46 yo man admitted with inferior STEMI (ECG1). No prior cardiac history.
ECG2 is obtained after revascularization.
Patient was asymptomatic and rhythm self terminated.
It look like bidirectional VT/idioventricular rhythm, yes?
No digoxin administration. No history of syncope or SCD.
I saw a few case reports about this in the setting of an inferior MI. Have you ever seen or heard about this?

Hombre de 46 años admitido con cuadro de infarto inferior con elevación del segmento ST “STEMI” inferior (ECG1). No hay historia previa de problema cardiaco.
ECG2 se obtiene después de la revascularización.
El paciente se encuentra asintomático y la arritmia terminó espontaneamente.
Parece una TV bidireccional o RIVA?
No huvo administración de digoxina. No tiene antecedentes de síncope o MCS.
Vi algunos informes de casos sobre esto en el contexto de un infarto de miocardio inferior. ¿Alguna vez has visto u oído hablar de esto?

Thomas A. Dewland, MD
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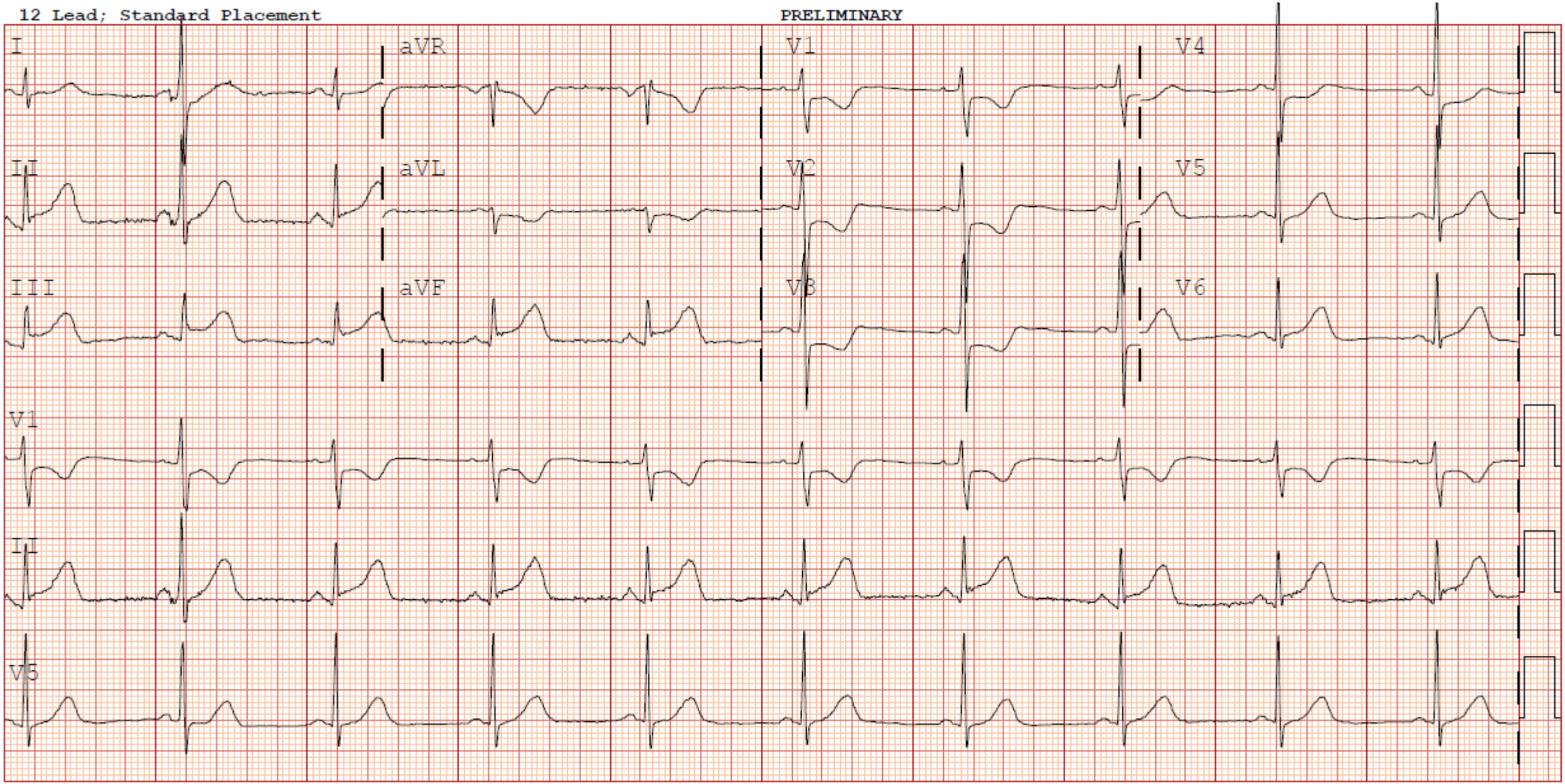
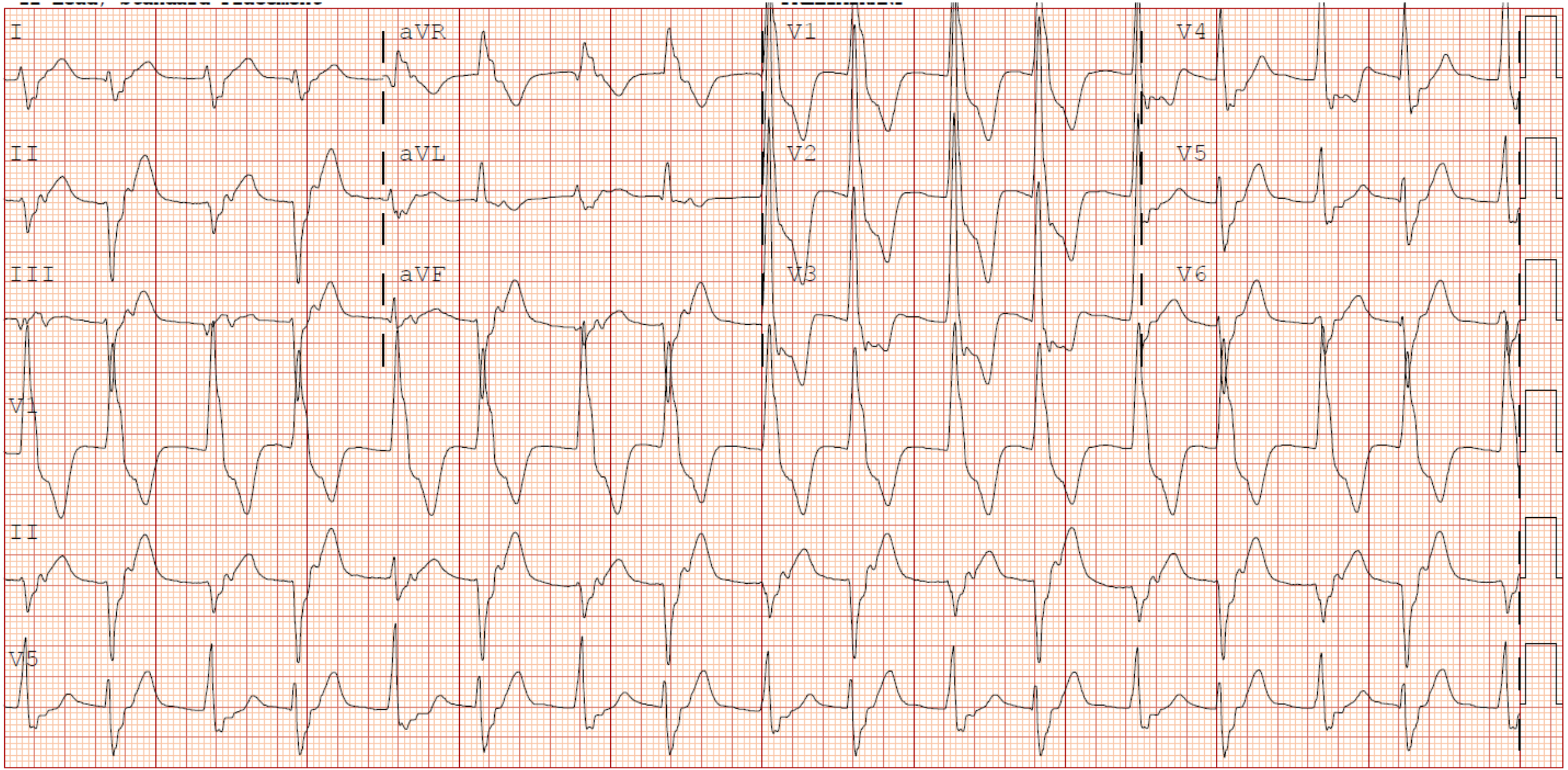
ECG1


