Acute Myocardial Infarction Complicated With Ventricular Tachycardia

Infarto Agudo del Miocárdio complicado con taquicardia ventricular

Infarto agudo do miocárdio complicado com taquicardia ventricular

Where is located the "culprit" artery? ¿Donde está localizada la artéria culpada? Onde está a artéria culpada?

Which is the diagnose of the sustained tachyarrhythmia? ¿ Cual es el diagnóstico de la taquiarritmia? Qual o diagnóstico da taquiarritmia?

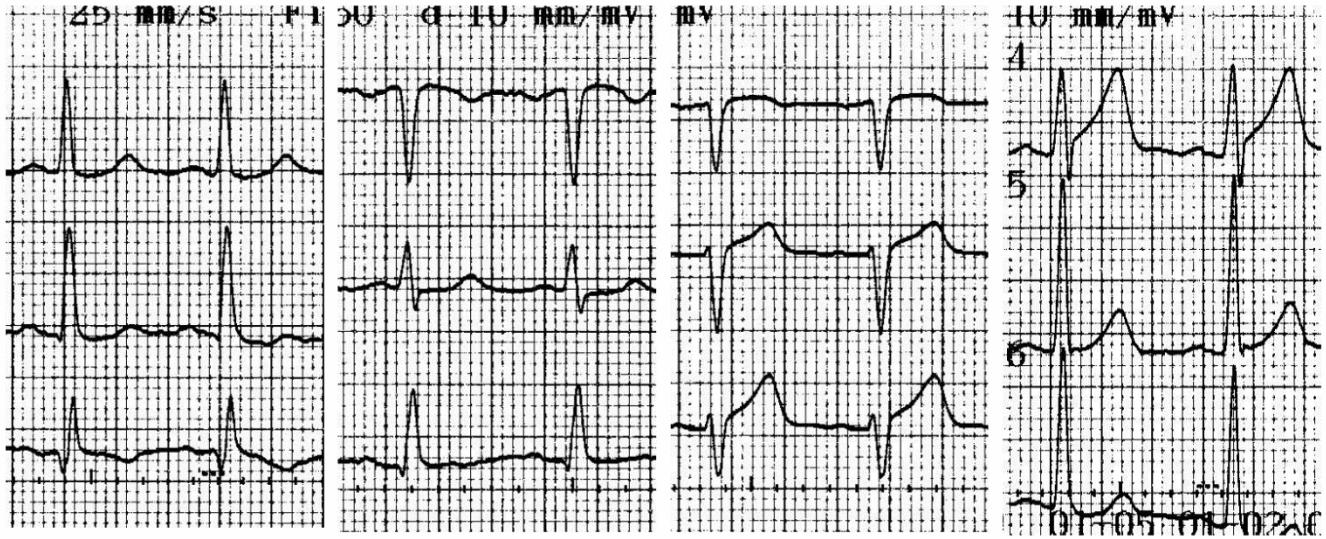
When polymorphic VT appears ST segment elevation degree decreases. Reperfusion Arrhythmia? Coronary angiography showed coronary oclusion + Stent primary angioplasty. Cuando la TV polimorfa aparece la elevación del segmento ST disminuye: ¿Arritmia de reperfusión? Coronariografia mostró oclusão con angioplastia primaria + Stent.

Quando a TV polimórfica aparece a elevação do segmento ST diminui: Trata-se de arritmia de reperfusão? A coronariografia mostrou oclusão com angioplastia primária + stent

Case from Dr Oscar Pellizón MD Argentine

DI DII DIII aVR aVL aVF V1 V2 V3 V4 V5 V6

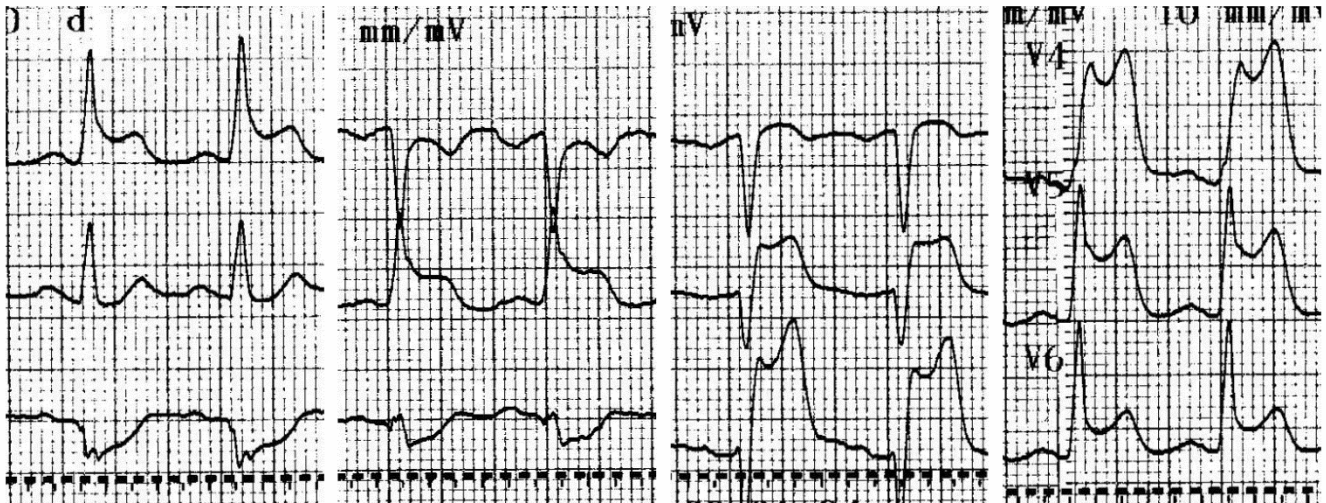
11,18 hs.



Angor

DI DII DIII aVR aVL aVF V1 V2 V3 V4 V5 V6

11,27 hs.



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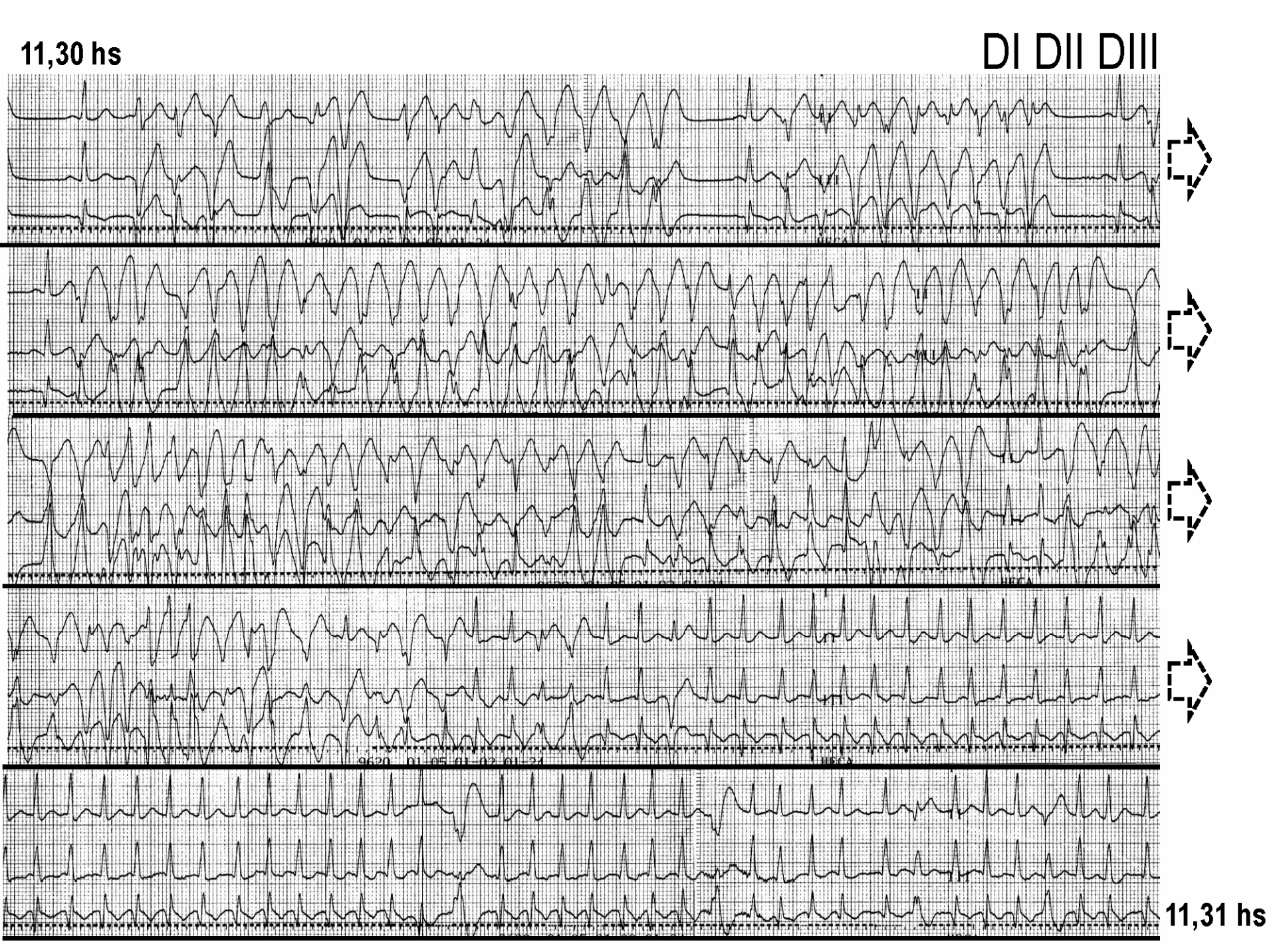

11,30 hs
DI DII DIII
11,31 hs

Caro Andrés:
O infarto é de localização ântero-lateral e a artéria culpada, provalmente é a DA. A taquicardia ventricular pode representar neste caso, arritmia de reperfusão. Sabemos que as arritmias de reperfusão se manifestam, em se tratando de taqui-arritmias, seja como TVNS, TV-Sustrentada, ritmo idioventricular acelerado ,torsades de pointe e fibrilação ventricular, seja por reperfusão espontânea, seja determinada por angioplastia coronária

Um grande abraço do amigo Hélio Germiniani.

