Estimados compañeros del foro,
Les remito este interesante ecg para su discusión y escuchar sus valiosas opiniones.
Se trata de una mujer de 37 años con clínica de palpitaciones de 48h de evolución sin otros antecedentes de interés.
El primer ECG es a su llegada al hospital.
Los otros dos después de diltiazem intravenoso.
Se le practico un ecocardiograma transesofágico (no dispongo aun del informe, pero en principio la fracción de eyección esta en el limite de la normal y no informan anormalidades en el VD a priori) durante el cual tuvo náuseas, pasando a ritmo sinusal y volviendo rápidamente a este ritmo incesante (no dispongo del registro).
A la espera de sus valiosas opiniones.
Gracias!
Dr. Carlos Lopez

Dear colleagues

I am sending this interesting ECG for your discussion.

She is a 37-year-old woman with a 48-hour history of palpitations with no other relevant history.

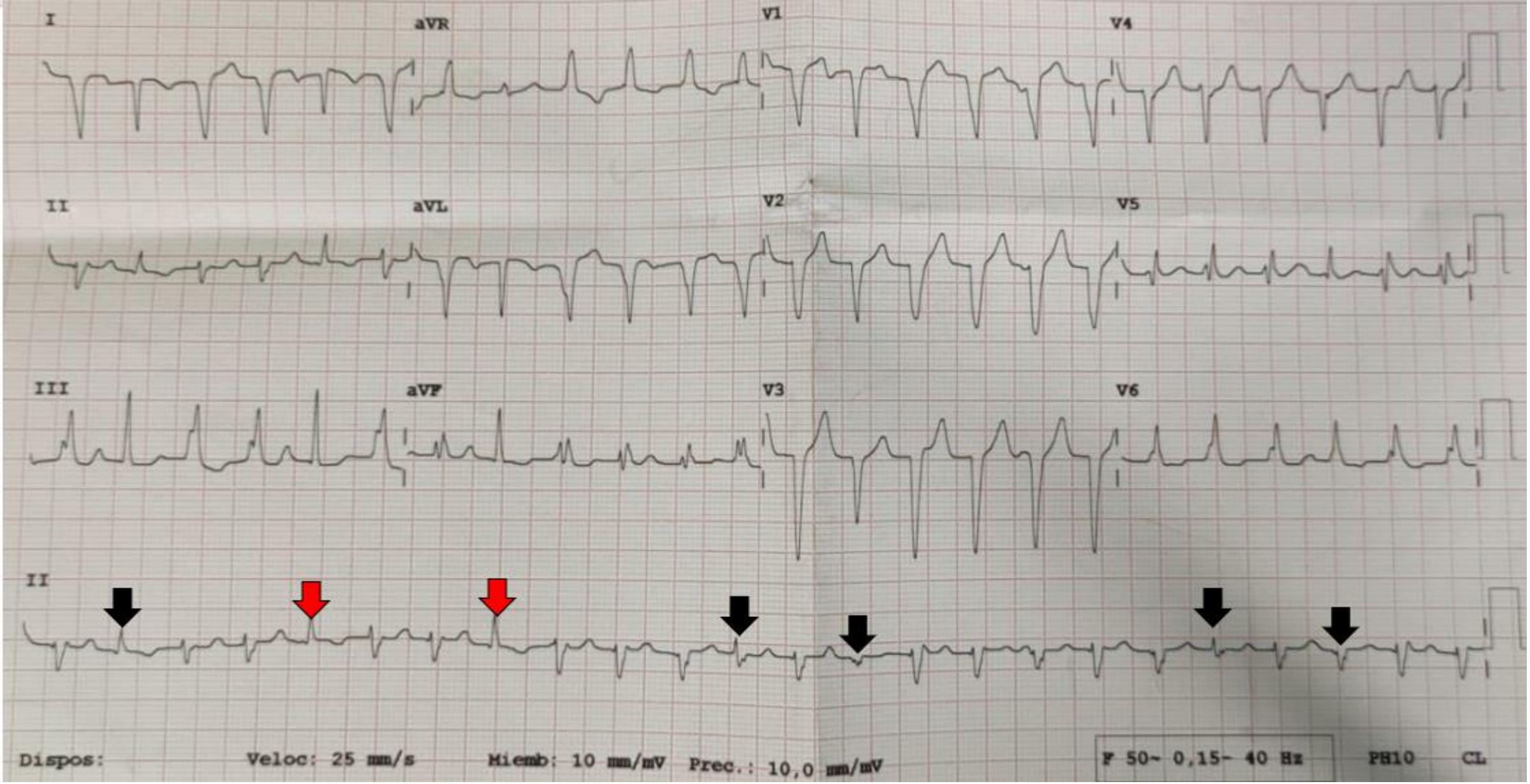
Her first ECG is upon her arrival at the hospital. The other two after intravenous diltiazem.

