. Sometimes the initial q wave in this lead can be attributed to the fact that the initial 10 ms vector is located on negative hemifield of its lead;

➢Increasing degrees of clockwise rotation of a vertical heart around its longitudinal axis (6) (in this case the patient does not have a vertical heart);

➢Initial negativity of the right septal surface may be due to a decrease in certain areas of the density of the union between Purkinje arborizations and muscle fibers as the result of dilatation of the ventricle (7);

➢Situs inversus (ventricular inversion with right-to-left septal depolarization) the typical example of which is the congenital corrected transposition of the great arteries;

➢Pectus excavatum;

➢CRBBB associated with LSFB.

ECG characterization of Accelerated Idioventricular Rhythm(AIVR) or slow VT

➢ Duration of QRS complex: ≥ 120 ms;

➢Constant and bizarre morphology of QRS complexes (monomorphic);

➢Slow rate: between 50 bpm and 130 bpm (usually between 70 and 85 bpm);

➢Regular or almost regular R-R;

➢Event SÂQRS different from basic rhythm SÂQRS;

➢Onset and end of event, gradual and non paroxysmal. The former, marked by delayed or telediastolic premature ventricular contraction (initial beat with prolonged coupling) or with idioventricular escape if the basic rhythm was very slow; the end occurs by acceleration of sinus rhythm or by slowing of tachycardiac rhythm;

➢Depressed sinoatrial activity, with frequent absence of P wave;

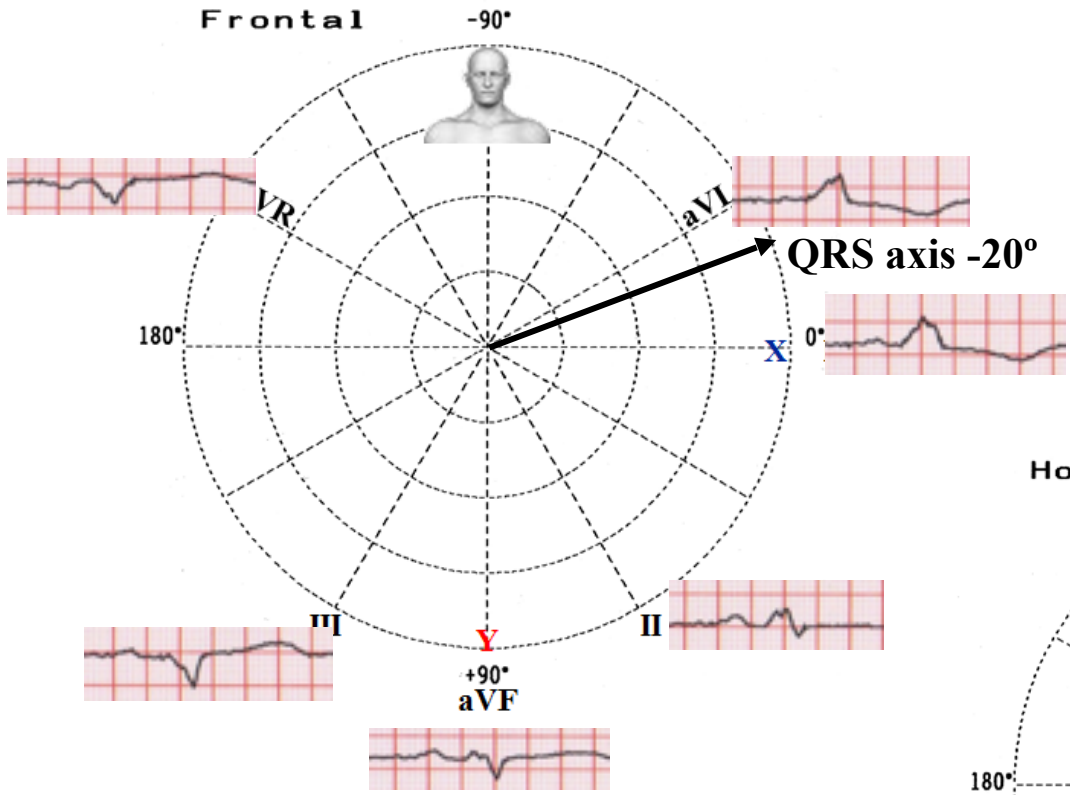
➢AV dissociation: 70% of the cases;