Dear Andrés:
The myocardial infarction is located in anterolateral wall, and the culprit artery, is probably the left anterior descending artery (LAD). Ventricular tachycardia can represent in this case a reperfusion arrhythmia. We know that reperfusion arrhythmias manifest in the case of tachyarrhythmias as non-sustained ventricular tachycardia (NS-VT), sustained ventricular tachycardia(S-VT), accelerated idioventricular rhythm, torsades de pointes and ventricular fibrillation, either by spontaneous reperfusion, or determined by coronary angioplasty

A big hug from friend Professor Dr. Hélio Germiniani M.D.Ph.D. Curitiba Paraná PR/Brazil
Ex-President of Paranense Society of Cardiology (SPC) Curitiba - PR

Prezado Maestro

Respostas - Respuestas - Answers

1. Infarto ântero-lateral alto - Infarto de la parede antero-lateral e alta - High anterolateral AMI - I - aVL + V3 a V6

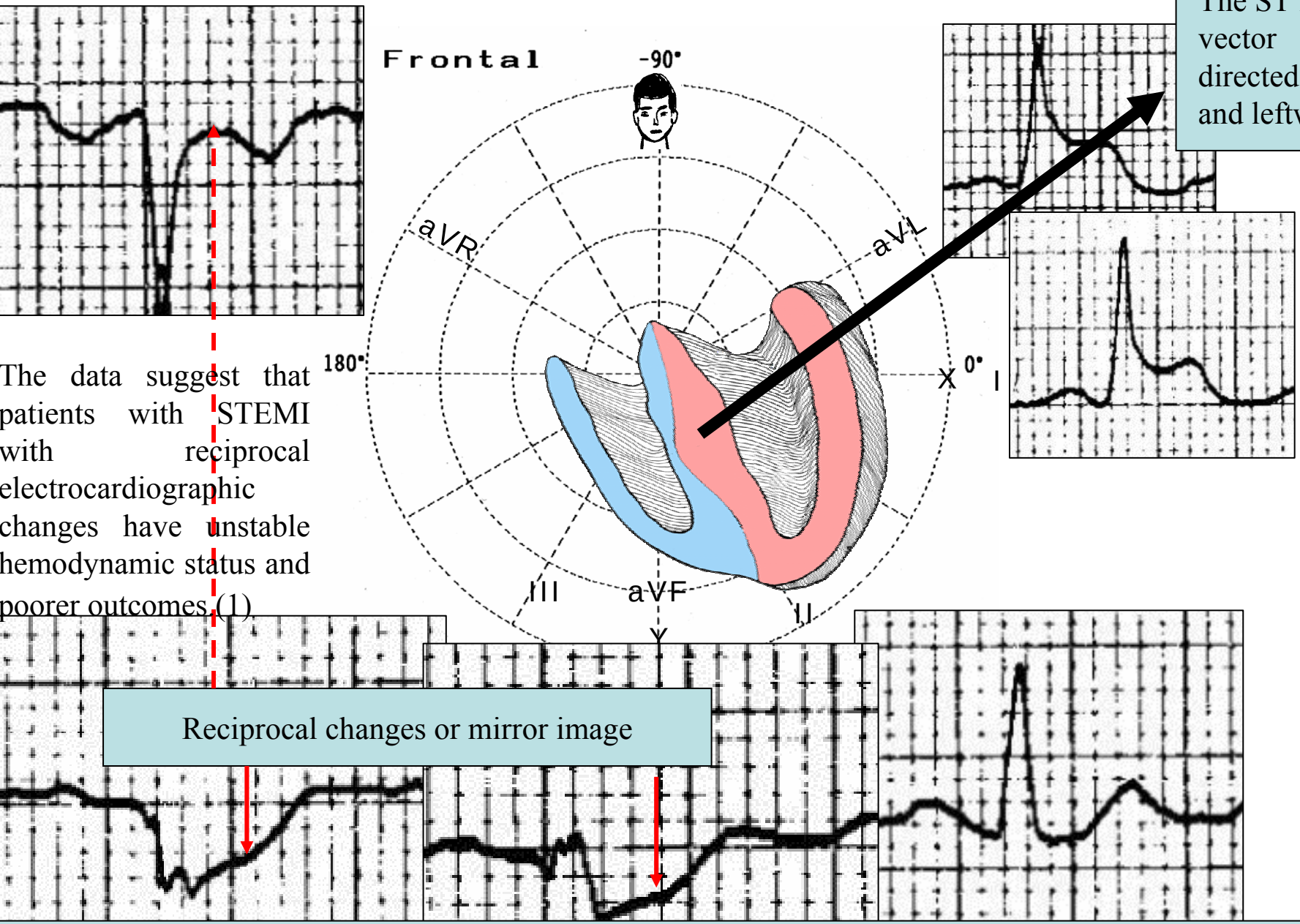
2. Artéria culpada: ADA antes do 1º Ramo diagonal 1 . "Culprit" Artery: Left Anterior Descending Artery before the First diagonal branch

3. Taquicardia ventricular polimórfica típica de reperfusão (inversão de 180º no eixo: 2ª e 3ª tira loga no inicio, batimentos de fusão e dissociação) com desapareciemento do supra ao cessar a TV (Sclarovsky).
Polymorphous ventricular tachycardia (c/ TP) it´s typical post reperfusion arrihytmia

Adail – Vitória da Conquista - Bahia - Brazil

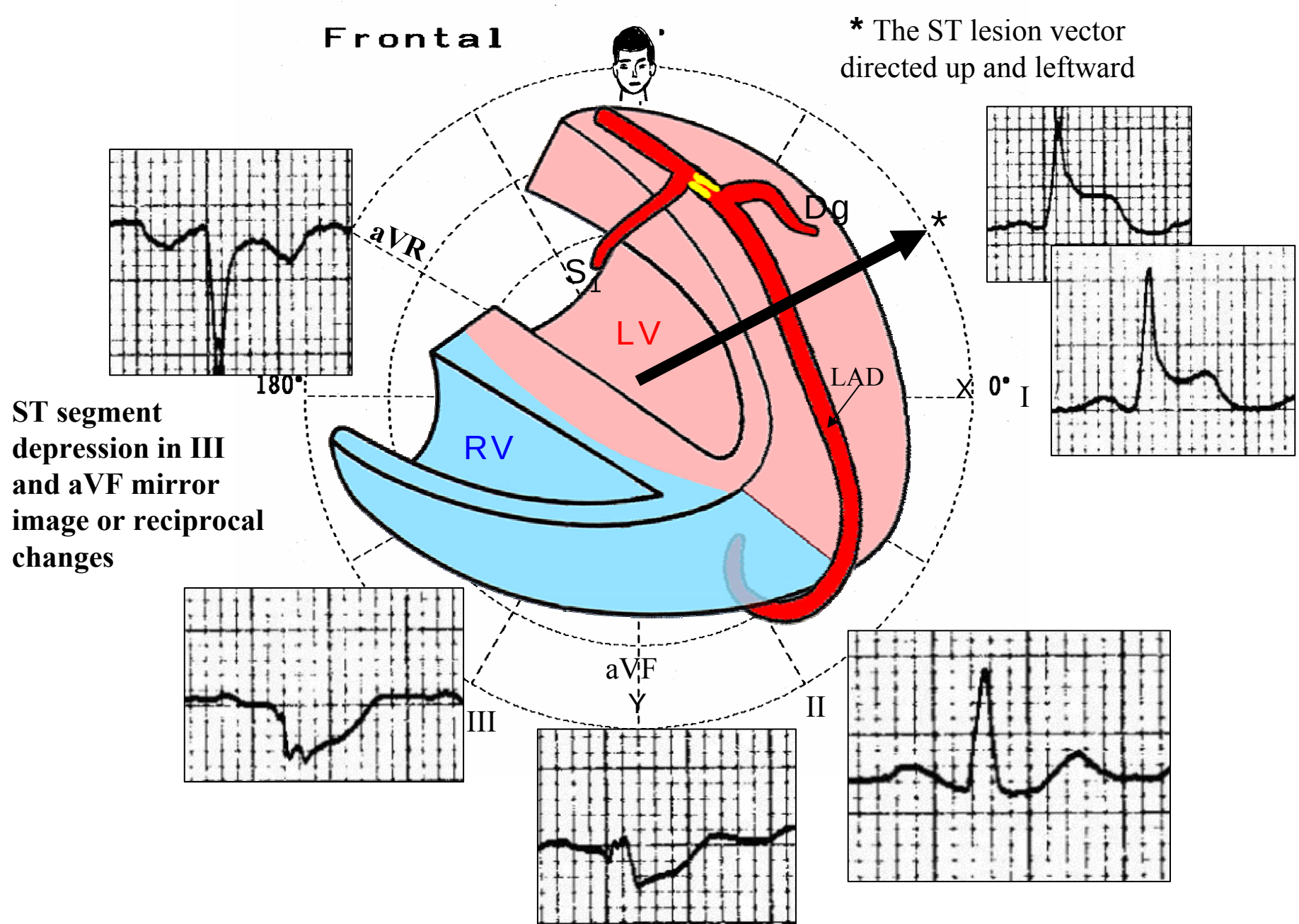
Final comments

By Andrés Ricardo **Pérez-Riera** M.D.Ph.D.