A transesophageal echocardiogram was performed (I do not yet have the report, but the LVEF is normal as well as the RV. During the event she had náusea with reversion to sinus rhythm and quickly returning at this incessant rhythm (I don't have this tracing).

Waiting for valuable opinions from her.

Thanks!

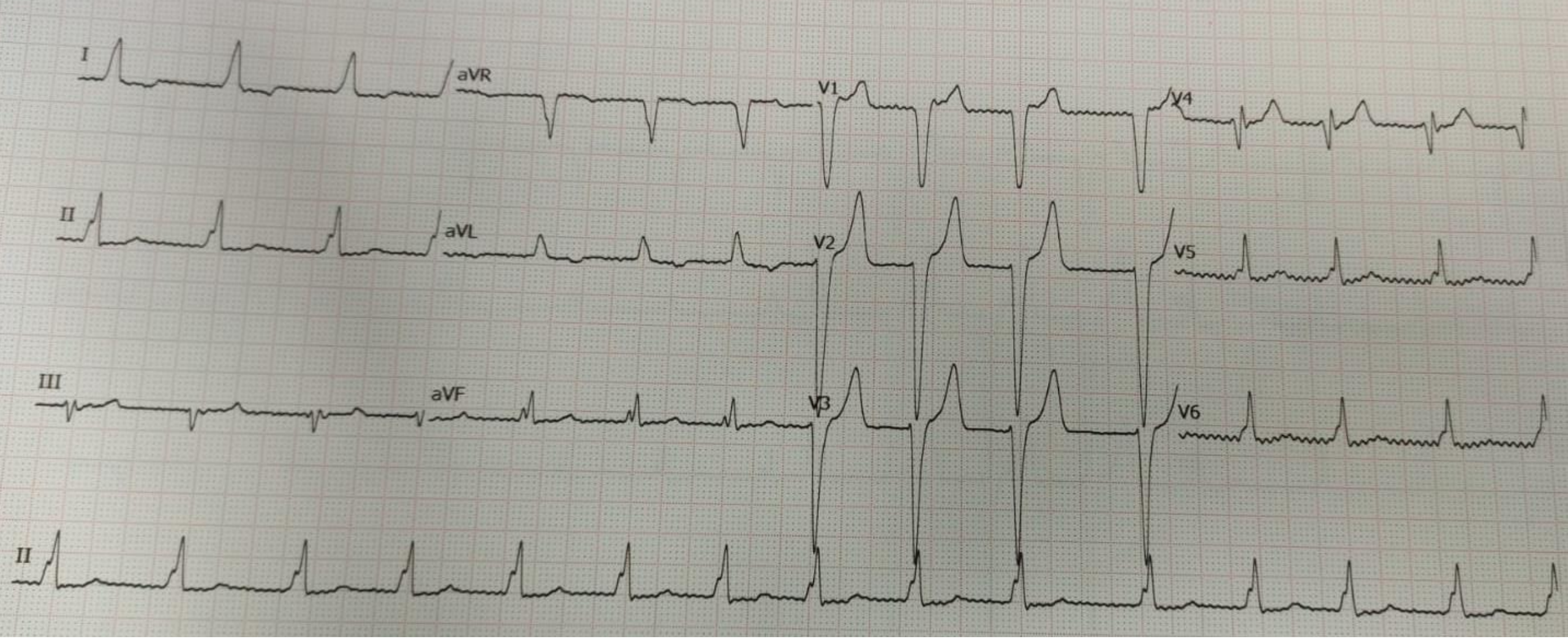
Dr. Carlos Lopez



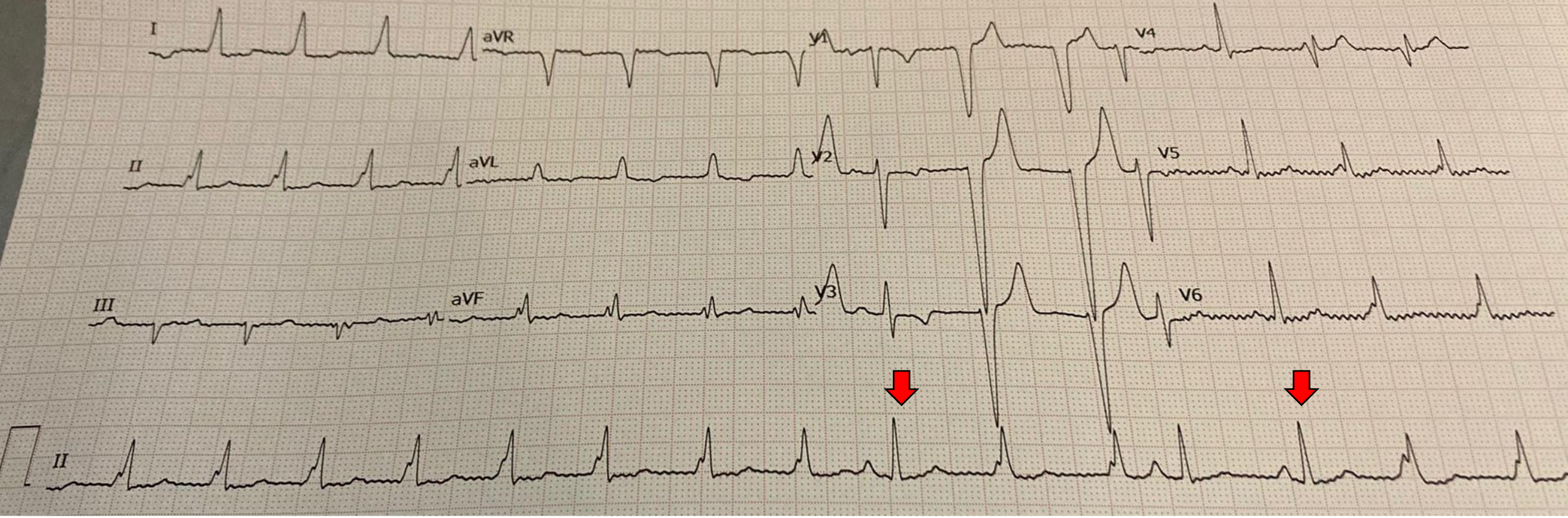
HR: 144 bpm, P axis 0°, PR interval 52 ms, QRS axis 163°, QRS duration 120 ms, QT/QTc 319/494, T axis 9°

This ECG has a lead placemet error (R-arm, L-arm with interchange)

F= fusion beats ⬇; C= Capture beats ⬇ The rhytm is Ventricula tachycardia, AV dissiation na with fusions(F) and capture beats (C)



This EC has correct lead placement: Accelerated ventricular rhytm (HR ≈ 75-90bpm



Accelerated ventricular rhytm (HR ≈90bpm) + AV dissociation with 2 captures ↓ and sinus rate HR ≈75bpm

Accelerated Idioventricular Rhythm

Concept: It is a ventricular rhythm with a sequence of ≥ 3 consecutives monomorphic 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**).

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

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.