12 Lead; Standard Placement
PRELIMINARY
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

ECG2



Thanks for the tracings. It is not a classic bidirectional VT since you do not have the classic 180 degree shift in the frontal plane axis. I believe you are dealing with VT from near the inferior apex due to the inferior posterior MI with QRS shifts due to cycle length alternations i.e. Short cycles show a greater degree of QRS widening. The long short sequences may be related to focal discharge with 3:2 Wenkebach exit either from abnormal automaticity or micro reentry. Will send to the master for his interpretation.

Gracias por los trazados. No es un TV bidireccional clásica ya que no tiene la clásica mudanza del eje de 180 grados en el plano frontal. Creo que se trata de una TV con foco localizado cerca de la punta debido a IM posterior inferior con QRS desviado por alteraciones en la longitud de los ciclos, es decir, los ciclos cortos tienen un QRS más anchos. Los ciclos longos pueden estar relacionados con la descarga focal con 3: 2 Wenkebach salida ya sea por la automaticidad anormal o micro reentrada. Enviaré al maestro para su interpretación.

Melvin M. Scheinman
University of California San Francisco, CA, USA.

Dear friend:

I do agree with Mel. This is not a "bidirectional VT" but merely an irregular accelerated IVR having 2 alternating QRS morphologies, both suggesting some "reperfusion" and both originated from / close to the infarcted area.
I did see some similar traces.
Best regards

Professor Bernard Belhassen
Cardiologist, Educator, Tel-Aviv, Israel

Estimado Potro: El paciente presenta criterios de TV en el contexto de un IAM. Considerando que se ha reperfundido la actividad eléctrica heterogénea del miocardio previamente isquémico brinda el sustrato electrofisiológico de las arritmias por reentrada (macro y microreentradas) y otros mecanismos, como el aumento del automatismo. Estas TVs transitorias en especial el RIVA.
No encuentro criterios para TV bidireccional, sino que presenta 2 intervalos de ciclos de la TV que solo desvían levemente el eje de la TV durante la misma. Alterandose ciclo corto-ciclo largo, dando la falsa impresión de una TV bidireccional. Los ciclos cortos evidencian una conducción mas prolongada desviando el eje eléctrico de la TV más a la izquierda, por el contrario los ciclos prolongados presentan mejora de la conducción con disminución en la duración del QRS y menor desviación de la TV a la izquierda. Porque la TV presenta diferentes longitudes de ciclo? pienso justamente tratándose de un RIVA el mecanismo de reentrada es la causa del mismo, en este caso macroeentrada de un foco automático reperfundido. Con una TV con dos longitudes de ciclo y de acuerdo a esto las diferencias en la conducción y leve desviación del eje eléctrico.
Un cordial saludo
Martin Ibarrola

Dear Andrés: The patient presents criteria of VT in an AMI scenario. Considering that the patient previously reperfused this may explain the mechanism or electrophysiological substrate for macro and/or micro reentry and other mechanisms, such as increasing automatism. These VTs are transient features in the accelerated idioventricular rhythm (AIVR) or slow VT. During short cycles the conduction is more prolonged shifting the electrical axis of the TV to the left, on the other hand, during long cycles the conduction improves, consequently the QRS duration is lesser and the QRS axis has less deviation.
I see no criteria for bidirectional TV, but presents two cycle intervals TV only slightly deviate the axis of the VT during it. Short-cycle alterning long cycle, giving the false impression of a bidirectional TV.
Why does the VT have different cycle lengths? Answer: because in AIVR VT the reentry mechanism is the cause thereof, in this case macroeentry of a reperfused automatic focus. With a VT with two cycle lengths and accordingly differences in conduction explain the slight deviation of the QRS axis.
Warm regards
Martin Ibarrola MD Argentina