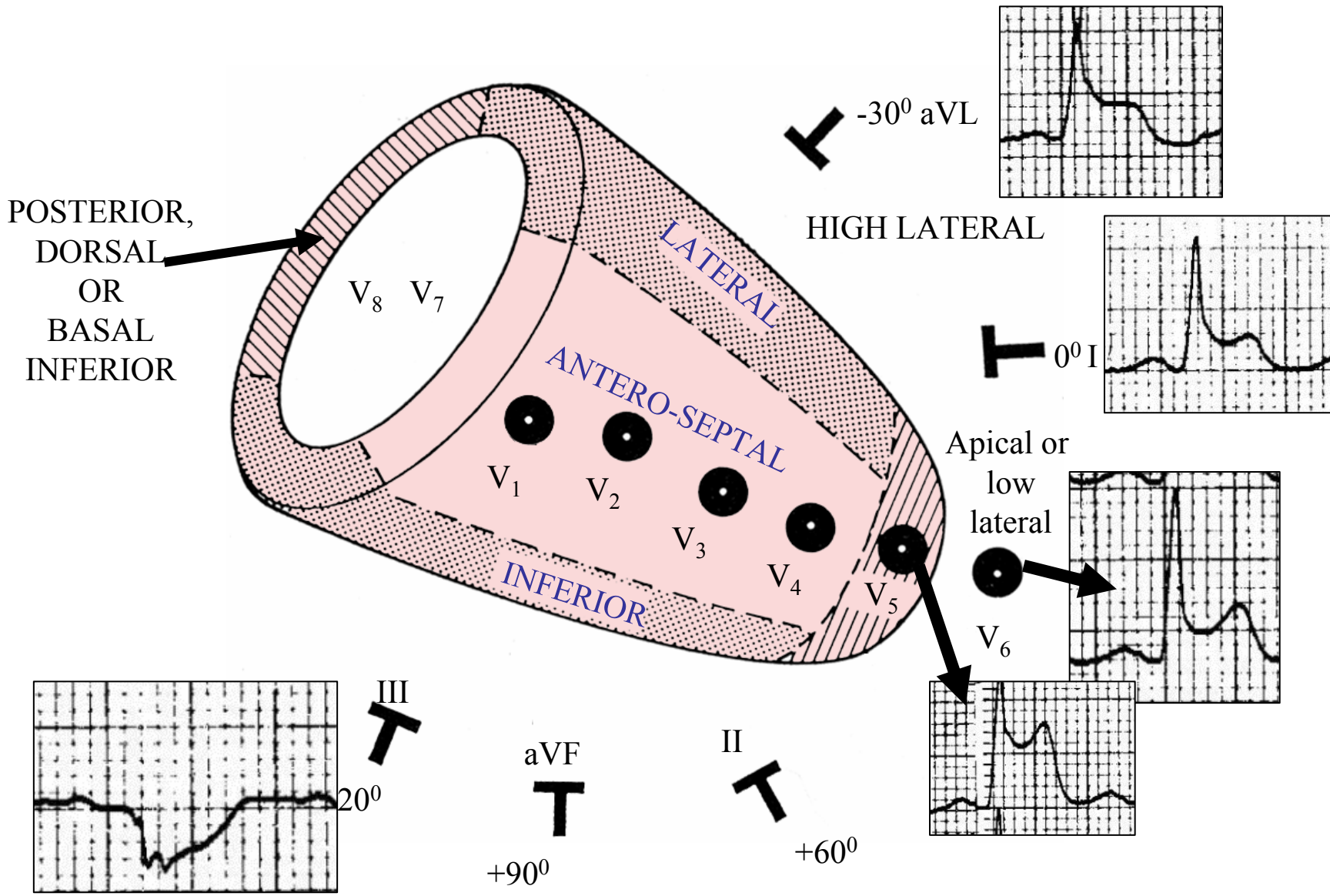


1. **Chen TE, Lo PH, Li TC, et al. Prognostic significance of reciprocal ST-segment depression in patients with acute ST-segment elevation myocardial infarction undergoing immediate invasive intervention. Am J Emerg Med. 2012 May 23. [Epub ahead of print]**

ST segment elevation in I and aVL. ST segment depression in III



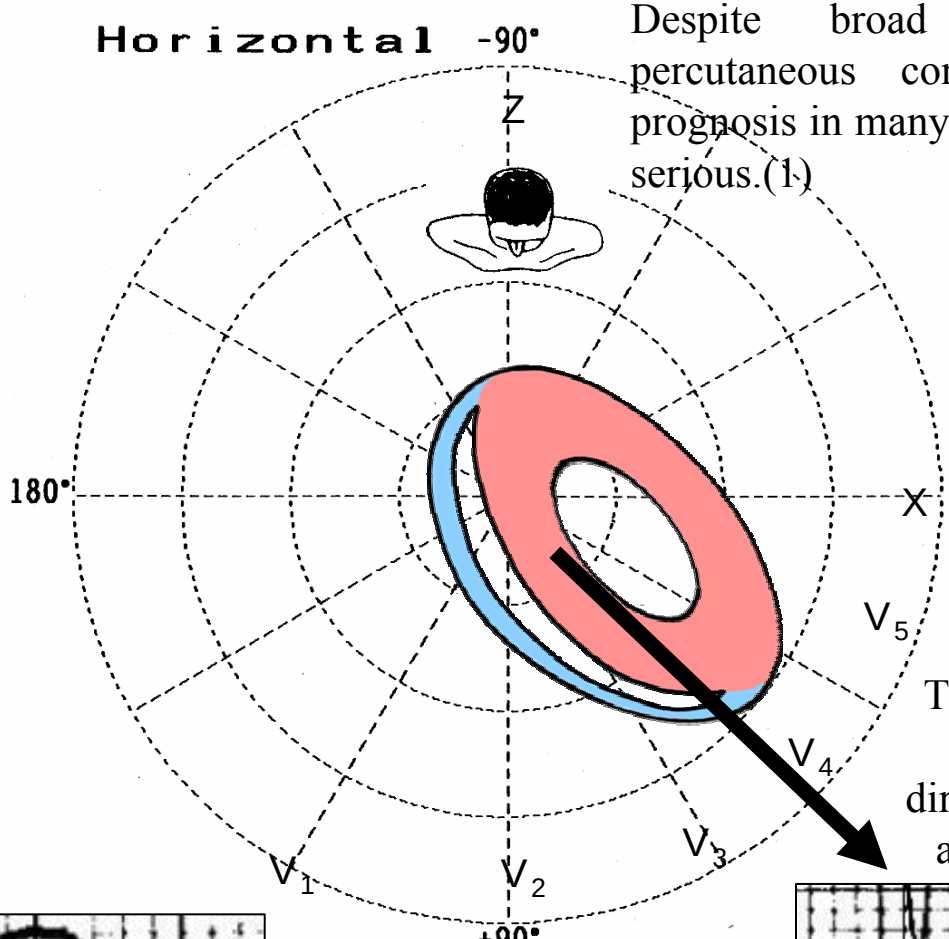
TOPOGRAPHIC LOCATION OF MYOCARDIAL INFARCTION



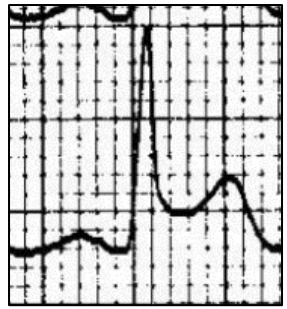
The cardiac cone, its sides and corresponding leads: Antero-septal: V1 to V4; Apical or low lateral: V5 and V6; High lateral: DI and aVL; Inferior or diaphragmatic: DII, DIII and aVF; Dorsal, posterior or postero-basal: V7 and V8.

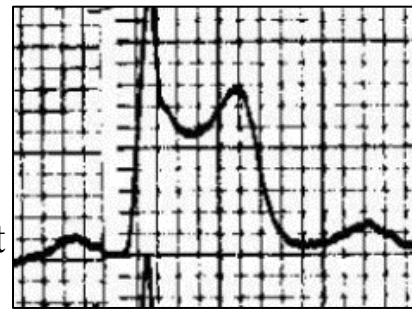
ST segment elevation from V2 to V6 and isoelectric in V1

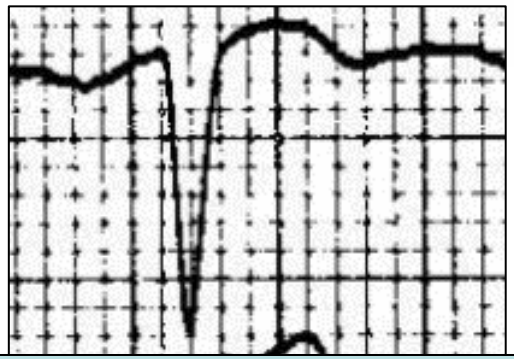
Despite broad implementation of primary percutaneous coronary intervention (pPCI), the prognosis in many patients with large infarcts remains serious.(1)

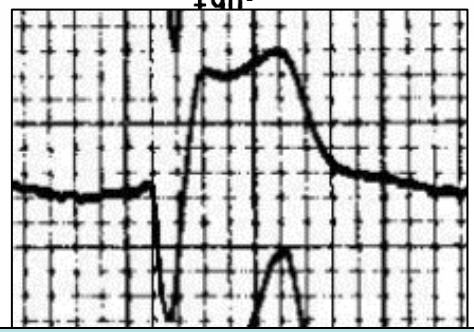


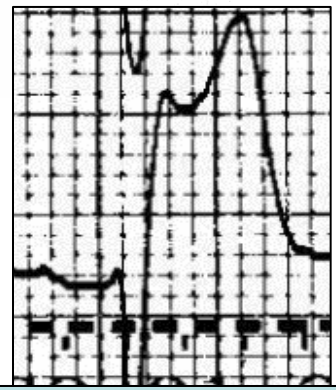
The ST injury vector directed to front and leftward

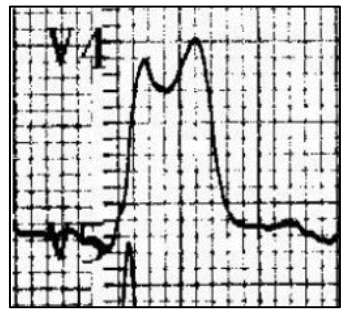












1. Koziński M, Kasprzak M, Rychter M, et al. Primary percutaneous coronary intervention facilitated with supersaturated oxygen therapy in a patient with anterior myocardial infarction: a case report and literature review. Kardiol Pol. 2012;70:172-174.