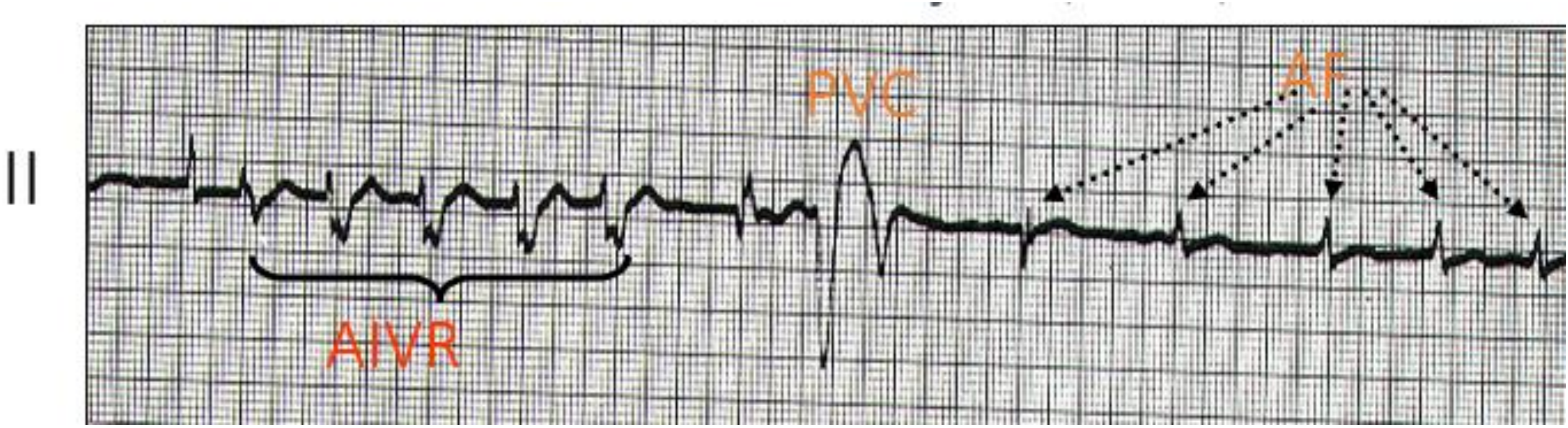
Possibles etiologies

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**)

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.

Significance of AIVR in AMI scenario

Post-reperfusion during coronary thrombolysis in the restoration of the anterograde coronary flow, which indicates reperfusion. 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.

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).

Most AIVRs originate from a single focus. Occasionally, in patients with acute myocardial ischemia and myocarditis, AIVR can originate from multiple foci (Sclarovski 11983; Nakayama 1988). The ventricular rate of AIVR is generally between 40 to 100-120 bpm.