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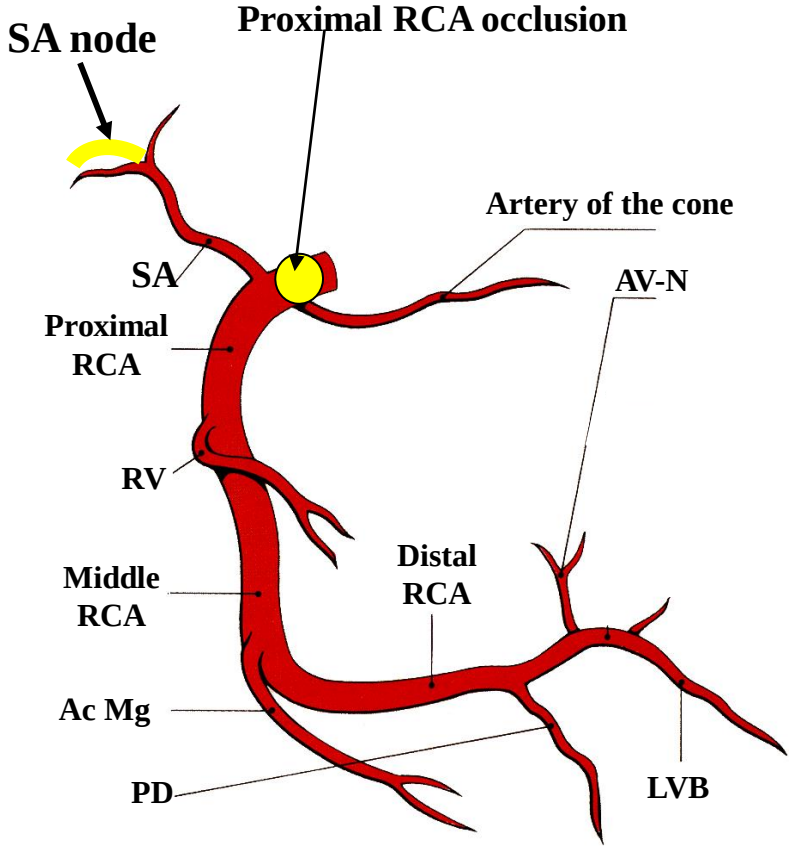
Me parece una TV lenta, no la veo como siendo ina TV bidireccional tampoco.
Lo que no puedo imaginarme es un mecanismo de Wenckebach en su explicación.
La primera parte del QRS es rápida, luego lenta la activación hacia la derecha como si surgiera de la hemirama anterior izquierda, con imagen de HBPI y BRD, con alternancia en el ciclo y en la aberrancia derecha, con salida alternante por 2 sitios? desde el foco (automático o microreentrada?)
También espero la interpretación de algún maestro!
José Luis Serra MD Córdoba Argentina

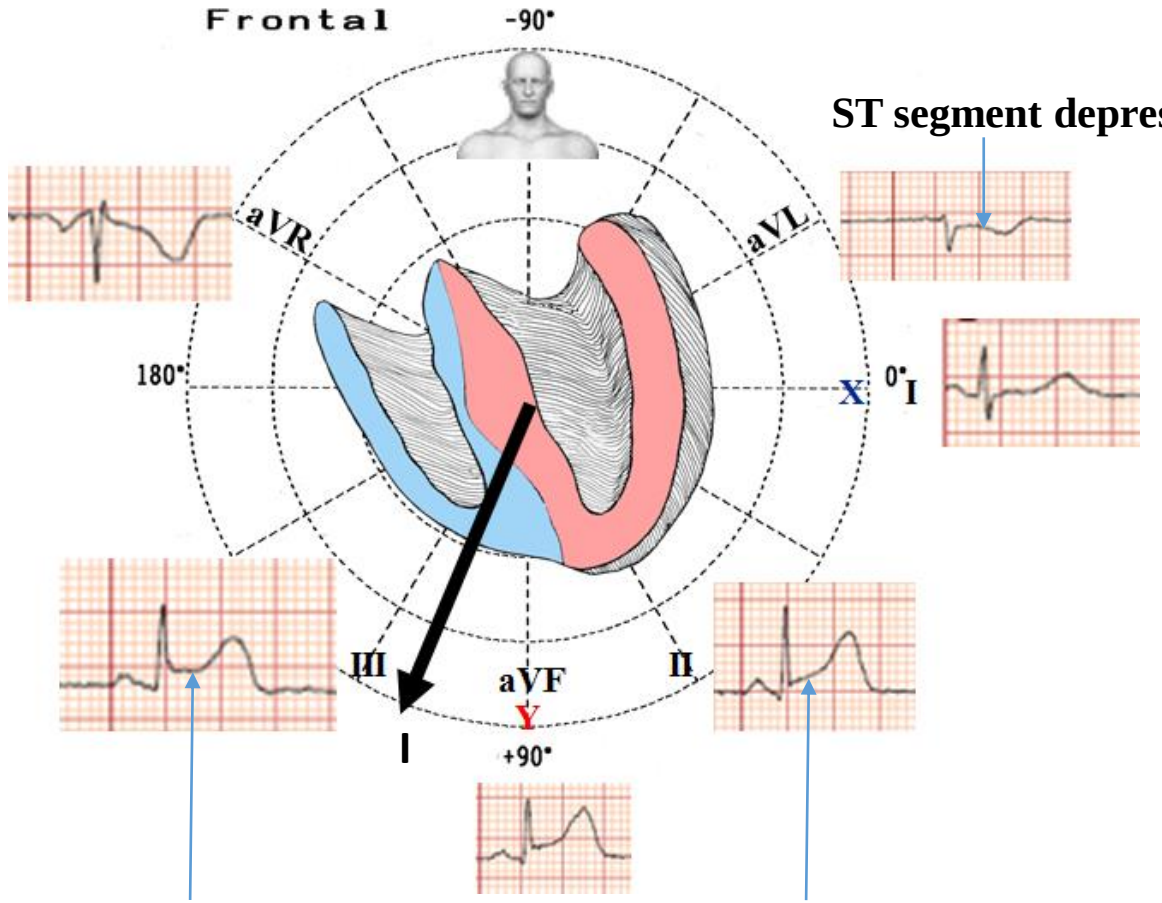
It seems an slow VT, I do not think that this is a bidiretional VT.
I can not imagine the explanation by Wenckebach mechanism.
The first part of the QRS complexes has fast inscription and the final one slow. The QRS complex has LAFB associated with RBBB pattern with alternant cycle: Does it occur alternate output by two focus? Automatism? or microrrentry?
I also hope the interpretation of some expert

Final comments by Andrés Ricardo Pérez-Riera M.D.Ph.D.