11,18 hs.

New recent J point and ST segment elevation in 2 or more adjacent leads ≥ 2 mm in V_1, V_2 or V_3(in the present case from V_1 to V_6) Subepicardial injury current

Preserved initial embryonic r wave

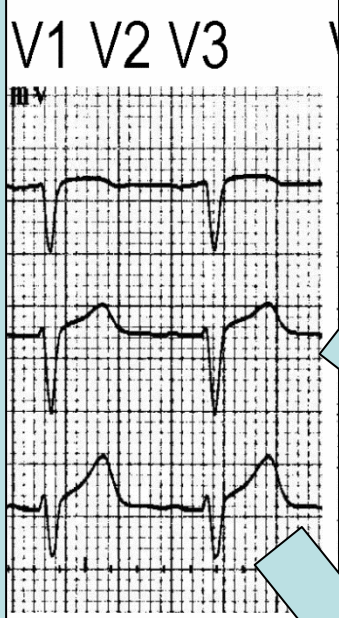
Indicative of hyper acute phase MI

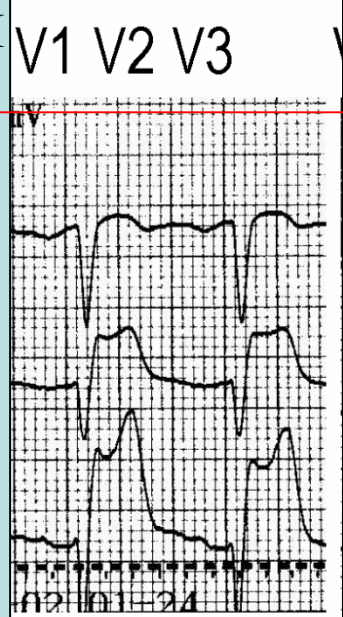
11,27 hs.

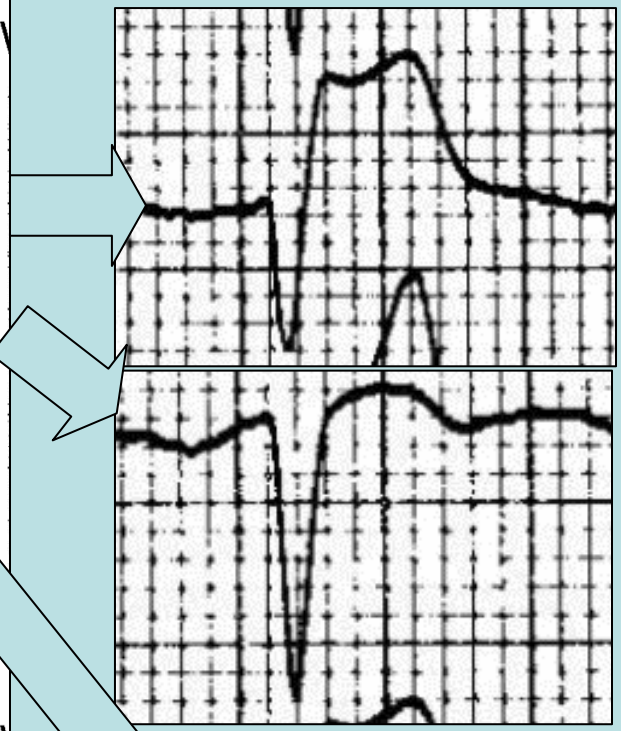
Second ECG preformed 9´ later

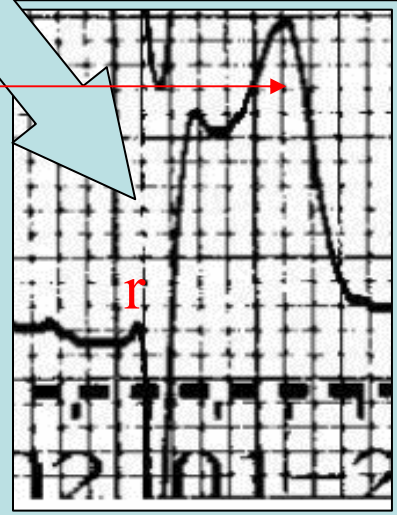
The T wave becomes wider, symmetrical, with a wider base and pointy: it is the image of subendocardial ischemia.

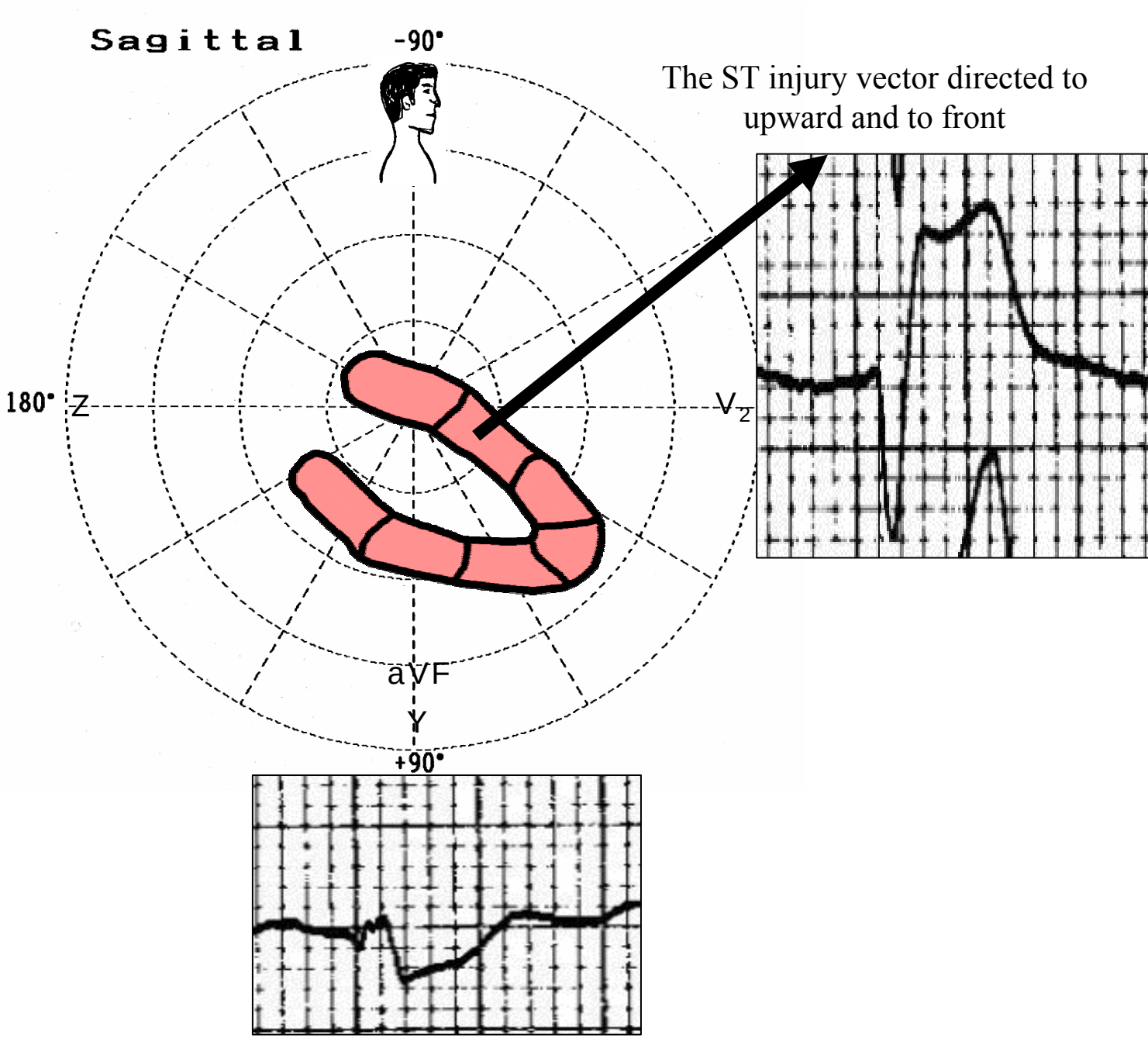
The earliest changes are: tall, positive, hyper acute T waves in the ischemic vascular territory. This is followed by elevation of the ST segments (myocardial "injury pattern").