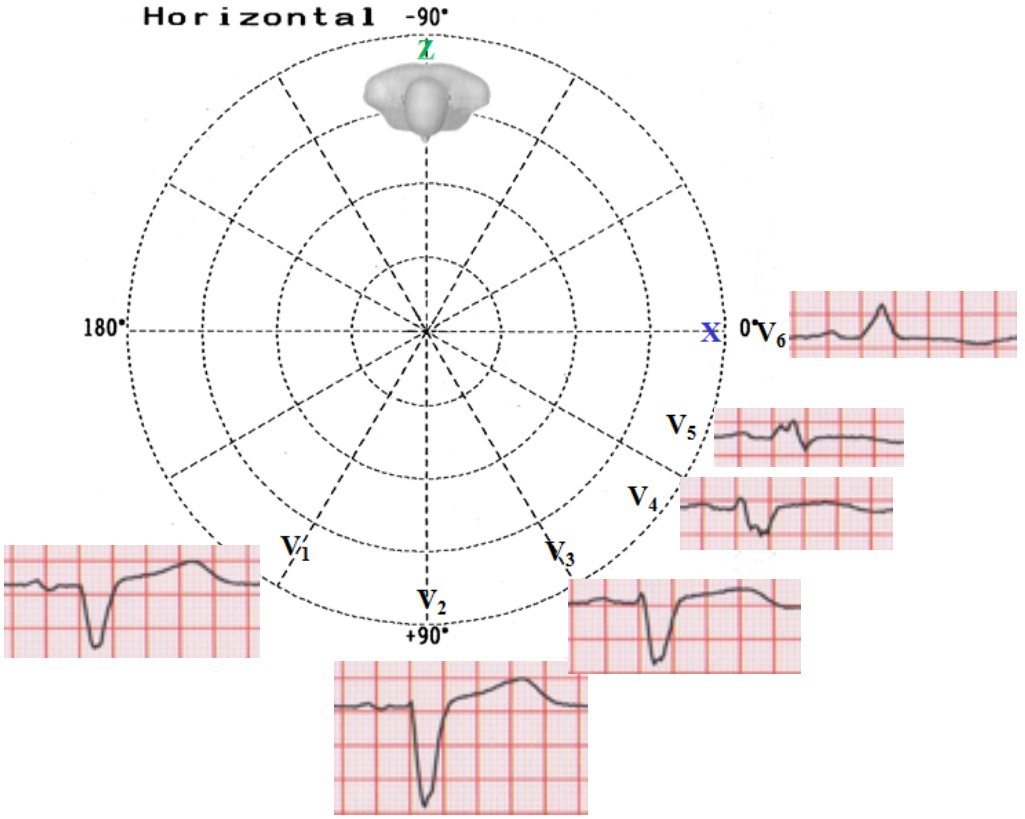
➢Frequent fusion beats at the onset and the end of the event;

➢Capture and fusion beats, much more frequent than in paroxysmal VT;

➢Frequent coexistence with extra systolic VT in its unstable form.