ECG-1 frontal plane: Proximal RCA occlusion pattern STEMI



RCA: Right Coronary Artery
SA: Artery of the Sino-Atrial Node
RV: Branch of the Right Ventricle
Ac Mg: Acute Marginal
PD: Posterior Descending
AV-N: Artery of the Atrio-Ventricular Node
LVB: Left Ventricular Branch

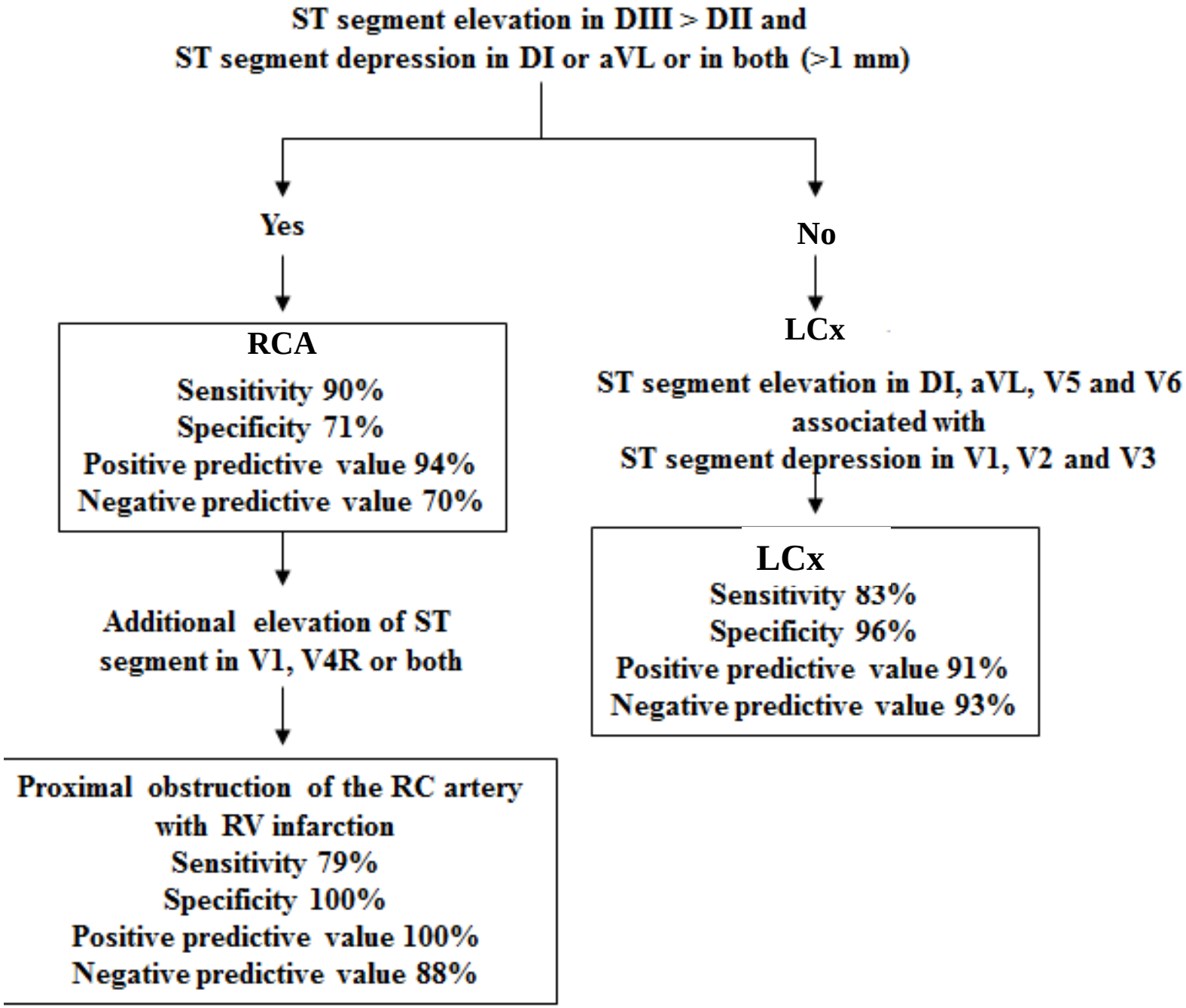


ST segment depression in aVL >1 mm)

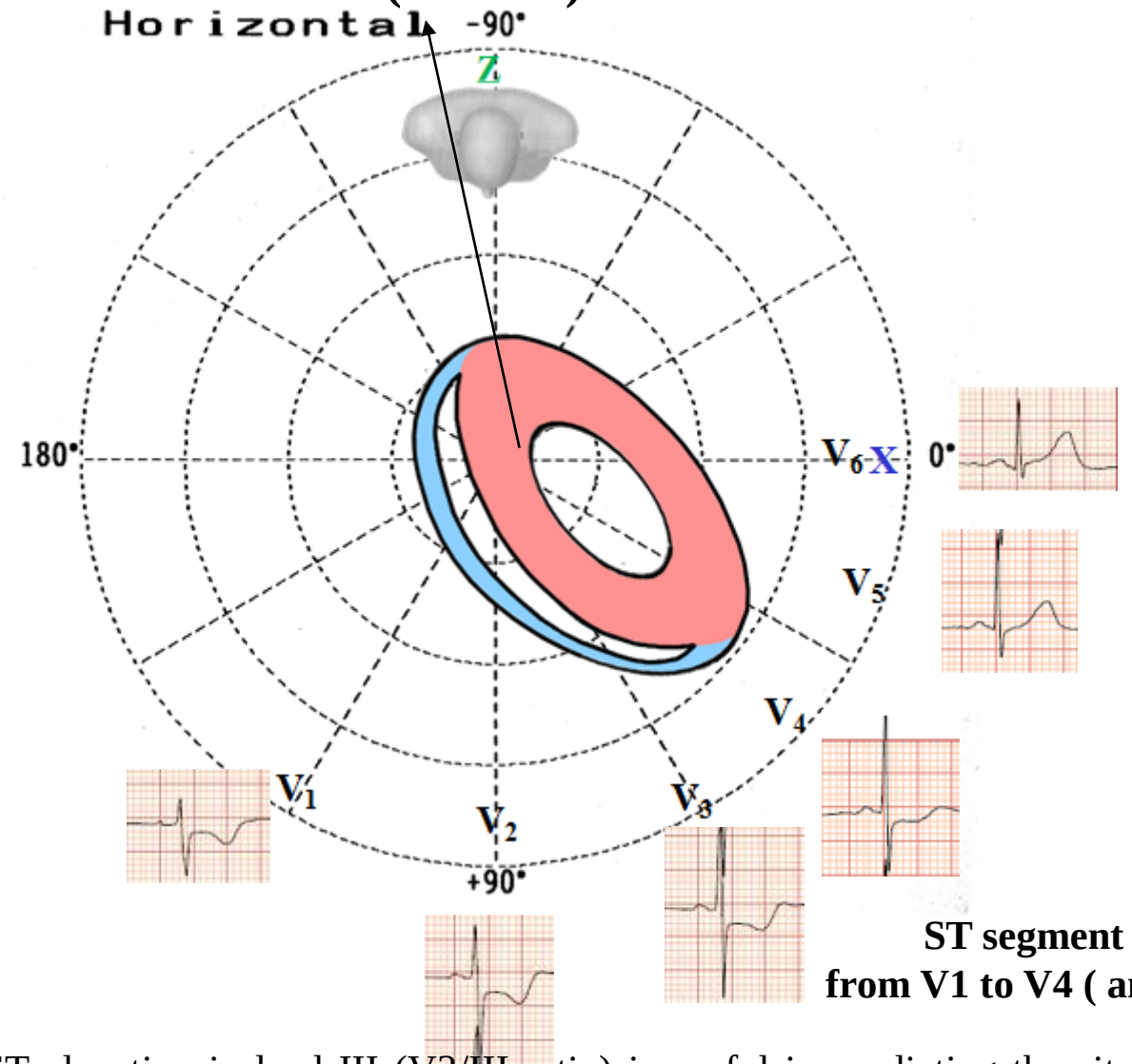
ST segment elevation in inferior leads SIII > SII