Sagittal
-90°
180°
Z
V2
aVF
Y
+90°
The ST injury vector directed to upward and to front

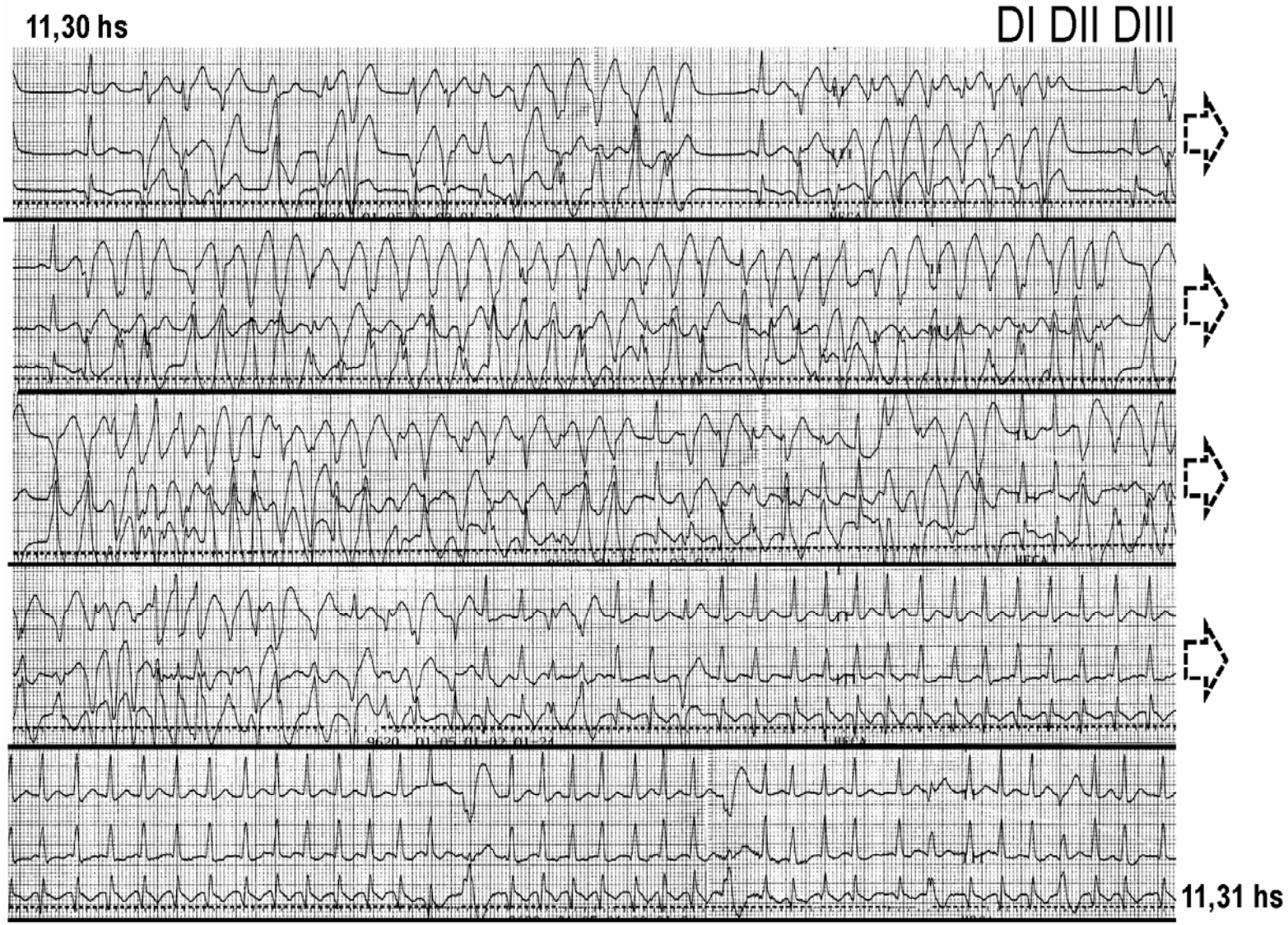
Anterolateral myocardial infarctions Extensive anterior infarction producing indicative changes across the precordium as well as in leads I and aVL. This kind if MI is frequently caused by occlusion of the left main coronary artery(LMCA) proximal left anterior descending coronary artery (LAD), or combined occlusions of the LAD together with the right coronary artery or left circumflex artery. When LMCA occlusion is the subjacent cause, cardiogenic shock is the rule (cardiac index < 2.0, left ventricular end-diastolic pressure > 25, and pulmonary edema). Patients with an anterolateral acute myocardialinfarction (AMI) have a worse prognosis, and those with addit ional inferolateral wall involvement might be higher risk because of more extensive area at risk. Lead –aVR obtained by inversion of images in lead aVR has been reported to provide useful information for inferolateral lesion. in patients with an anterolateral AMI, ST-segment depression in lead aVR on admission ECG is useful for predicting larger infarct and LV dysfunction despite successful reperfusion.(1) Arrythmias which commonly preclude the diagnosis of anterolateral MI on ECG and therefore possibly identify high risk patients include:

1) Right and left bundle branch blocks
2) Fascicular blocks: LAFB and LSFB
3) Mobitz type II second degree atrioventricular conduction blocks.

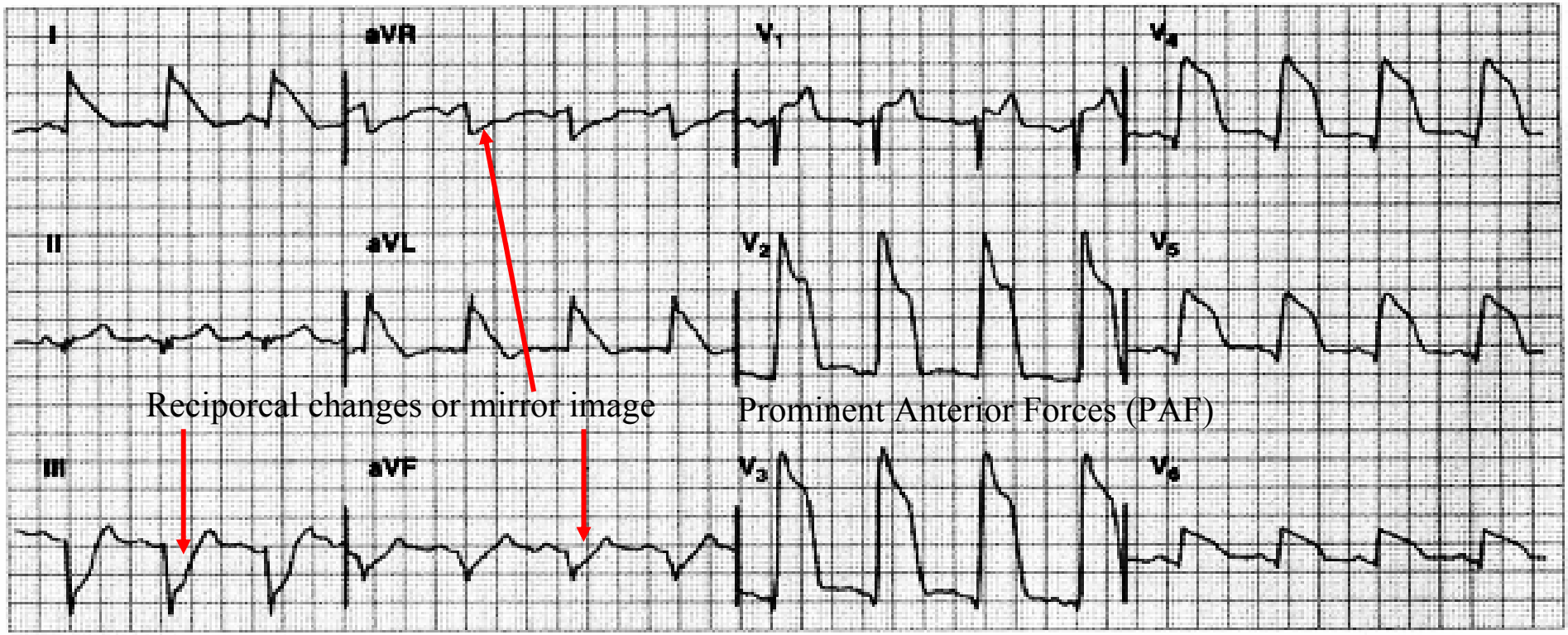
ST-segment elevation in lead V_1 during the acute phase of anterior AMI is associated with a high incidence of regional abnormality of wall motion in the basal anterior, anteroseptal, and anterior regions, whereas ST-segment elevation in lead V2 is more often associated with regional abnormality of wall motion in the inferoapical region. ST-segment elevation in aVL leads is related to mid-lateral regional abnormality of wall motion.(2)

1. Porter A, Strasberg B, Vaturi M, et al.Correlation between electrocardiographic subtypes of anterior myocardial infarction and regional abnormalities of wall motion.Coron Artery Dis. 2000 Sep;11:489-493.
2. Kosuge M, Kimura K, Ishikawa T, et al. ST-segment depression in lead aVR predicts predischarge left ventricular dysfunction in patients with reperfused anterior acute myocardial infarction with anterolateral ST-segment elevation.Am Heart J. 2001 Jul;142:51-57.

The mode of onset of polymorphic VT (PVT) in acute MI patients is often preceded by PVC in acute MI patients. PVT is defined as sudden-onset tachycardia if it was not preceded by PVCs. Nonsudden-onset PVT that was preceded by single or multiple PVCs is considered as non sudden-onset tachycardia. PVT is usually characterized by a lower coupling interval, shorter PVT cycle length and an associated lower LVEF. (1)