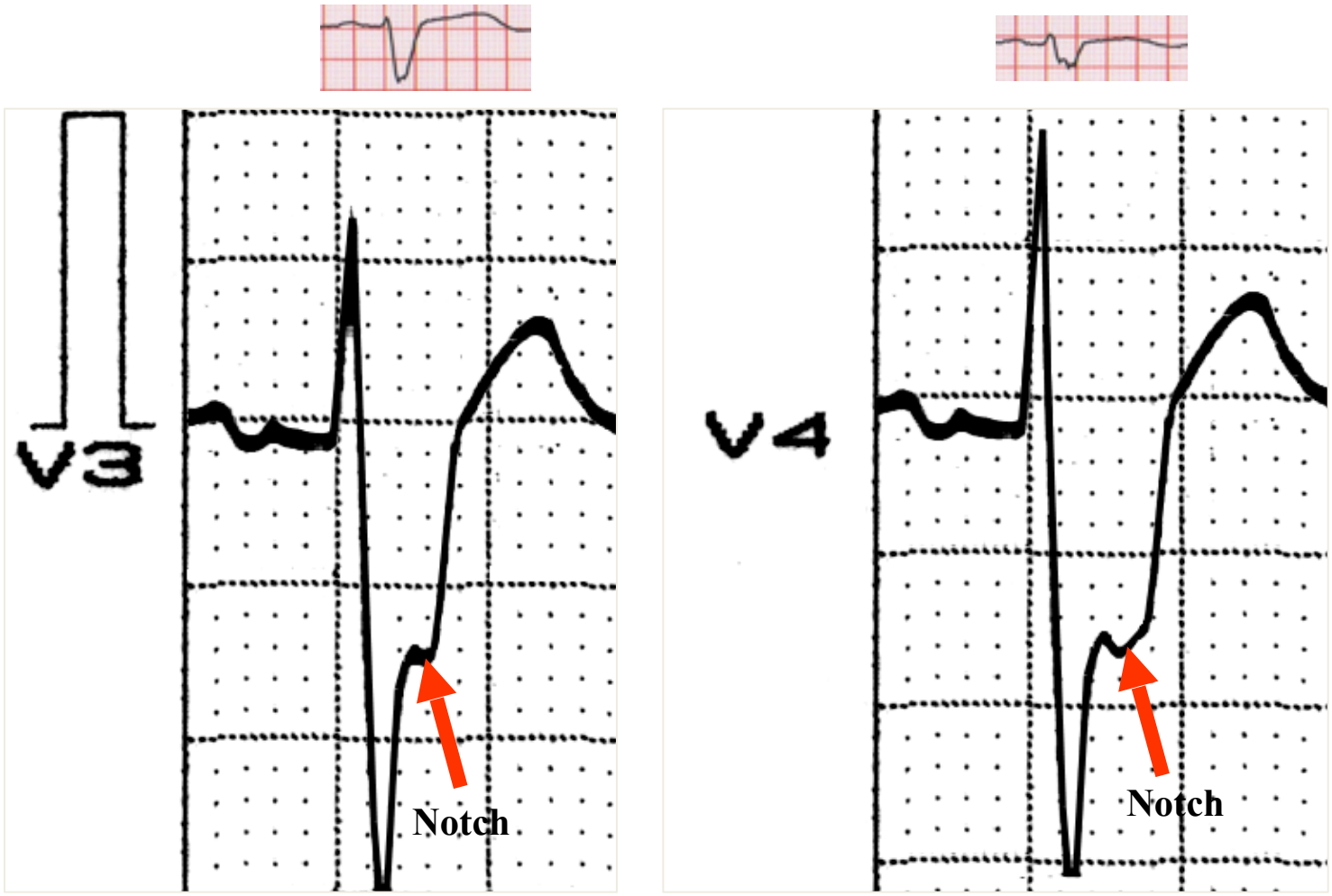
Sinus rhythm LBBB + anterolateral MI + low QRS voltage





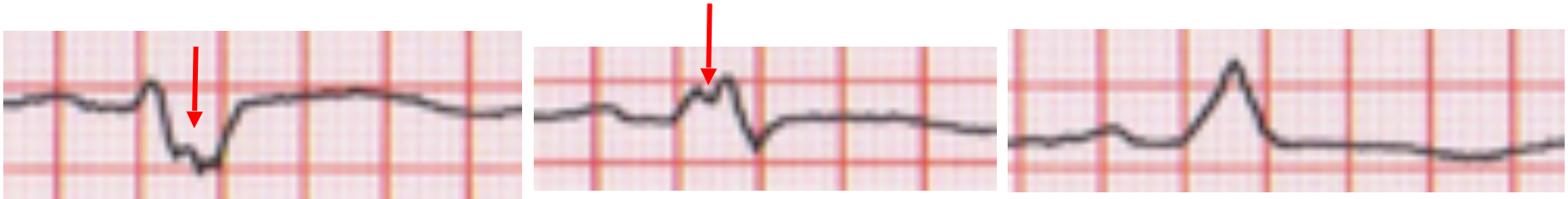
Low QRS voltage on FP, because no wave exceeds 5 mm (1 large square or 5 small squares, vertically).Causes of low voltage include extensive or large MI, cardiomyopathies, myxedema, amyloidosis, obesity, anasarca, pleural or pericardial effusion, hemochromatosis, heart failure, emphysema, hypothermia and other miscellaneous causes.