I The ST injury vector directed to downward and to right.

Algorithm to identify through ECG the artery involved in Acute Inferior Myocardial Infarction with STSE



ECG-1 Horizontal plane: Proximal RCA occlusion pattern ST-segment elevation myocardial infarction (STEMI)



ST segment depression from V1 to V4 (anteroseptal wall)

The ratio of ST depression in lead V3 to ST elevation in lead III (V3/III ratio) is useful in predicting the site of coronary artery occlusion in patients with inferior wall AMI. The V3/III ratio < 0.5 identified proximal RCA occlusion, 0.5 < V3/III ratio ≤ 1.2 identified distal RCA occlusion, and 1.2 < V3/III ratio identified LCx occlusion with sensitivities of 91%, 84%, and 84%, and specificities of 91%, 93%, and 95%, respectively. (Kosuge 1998)

The second ECG-2 is a clear accelerated idioventricular rhythm (AIVR)

Synonymous: Non-paroxysmal VT, slow ventricular tachycardia (Leitz 2008), ventricular rhythm with isorrhythm, benevolent rhythm (Martinez-Lopez 1993), non-paroxysmal VT or slow ventricular tachycardia.

In the present case the HR is between 94 to 107 bpm.

Most AIVRs originate from a single focus. Occasionally, in patients with acute myocardial ischemia and myocarditis, AIVR can originate from multiple focci (Sclarovski 1983; Nakayama 1988). The ventricular rate of AIVR is generally between 40 to 100-120 bpm.

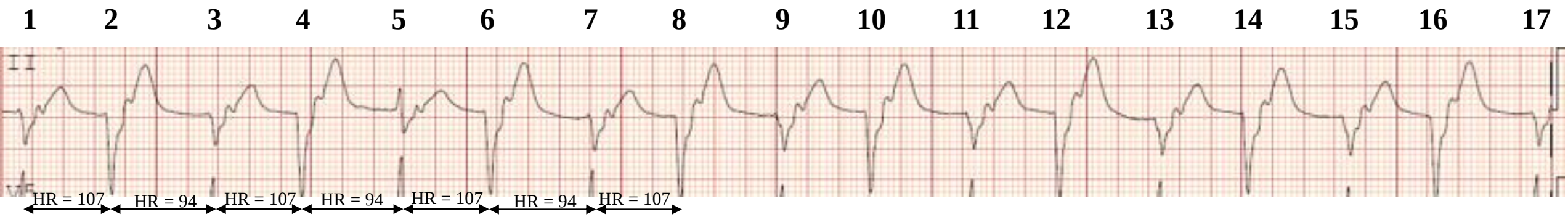
Semantic discussion: The term tachycardia implies the existence of a rhythm with a natural rate above what is considered to be normal for sinus rhythm; i.e. greater than 100 bpm for adults; therefore, the majority of the improperly called slow VT, with rates between 50 and 130 bpm, would be left out of this concept.

Significance of AIVR in AMI scenario

Post-reperfusion during coronary thrombolysis in the restoration of the anterograde coronary flow, which indicates reperfusion. AIVR is present in 90% of the cases in the first 24 hs. The incidence of AIRV is six times greater in the patients with reperfusion confirmed by 90-minute angiography after chemical thrombolysis. There is still no consensus about whether the AIRV constitutes a marker for myocardial reperfusion, since there are papers that show absence of significant difference between reperfused and non-reperfused patients. The value of the presence of AIVR as a marker of reperfusion is small, but in combination with other non-invasive markers (ST-segment resolution), its presence is connected with a high probability of successful reperfusion. Early ventricular arrhythmias are a serious complication of MI. However, if they are revealed and treated in time, they apparently do not represent a negative prognostic factor (Osmancik 2008). AIRV is an nonspecific marker for reperfusion of the infarct-related artery in AMI and thus, predate previous observations of the thrombolytic era. Even though, AIRV was associated with higher tonic vagal tone and lower sympathetic activity, the occurrence of AIRV had no prognostic impact on the clinical course and was not able to discriminate between complete and incomplete reperfusion (Bonnemeier 2005). Ever since the beginning of the thrombolytic era, the occurrence of AIVR in patients with acute MI has been considered a specific marker of successful reperfusion following the infusion of the lytic agents.