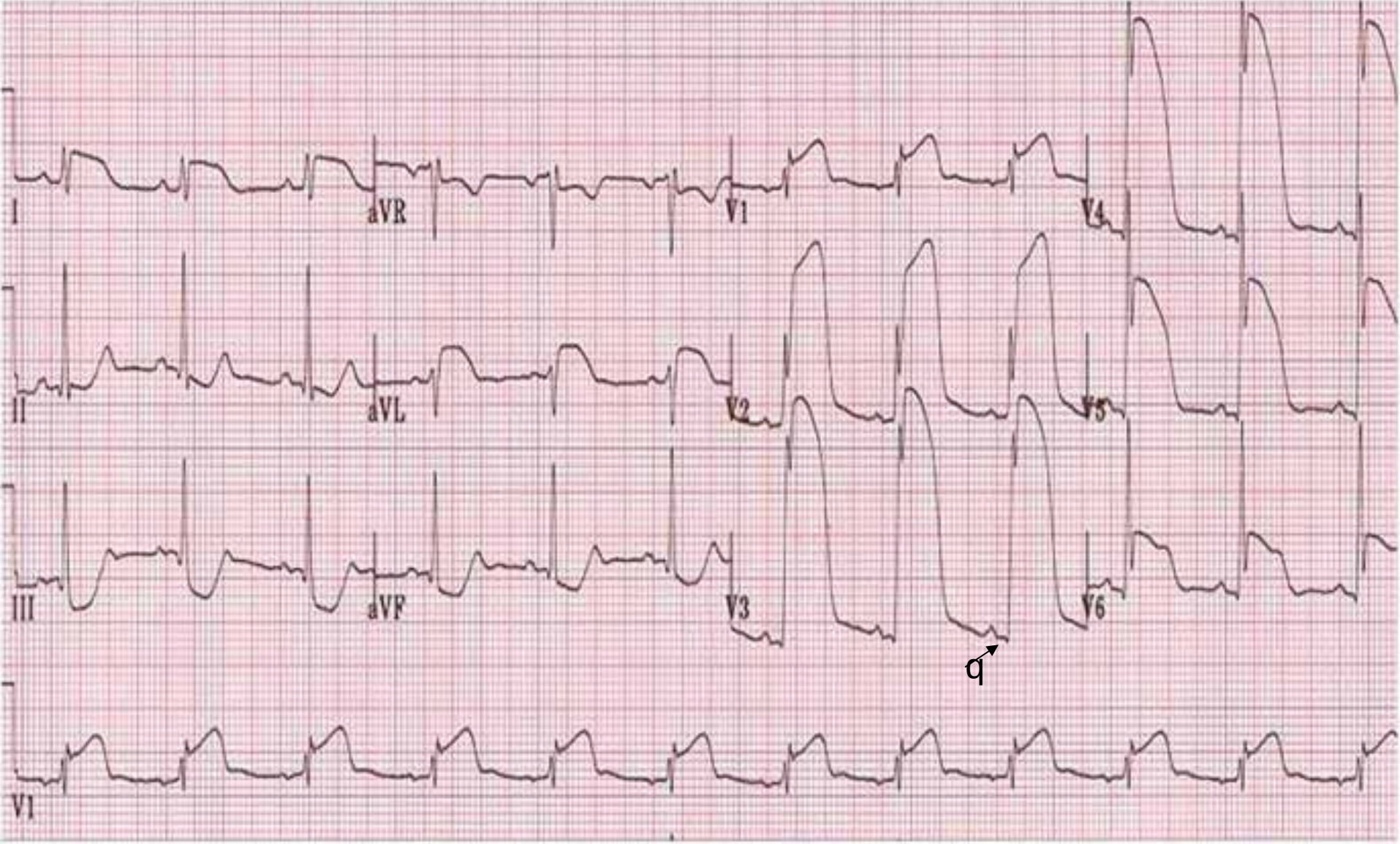
1. **Gorenek B, Cengiz O, Kudaiberdieva G, et al. Mode of onset of polymorphic ventricular tachycardia in acute myocardial infarction.Can J Cardiol. 2010 Aug-Sep; 26:e254-257.**



Source: Mirvis DM, Goldberger AL In Braunwald E, Zipes DP, Lubby P Heart Disease A TEXBOOL OF CARDIOVACULAR MEDICINE 6th edition Chapter 5 pp108 . Our ECG diagnose is different related the authors.

Another example of hyperacute phase of extensive anterolateral MI. Marked ST elevation is present across the precordial leads, as well as in leads I and aVL. The QRS complexes have shape very similar with monophasic action potential. Marked ST elevations caused by severe ischemia are sometimes referred to as a "monophasic current-of-injury pattern". ST segment depression, consistent with a reciprocal changes or mirror image, is seen in leads III, aVF and aVR. Embryonic initial q waves are present in leads V3 through V6. Increase in R wave amplitude (V2 and V3) is observed (Prominent QRS anterior forces). We believe that these prominent QRS anterior forces are consequence of left septal fascicular block (LSFB), This tracing also shows left axis deviation with small or absent inferior r waves, which raises the possibility of a prior inferior infarct associated with LAFB. (SIII>SII) (bifascicular block)

LAD artery occlusion after first perforator and before first diagonal is the location of culprit artery.



Hyperacute phase of anterolateral Myocardial Infarction: Widespread ST-elevation from V1 to V6 and I & aVL. Reciprocal changes or mirror image (ST depression) in II, III, aVF and aVR).

Paradoxical Prominent QRS Anterior Forces (PAF) consequence of left septal fascicular block Embryonic initial q wave in V3. Lesion vector directed to front, upward and leftward

Reperfusion time is a critical determinant of postinfarct ventricular electrical instability early (≤3 hours) and late after ST elevation myocardial infarction (STEMI) treated with primary percutaneous coronary intervention (pPCI) for STEMI.(1)

Resolution of ST-segment elevation in AMI has been widely used as a surrogate for treatment success. A recent randomized study suggested that after pPCI, the prognostic significance of ST resolution may have been overemphasized.

Wong et al (2) in a systematic review to discern if ST resolution achieved via pPCI has a different meaning to that achieved via fibrinolysis. The authors concluded that ST resolution after different reperfusion therapies has similar prognostic meaning.

The ST-segment resolution at 90 min was more complete after pPCI, suggesting better epicardial and microvascular reperfusion, whereas no difference between treatment strategies was seen at 4 h. The ST-segment resolution at 4 h correlated with decreased mortality, but increased reinfarction rates among patients receiving fibrinolytic therapy, whereas no association was seen for patients receiving pPCI. Consequently, 4-h ST-segment resolution remains an important prognosticator after fibrinolysis, but may be overemphasized as a surrogate end point after pPCI.(2)

1. Kumar S, Sivagangabalan G, Thiagalingam A, et al. Effect of reperfusion time on inducible ventricular tachycardia early and spontaneous ventricular arrhythmias late after ST elevation myocardial infarction treated with primary percutaneous coronary intervention. Heart Rhythm.2011 Apr;8:493-449.
2. Wong CK, de la Barra SL, Herbison P. Does ST resolution achieved via different reperfusion strategies (fibrinolysis vs percutaneous coronary intervention) have different prognostic meaning in ST-elevation myocardial infarction? A systematic review. Am Heart J. 2010 Nov;160:842-848.
3. Sejersten M, Valeur N, Grande P, et al DANAMI-2 Investigators. Long-term prognostic value of ST-segment resolution in patients treated with fibrinolysis or primary percutaneous coronary intervention results from the DANAMI-2 (DANish trial in acute myocardial infarction-2). J Am Coll Cardiol. 2009 Nov 3;54:1763-1769.

Advances in electrocardiography and enzymology in the 1940s and 1950s have provided better knowledge of the clinical evolution of MI and recognition of the prognostic relevance of acute phase arrhythmias. This prompted the creation of intensive coronary care units in the subsequent decade. After the successful resolution of acute phase arrhythmias, it became clear that the myocardium necrotic area size was a determining factor in the long-term prognosis. The Killip-Kimball clinical classification in the 60s helped to clarify the role of infarct size on LV dysfunction, from Class I to Class IV with major necrosis, (involving more than 30% of the LV free wall area, the majority of these being fatal). Along with these advances, a series of experimental studies have shown that myocardial ischemia depends on the oxygen supply-demand imbalance, highlighting the factors affecting oxygen consumption. The study of various physiological, pharmacological or mechanical interventions on these factors became the next step towards optimizing the supply-demand relation. Several animal experiments were conducted in the 1970s, followed by the first clinical studies to reduce infarct size, particularly by increasing the oxygen supply either with fibrinolytic agents or with pPCA. The clinical experience of coronary reperfusion showed that LV function did not normalize in 30% of the patients. In spite of unblocking the epicardial vessel, demonstrated hemodynamically, no equivalent myocardial perfusion was observed in these studies. New concepts emerged such as reperfusion injury, coronary microvascular dysfunction or small vessel disease, "no-reflow" phenomenon, stunned myocardium, and hibernating myocardium, which have become the target of basic research and clinical investigation. The replication of these phenomena in experimental models has attempted on the one hand to improve characterization with the use of different technologies, e.g. contrast echocardiography, isotopic studies including positron tomography, and magnetic resonance. On the other hand it has tested new therapeutic approaches as adjuvants of coronary reperfusion. Reperfusion injury is responsible for 50% of infarct size, so it became the target of research on cardiac protection. Post-reperfusion arrhythmias, stunned myocardium, microvascular obstruction that translates into the "no-reflow" phenomenon, are reperfusion injury manifestations. Imaging technology developments made it possible to demonstrate that microvascular obstruction occurs in 40% of patients who underwent pPCI. Several therapeutic approaches to prevent microembolization have been studied such as glycoprotein IIb/IIIa receptor blockers. Ischemic myocardium conditioning is one of the new strategies to reduce reperfusion injury.

The concept of pre-conditioning, defined experimentally in 1986, establishes that multiple brief episodes of ischemia may protect the heart from a subsequent prolonged infarction. Several observations have proved that pre-conditioning occurs in cardiac patients, for example, during coronary angioplasty and coronary bypass graft surgery, and so it is regarded as a promising approach to reducing infarct size. The concept of pre-conditioning was then enlarged by the demonstration, experimentally, that producing ischemia in a vascular bed could induce pre-conditioning in another vascular bed. Ischemia resulting from repeated successive insufflations of a blood pressure cuff on a lim, reduces myocardial necrosis after coronary angioplasty or coronary bypass graft surgery. This remote pre-conditioning seems to be a safe and effective non-invasive way of reducing the reperfusion injury. In 2002 a hypothesis was tested in studies on dogs that multiple repeated episodes of ischemia, produced in the beginning of reperfusion, would attenuate the reperfusion injury. This technique, called post-conditioning, was first used in patients in 2005, in AMI reperfusion, with beneficial short and long-term results. In the last 15 years, a large number of clinical studies have been carried with different pharmacologic groups to explore association pre- and post-conditioning concepts. Four agents were studied in particular: adenosine, nicorandil, atrial natriuretic peptide, and statins. The most important studies are reviewed, calling attention to disparities in results and discussing possible causes of negative outcomes. Cyclosporine, recently tested, opens a new field of investigation since it inhibits mitochondrial permeability and may directly attenuate the reperfusion injury. Microvascular dysfunction occurs in many patients after coronary angioplasty and is caused, in the first place, by distal embolization. The purpose of thrombectomy is to reduce the probability of distal embolization during angioplasty and stent placement. Available devices for clinical use include thrombus aspiration and thrombectomy catheters. Initial studies did not have the expected impact, but the number of patients studied was limited. A recent series involving more than 1000 AMI patients undergoing coronary angioplasty and thrombus aspiration has shown an improvement of myocardial perfusion indices and a reduction of mortality at 30 days. Distal embolization protection systems aim to prevent the embolic material entering the circulation and causing macro-or microembolization. The present small and controversial experience does not yet recommend the routine use of this technique. Reduction of infarct size has been the main objective of research on ischemic myocardial disease during the last 40 years.