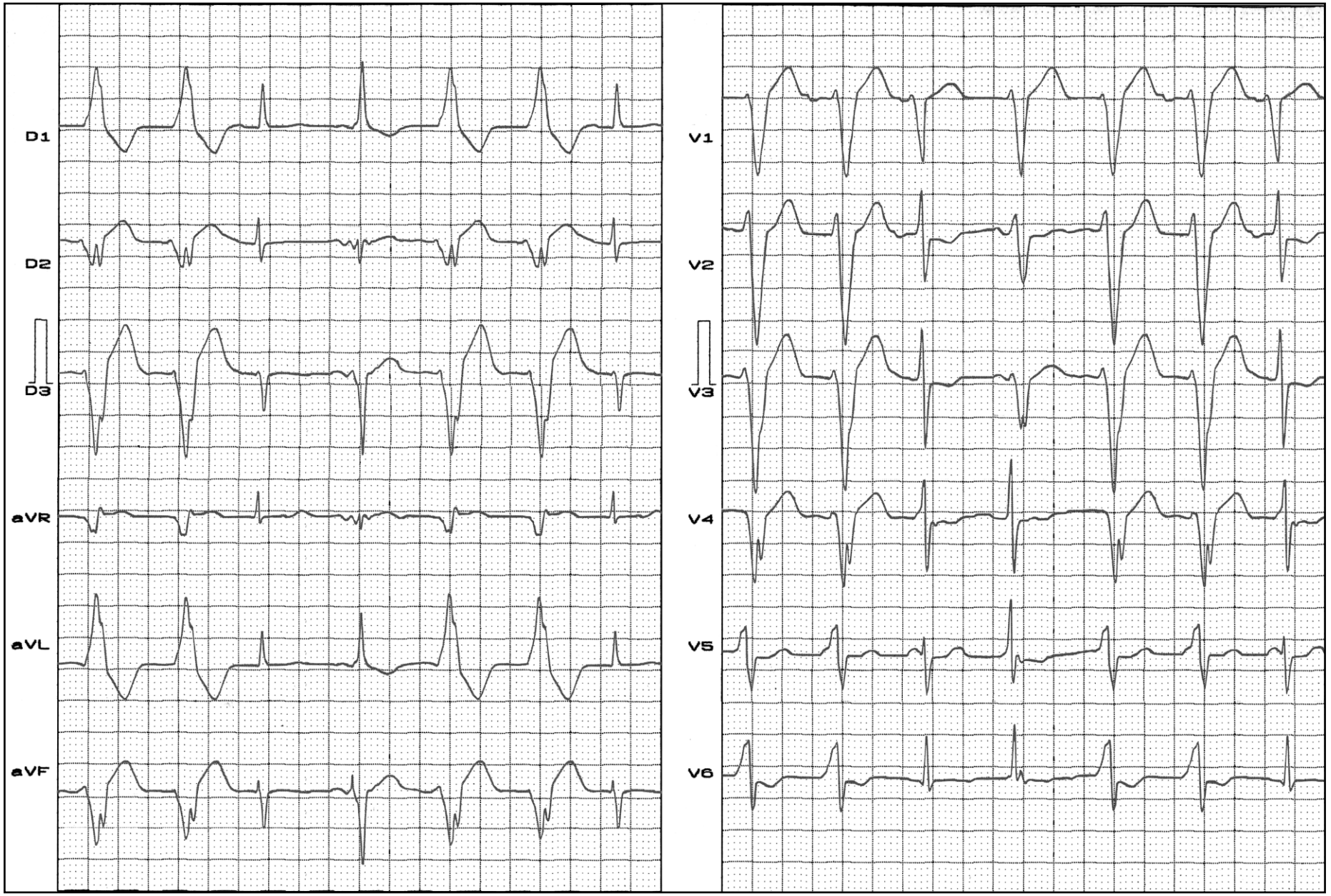
AIVR is hemodynamically well tolerated due to its slow ventricular rate. It is self-limited and resolves as sinus rate surpasses the rate of AIVR. Rarely, AIVR can degenerate into VT or VF. In patients with severe myocardial dysfunction, AIVR may lead to hemodynamic instability due to the loss of AV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

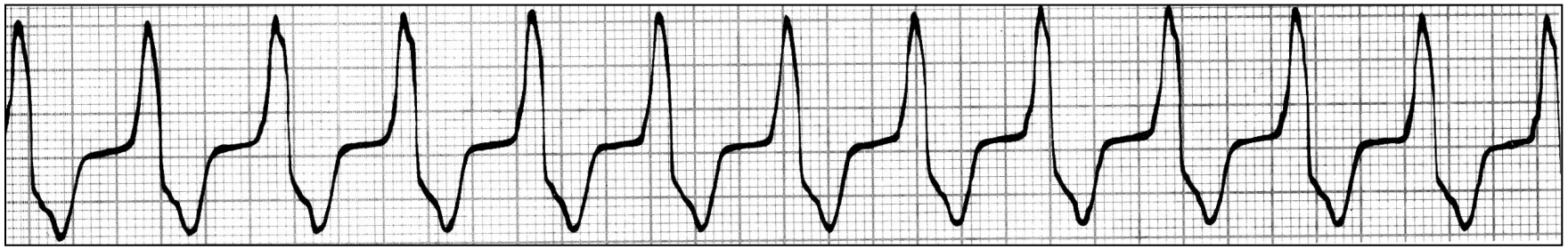
- *Duration of QRS complex: ≥ 120 ms;*
- *Constant and bizarre morphology of QRS complexes (monomorphic);*
- *Slow rate: between 50 bpm and 130 bpm (usually between 70 and 85 bpm);*
- *Regular or almost regular R-R;*
- *Event SÂQRS different from basic rhythm SÂQRS;*
- *Onset and end of event, gradual 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 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.*



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.