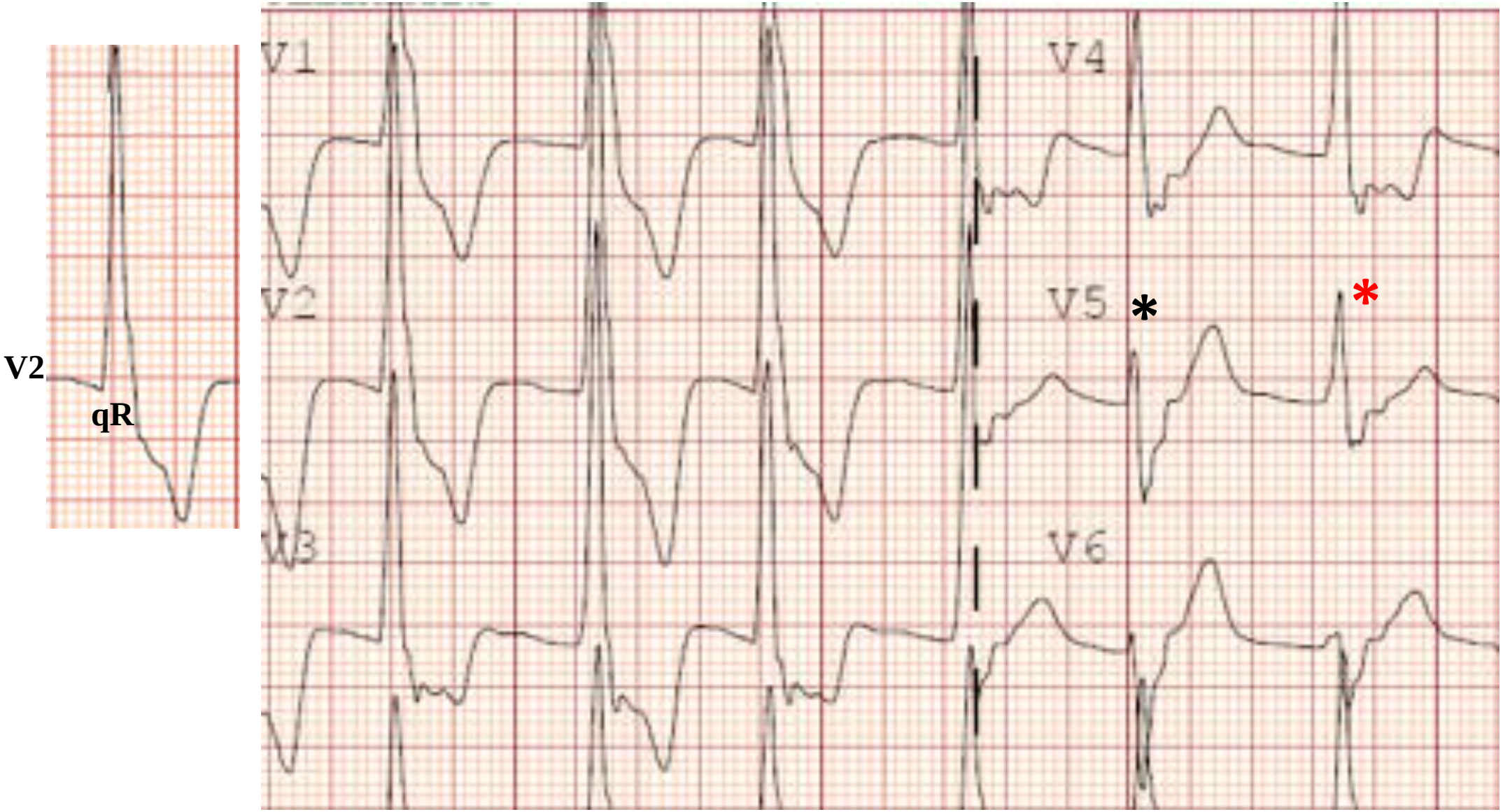
Whether such association exists with reperfusion through direct percutaneous coronary intervention was investigated in a study of 125 consecutive patients undergoing direct percutaneous coronary intervention for a first acute MI. 24-hour Holter monitoring revealed that AIVR appeared in 15.2% of the patients. The incidence of AIVR was not different between patients with TIMI grade 2 flow and those with TIMI grade 3

flow (13% vs 16%). No differences were reported in the incidence of major cardiac events within 12-month follow-up in patients with and without AIVR. It is a ventricular rhythm with a sequence of ≥ 3 consecutives ectopic ventricular beats, lasting less than 30 s gradual onset with a long coupling interval and the end by a gradual decrease of the ventricular rate or increase of the sinus rate and, last but not least, by a good prognosis. Its heart rate between 50 bpm and 130 bpm. The rhythm is accelerated because it usually is ≥ to the sinus one, in this case, it is called ventricular rhythm with isorhythm. It is not an escape rhythm, it is a competing rhythms self-limited and it is usually related to myocardial ischemia. In Accelerated Idioventricular Rhythm (AIVR), the rate of cardiac contraction is determined by the intrinsic rate of depolarization of the cardiac cells. It can be present at birth. In this last case, the patient had an excellent prognosis because the tachycardias resolved, and eventually the patients were in sinus rhythm. It is important to establish the diagnosis when it occurs to differentiate this benign phenomenon from dangerous paroxysmal ventricular tachycardia (Freire 2008).



ECG-2 lead II long shows variations on the HR. So with higher HR (107 bpm) a complete LAFB pattern is registered, and in the other hand, with lower HR (94 bpm) decreases the degree of LAFB (odd beats). Consequently, with an increase of the HR, decreases the dromotropic disturbance inside the left anterior fascicle. This is an unstable form of AIVR. Why? Because the event shows acceleration and deceleration HR.

VT/PVCs that originate in the LV 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 VT/PVC originates in the posteroinferior region of the LV, 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 VT/PVC originates in the anterosuperior region of the LV, 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 VT/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.



Precordial leads show complete RBBB pattern (QRS duration = 160 ms) and embryonic initial Q wave followed by giant R wave. Additionally in V5 we can see beats with complete LAFB influence (*) and incomplete LAFB (*). Prominent anterior forces with increased ventricular activation time in V1 and V2, small (embryonic) initial q wave in V2-V3, R wave of V2 and V3 >15 mm, sharp-pointed R wave in V2-V3 leads with slow descendent ramp, absence of q wave in left precordial leads V5, V6 and I (by absence of the vector 1_{AM}?), increasing voltage of R wave for all intermediary precordial leads, and decreasing from V5 to V6. We suggest RBBB with LSFB in association?

AIVR presentation forms

This characterization is very important, because it is one of the criteria to follow a therapeutic attitude or not.

A) Stable form:

Alternation between sinus and ventricular rhythm. The coupling of the first beat is usually fixed and delayed, and the rate of the different events is usually similar.

B) Unstable form:

1) Variable couplings of the different tachyarrhythmic events.
2) Rate of each arrhythmic event is different.
3) Within each arrhythmic event, rate acceleration and deceleration.
4) Frequent association with tachyarrhythmias of rapid rates, generally from the same focus.