Myocardial reperfusion is a major accomplishment in this field. But it is like a double-edged sword because reperfusion injury significantly reduces the potential benefits of reperfusion. The huge amount of research undertaken in the past 20 years constitutes a paradigm of the relationship between experimental work and clinical practice, and has improved the prospects for diminishing infarct size, in both the short and long-term.

1. Reperfusion injury: is the tissue damage caused when blood supply returns to the tissue after a period of ischemia or lack of oxygen. The absence of oxygen and nutrients from blood during the ischemic period creates a condition in which the restoration of circulation results in inflammation and oxidative damage through the induction of oxidative stress rather than restoration of normal function. Cardiology Myocardial injury caused by rapid flow of blood into areas previously rendered ischaemic by coronary artery occlusion. Reperfusion injury is attributed to oxidative stress, which may cause arrhythmia, infarction, myocardial stunning
2. Coronary microvascular dysfunction (CMVD) or small vessel disease Small vessel disease, also known as coronary microvascular disease or small vessel heart disease, is a condition in which the small arteries in the heart become narrowed. Small vessel disease causes signs and symptoms of heart disease, such as chest pain (angina). Small vessel disease is usually diagnosed after a doctor checks for blockages in the main arteries of the heart that cause coronary artery disease, but finds little or no narrowing in the large vessels even though your symptoms persist. Although anyone can have small vessel disease, it's more common in women and in people who have diabetes. Small vessel disease is treatable, but can be difficult to detect. Positive exercise stress test (EST) after elective successful pPCI consistently reflects impairment of hyperemic coronary blood flow (CBF) due to CMVD, which persists over a follow-up period of 6 months.(1)

1. Milo M, Nerla R, Tarzia P, et al. Coronary microvascular dysfunction after elective percutaneous coronary intervention: Correlation with exercise stress test results.Int J Cardiol. 2012 Oct 8. pii: S0167-5273(12)01165-5. doi: 10.1016/j.ijcard.2012.09.059. [Epub ahead of print]

3. "No-reflow" phenomenon: When a coronary artery is occluded, detrimental changes occur in the cardiac capillaries and arterioles. After relief of the occlusion, blood flow to the ischemic tissue may still be impeded, a phenomenon known as no reflow. Understanding the pathophysiology of the no-reflow phenomenon is the key for managing this condition. After prolonged cessation of coronary occlusion and restoration of blood flow to the epicardial coronary arteries, there is sufficient structural damage to the microvasculature to prevent restoration of normal blood flow to the cardiomyocytes. This may lead to inadequate healing of the cardiac scar. In addition, it may prevent the development of future collateral flow. This phenomenon appears to be more pronounced in the subendocardium in a manner similar to the wavefront phenomenon of the ischemic cardiac death.(1) It is more pronounced with longer periods of coronary occlusions. (2) No reflow appears to be a process rather than an immediate event that occurs at the moment of reperfusion. Experimental studies showed that the no-reflow area increases with time after reperfusion.(3) Although it is clear that abnormalities at the level of the microvasculature caused the no-reflow phenomenon, the exact mechanism is uncertain; a variety of factors probably contribute to it. Microscopic examination showed that the cardiac cells within the no-reflow area were swollen. The capillary endothelium was damaged and exhibited areas of regional swelling with large intraluminal protrusions that in some cases appeared to plug the capillary lumen.(2)

1. ***Reimer KA, Lowe JE, Rasmussen MM, et al. The wavefront phenomenon of ischemic cell death, 1: myocardial infarct size vs duration of coronary occlusion in dogs. Circulation 1977;56: 786-794 .***
2. ***Kloner RA, Ganote CE, Jennings RB. The "no-reflow" phenomenon after temporary coronary occlusion in the dog. J Clin Invest 1974:54: 1496–1508.***
3. ***Kloner RA. Does reperfusion injury exist in humans? Am J Cardiol 1993;21: 537–545.***

Cellular edema compressing the capillaries was confirmed experimentaly.(1) This may explain the occasional benefit noted with dexamethasone(2) or mannitol.(1)Cell contracture in the ischemic zone also may contribute to the microvascular compression. (3) Intravascular plugging by fibrin or platelets may also contribute to the no-reflow phenomenon.(5) Beneficial effects of ibuprofen,**(6)** prostaglandin E1,**(7)** and vascular washout with heparinized saline**(7)** support the concept that these blood elements may be important. In a no-reflow model of a New Zealand white rabbit study by Golino et al,**(8)** platelet depletion markedly reduced the extent of no-reflow zones. Leukocyte intravascular plugging appears to play an important role in the pathophysiology of no reflow. Engler et al**(9)** showed that the no-reflow areas had evidence of capillary leukocyte plugging. Although there was no difference in no-reflow zones between the neutropenic animals and the control group in a gerbil cerebral ischemia model(10)

1. *Manciet LH, Poole DC, McDonagh PF, et al. Microvascular compression during myocardial ischemia: mechanistic basis for no-reflow phenomenon. . Am J Physiol; 1994:266 : H11541–H1550*
2. *Nellis SH, Roberts BH, Kinney EL, et al. Beneficial effect of dexamethasone on the "no reflow" phenomenon in canine myocardium. Cardiovasc Res. 1980 Mar;14:137-141.*
3. *Carlson RE, Aisen AM, Buda AJ. Effect of reduction in myocardial edema on myocardial blood flow and ventricular function after coronary reperfusion. Am J Physiol. 1992 Mar;262:H641-648.*
4. *Diaz RJ, Wilson GJ. Studying ischemic preconditioning in isolated cardiomyocyte models. Cardiovasc Res. 2006 May 1;70:286-96.*
5. *Gavin JB, Thomson RW, Humphrey SM, et al. Changes in vascular morphology associated with the no-reflow phenomenon in ischaemic myocardium. Virchows Arch A Pathol Anat Histopathol. 1983;399:325-332.*
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Byrne et al**(1)** found that reperfusion with leukocyte-depleted blood may reduce cardiac no reflow. Furthermore, in a rat model of irreversible hemorrhagic shock, the no-reflow phenomenon was prevented by rendering the animals neutropenic. Leukocytes may interfere with blood flow by mechanical plugging and perhaps by their release of oxygen free radicals that will add further injury to the capillary endothelium.Thus, the no-reflow phenomenon is likely multifactorial. During the ischemic phase, endothelial damage, including endothelial swelling and myocyte edema, led to initial no-reflow zones. With reperfusion, additional edema, myocyte contraction, platelets, fibrin, and leukocyte plugging resulted in expansion of the no-reflow zones over the early hours of reperfusion. Platelet and leukocyte depletion and vasodilators appeared to lessen no reflow.

Diminished flow through the microvasculature compared with normal zones is usually referred to as “low reflow.”An additional mechanism plays a very important role during short-term intervention in AMI. Microemboli of atherosclerotic debris, blood clots, and platelet plugs are released into the microcirculation, particularly with restoration of normal blood flow by thrombolysis, angioplasty, stenting, or other PCI. Although this is more common in vein graft intervention, it is to be expected in native coronary arteries. A variety of new, innovative devices are now in clinical practice and in the research phase to filter these microemboli during the interventional procedure.

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4. Stunned myocardium: It is a condition of impaired myocardial contractile function, cellular biochemical characteristics, and microvasculature function in the absence of gross myocardial necrosis. It can last for minutes to days and is caused by ischemia that is either brief or occurs in the area immediately outside an infarct zone. Transient–hrs to days in duration postischemic contraction defects that follow myocardial reperfusion in acute MI.
5. Hibernating myocardium Cardiology Regional dysfunction of myocardial tissue due to prolonged local hypoperfusion, which is completely reversible upon restoration of adequate blood flow; hibernation occurs in Pts with CAD and impairment of LV function at rest Hibernating myocardium is defined as persistently impaired myocardial and LV function at rest resulting from reduced myocardial blood flow. It is postulated that despite the reduced coronary blood flow, metabolic activity is sufficient to prevent tissue necrosis. Recovery of the hibernating myocardium has clearly been shown to occur with the establishment of successful revascularization either by coronary bypass surgery or by percutaneous transluminal coronary angioplasty. The differentiation of viable, hibernating myocardium from non-viable myocardium in patients with coronary artery disease and left ventricular dysfunction is a key issue in the current era of myocardial revascularization.

In patients with ischemic heart disease detection of myocardial viability is of major clinical and prognostic importance and may significantly affect therapeutic decisions. Reversible LV dysfunction may be due to different pathophysiological mechanisms, including myocardial hibernation and stunning, structural and ultrastructural myocardial changes and alterations in gene expression leading to myocardial cell dedifferentiation. Each of these mechanisms may have different importance related to the clinical history of the patient and severity and duration of LV dysfunction and may significantly influence the extent and time course of functional recovery after myocardial revascularization. Several imaging techniques are available for the assessment of viable myocardium, based on the detection of preserved perfusion, preserved glucose metabolism, intact cell membrane and mitochondria, and presence of contractile reserve. Nuclear cardiology techniques, dobutamine echocardiography and positron emission tomography are used to assess myocardial viability. In recent years, new advances have improved methods of detecting myocardial viability.