Cabrera's sign: LBBB complicated with anterior infarction



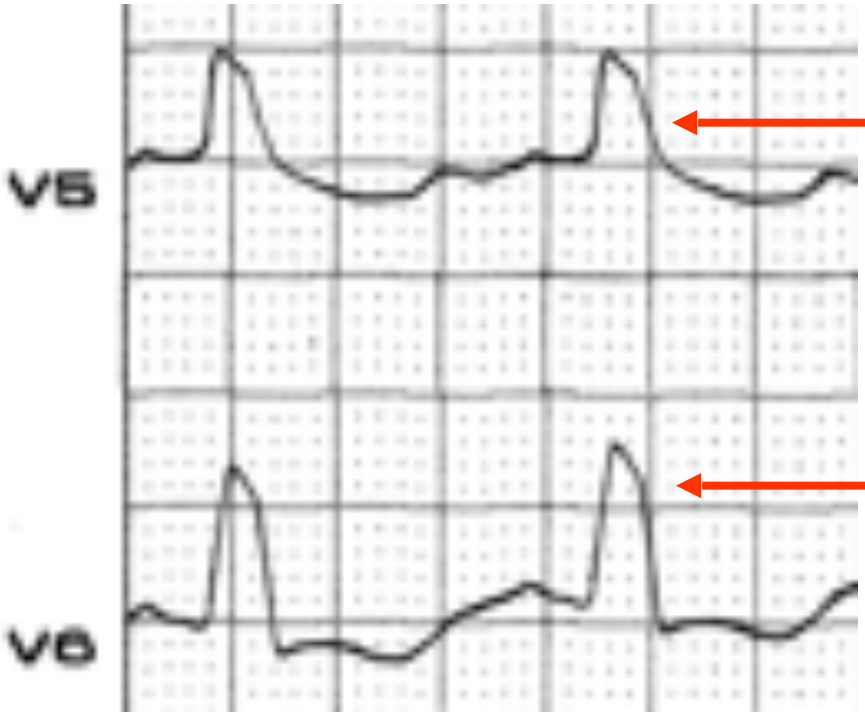
Notch of 50 ms in the ascending ramp of S wave of V_3 and V_4. It is seen more often with MI than without (anterior more often than inferior), and the left axis increases its sensitivity (Kinwall 1986; Cabrera 1953).

The presente case wide fragmented QRS (red arrow)

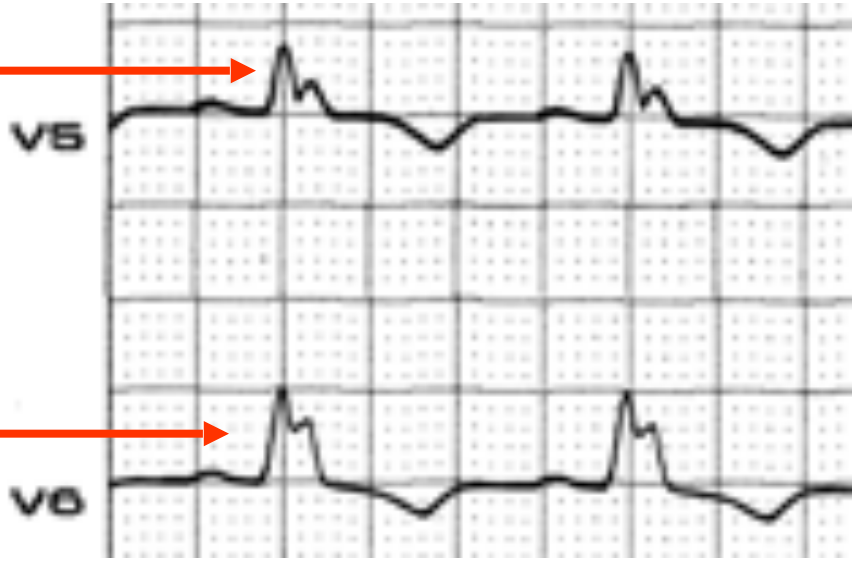


V_4 V_5 V_6

Decrease of voltage of R wave from V4 to V6, indicating additional involvement in LV free wall, associated with LBBB.



Uncomplicated LBBB



Complicated LBBB with free wall MI