Summary of Accelerated Ventricular Rhythms

I. An “active” ventricular rhythm due to enhanced automaticity of a ventricular pacemaker (reperfusion after thrombolytic therapy is a common causal factor).
II. Ventricular rate 60-100 bpm (anything faster would be ventricular tachycardia)
III. Sometimes called ***isochronic ventricular*** rhythm because the ventricular rate is close to underlying sinus rate
IV. May begin and end with fusion beats (ventricular activation partly due to the normal sinus activation of the ventricles and partly from the ectopic focus).
V. Usually benign, short lasting, and not requiring of therapy.

Possibles etiologies

1. Acute phase of myocardial infarction (MI): present in 15% of the cases (**Chiladakis 2001**).

   A) Inferior or inferoposterior wall: in this case they originate in the posterior fascicle of the left bundle branch. Myocardial ischemia (especially inferior wall ischemia or infarction

   B) Acute phase of MI of anterior wall: in this case, they originate in the anterior fascicle of the left bundle branch
2. Chronic phase of infarction
3. Thromboangiitis obliterans (Buerger's disease) (**Hsu 2008**)
4. During inhalational induction with halothane (**Chhabra 2008**)
5. Associated to ophthalmic timolol/dorzolamide solution (**Attanasio 2004**)
6. After aconite poisoning (**Fujita 2007**)
7. Associated with desflurane administration (**Marret 2002**)
8. Electrolyte imbalance: Extreme hyperkalemia (K+ > 10. mmol/l) (**Kes 1995**) and hypokalemia.
9. No underlying heart disease
   - In young patients and in newborns (**Freire 2008**)
   - Hypervagotonia in highly conditioned athletes (**Nasir 2007**)
   - During the antenatal period (**Dulac 2004**)
10. Coronary artery dissection (**Karabinos 2006**)
11. Congenital diseases (**Reynolds 2001**)
12. Primary cardiomyopathies (**Grimm 2000**)
13. Post-resuscitation (**Tsai 2007**)
14. Hypertensive heart disease (**Sideris 1987**)

Electrophysiological mechanisms of AIVR

In most cases, the mechanism of AIVR appears to be related to the enhanced automaticity in His-Purkinje fibers and/or myocardium, sometimes accompanied with vagal excess and decreased sympathetic activity (Bonnemeier 2005). Ischemia, reperfusion, hypoxia, drugs, and electrolyte abnormalities can all accelerate the phase 4 action potential depolarization rates in His-Purkinje fiber and myocardium, leading to faster spontaneous cell depolarization (enhanced automaticity) (Hasin 1976). When the enhanced automaticity in His-Purkinje fiber or myocardium surpasses that of sinus node, AIVR manifests as the dominant rhythm of the heart. Sinus bradycardia may facilitate the appearance of AIVR.

Any cause that increases maximum diastolic potential (MDP) depth decreases automaticity. E.g.: acetylcholine. Abnormal enhanced automaticity generally is ascribed to phase-4 depolarization of the AP of the myocardial cell. AIVR can occur in the His-Purkinje fibers or myocardium under certain abnormal metabolic conditions. AIVR arises from second-order pacemakers and manifests itself when the patient's prevailing sinus HR becomes lower than the accelerated rate (AIVR) of the competing focus. Sinus bradycardia combined with enhanced automaticity of the subordinate site is the common pathophysiology. AIVR was associated with higher vagal tone and lower sympathetic activity, the occurrence of AIVR had no prognostic impact on the clinical course. Under certain conditions such as acute ischemia and digoxin toxicity, triggered activity has been suggested as the mechanism for AIVR (Holzmann 1977).

AIVR is hemodynamically well tolerated due to its slow ventricular rate. It is self-limited and resolves as sinus rate surpasses the rate of AIVR. Rarely, AIVR can degenerate into VT or VF. In patients with severe myocardial dysfunction, AIVR may lead to hemodynamic instability due to the loss of AV synchrony or relatively rapid ventricular rate.

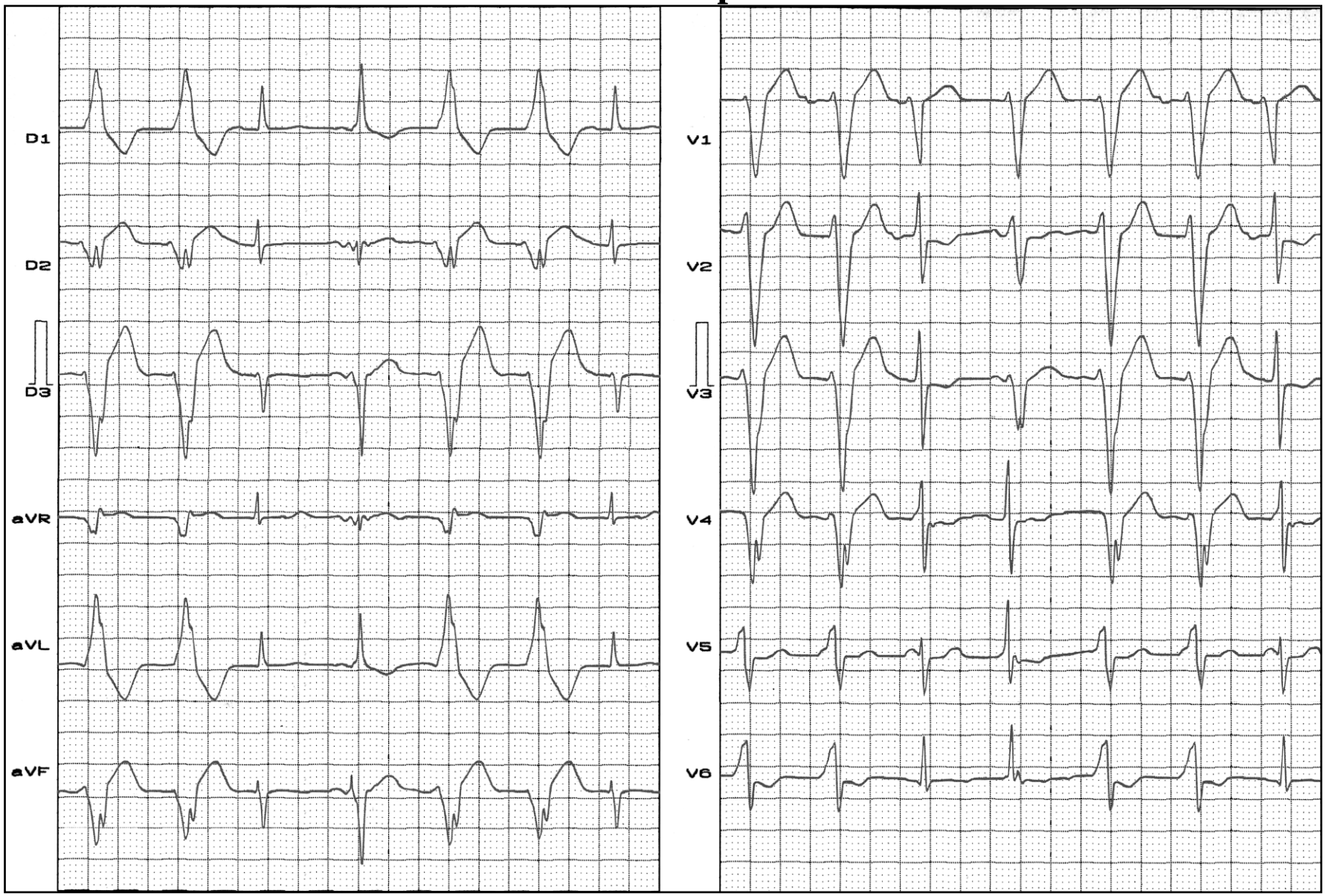
AIVR in acute myocardial infarction

Clinically, AIVR has been best studied in patients with acute STEMI. In the thrombolysis era, AIVR was noted to be a marker of reperfusion. However, not all patients with reopened coronary artery have AIVR. In patients with AMI treated with PPCI, the reported incidence of AIVR varied significantly, raging from 15-50%, depending on methods of monitoring. Studies in patients with STEMI treated with PPCI support that AIVR is a marker of occluded coronary artery reopening, but is not necessarily a marker for complete reperfusion. In fact, AIVR seems to be associated with more extensive myocardial damage and delayed microvascular reperfusion, although the mortality rates are similar in patients with and without AIVR.

ECG characterization

- 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 tachycardia 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.

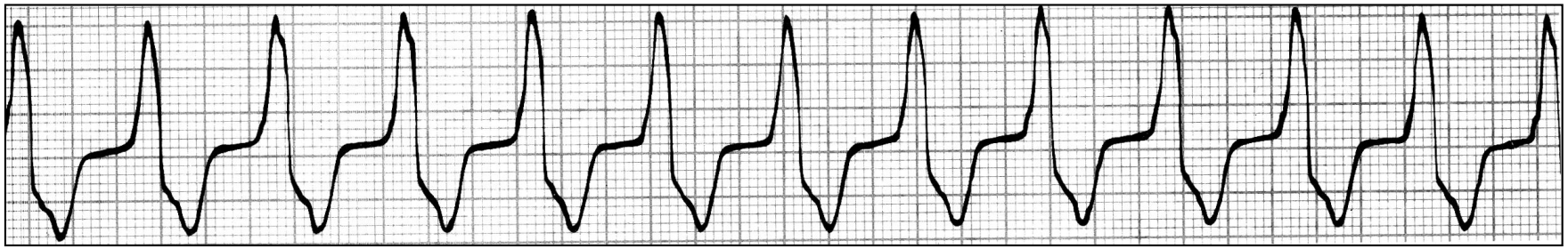
Another examples of AIVR



Clinical diagnosis: accelerated idioventricular rhythm, HR = 101/min dissociated from SR (rate=85min).
Beats 3 and 7 are capture beats. Beats 4 is fusion beat.
Delayed QRS nadir (90ms) in V1, delayed QRS peak in V6 fusion beats and capture beat.

AIVR: accelerated idioventricular rhythm or slow VT

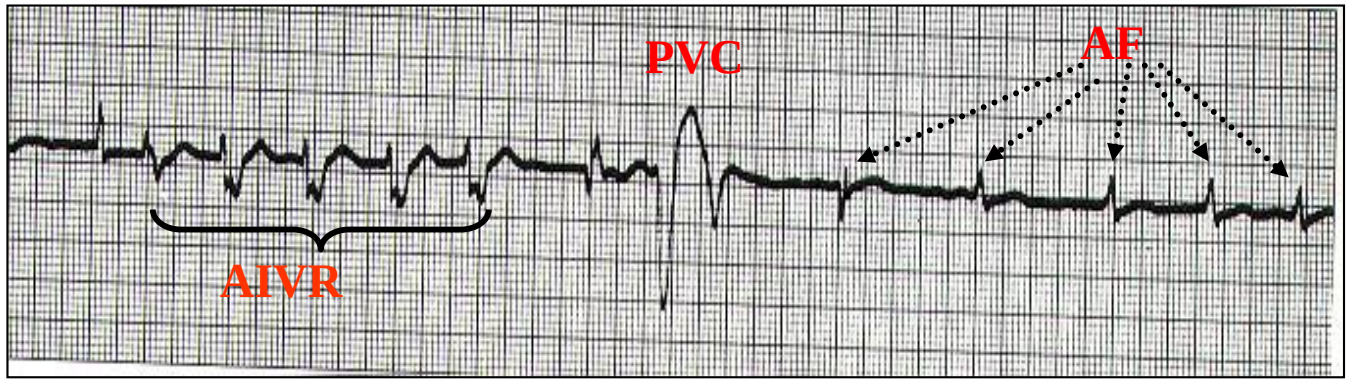
Monitor lead



Broadened QRS with “slow” rate (100 bpm)

Example of AIVR in the monitor.

Accelerated Idioventricular Rhythm(AIVR) or slow VT



Clinical features: Elderly patient, 82 years old, myocardiosclerotic. Using Digoxin 0.25 mg/day for quite some time.

ECG analysis: Absence of P wave, f waves: atrial fibrillation. From the third beat to the sixth, wide and regular QRS with rate of 110 bpm: Accelerated IdioVentricular Rhythm (AIVR). The eighth beat is a PVC. In such case, AIVR indicates digitalis intoxication. The level of serum digoxin was 3 ng/mL.(Normal values range from 0.8 to 2.0 ng/ml) The levels above 2.5 ng/mL in adults are considered to be toxic. AIVR is currently defined as an enhanced ectopic ventricular rhythm with at least 3 consecutive ventricular beats, which is faster than normal intrinsic ventricular escape rhythm (≤ 40 bpm), but slower than VT (at least 100-120 bpm). There is HR overlap between AIVR and some slow VT. AIVR should not be diagnosed solely based on HR. AIVR is generally a transient rhythm, rarely causing hemodynamic instability and rarely requiring treatment. However, misdiagnosis of AIVR as slow VT or complete heart block can lead to inappropriate therapies with potential complications. AIVR is often a clue to certain underlying conditions, like myocardial ischemia-reperfusion, digoxin toxicity and cardiomyopathies.

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