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(1).

SYNONYMOUS: Non-paroxysmal VT, slow ventricular tachycardia(2), ventricular rhythm with isorrhythm, benevolent rhythm(3)

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.

1. Freire G, Dubrow I. Accelerated Idioventricular Rhythm in Newborns: A Worrisome But Benign Entity with or without Congenital Heart Disease. Pediat Cardiol. 2008; 29: 457-462.
2. Leitz N, Khawaja Z, Been M. Slow ventricular tachycardia. BMJ. 2008 Jul 3; 337:a424.
3. Martinez-Lopez JI. ECG of the month. Benevolent rhythm. Escape impulses; escape rhythm. J La State Med Soc. 1993 Jun;145:249-252.

POSSIBLES ETIOLOGIES

1. *Acute phase of myocardial infarction (MI): present in 15% of the cases(1).*
   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)(2)*
4. *During inhalational induction with halothane(3)*
5. *Associated to ophthalmic timolol/dorzolamide solution(4)*
6. *After aconite poisoning(5)*
7. *Associated with desflurane administration(6)*
8. *Electrolyte imbalance: Extreme hyperkalemia (K+ > 10. mmol/l)(7) and hypokalemia.*

1. Chiladakis JA, Pashalis A, Patsouras N, et.al. Autonomic patterns preceding and following accelerated idioventricular rhythm in acute myocardial infarction. Cardiology.2001;96:24-31.
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4. Attanasio A, Baglio S, Quatrana M, et. al. Accelerated idioventricular rhythm associated to ophthalmic timolol/dorzolamide solution. Int J Cardiol. 2004 Jun;95:343-345.
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7. Kes P, Orlić-Cunović D, Trubelja N. A life-threatening complication of extreme hyperkalemia in a patient on maintenance hemodialysis. Acta Med Croatica. 1995; 49:147-150.

POSSIBLES ETIOLOGIES

9. *No underlying heart disease*
   - *In young patients and in newborns(1)*
   - *Hypervagotonia in highly conditioned athletes(2).*
   - *During the antenatal period(3)*
10. *Coronary artery dissection(4)*
11. *Congenital diseases(5)*
12. *Primary cardiomyopathies(6)*
13. *Post-resuscitation(7)*
14. *Hypertensive heart disease(8)*

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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(1).

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(2).

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.

1. **Osmancik PP, Stros P, Herman D. In-hospital arrhythmias in patients with acute myocardial infarction - the relation to the reperfusion strategy and their prognostic impact. Acute Card Care. 2008;10:15-25.**
2. **Bonnemeier H, Ortak J, Wiegand UK, et al. Accelerated idioventricular rhythm in the post-thrombolytic era: incidence, prognostic implications, and modulating mechanisms after direct percutaneous coronary intervention. Ann Noninvasive Electrocardiol. 2005 Apr; 10:179-187.**

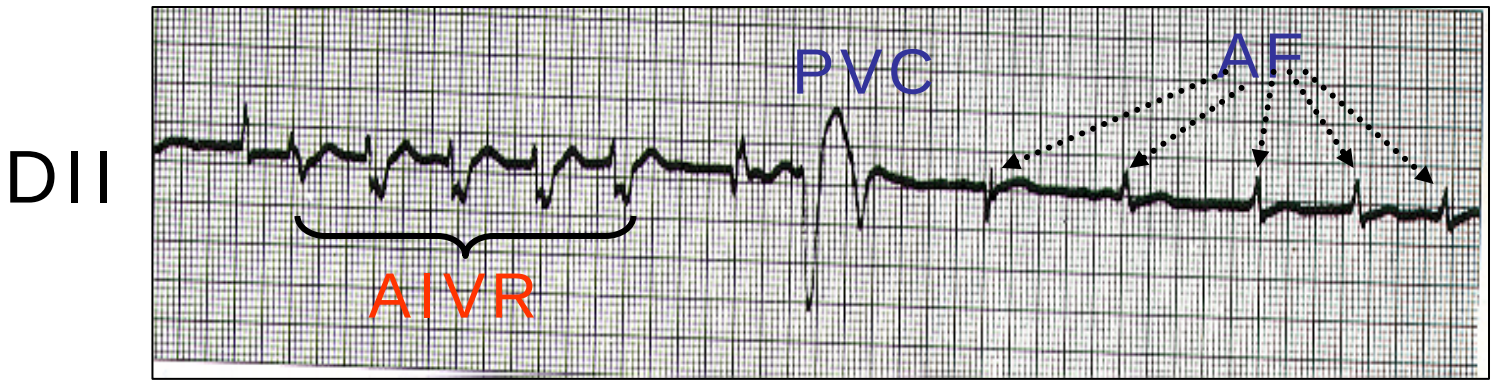
AIVR ELECTROPHYSIOLOGICAL MECHANISMS

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 rate 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.

ECG CHARACTERIZATION

1. Duration of QRS complex: ≥120 ms
2. Constant and bizarre morphology of QRS complexes (monomorphic)
3. Slow rate: between 50 bpm and 130 bpm (usually between 70 and 85 bpm)
4. Regular or almost regular R-R
5. Event SAQRS different from basic rhythm SÂQRS
6. 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
7. Depressed sinoatrial activity, with frequent absence of P wave
8. AV dissociation: 70% of the cases
9. Frequent fusion beats at the onset and the end of the event
10. Capture and fusion beats, much more frequent than in paroxysmal VT
11. Frequent coexistence with extrasystolic VT in its unstable form

AIVR: ACCELERATED IDIOVENTRICULAR RHYTHM OR SLOW VT



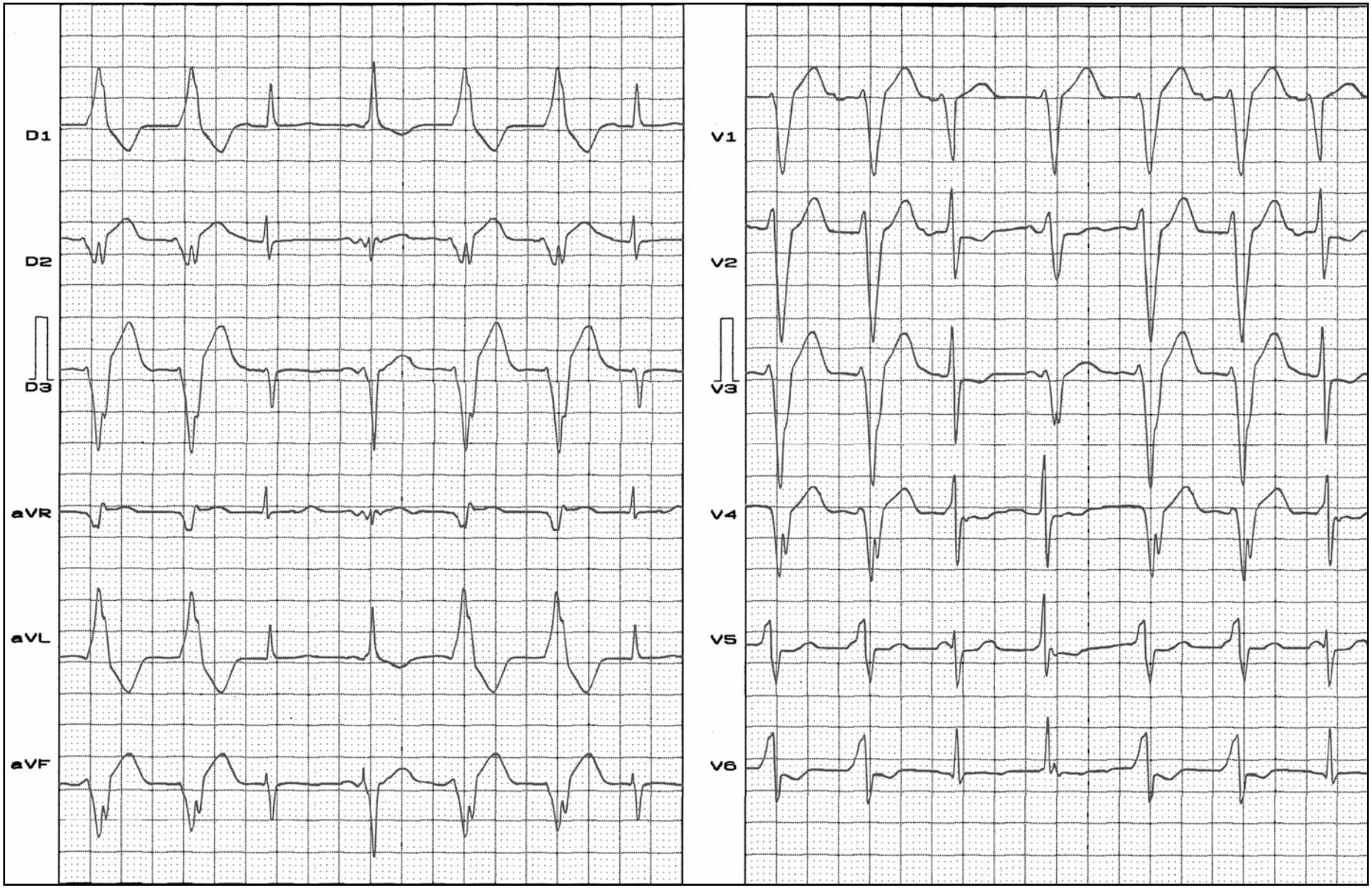
Elderly patient, 82 years old, myocardiosclerotic. Digoxin 0.25 mg/day for quite some time. Absence of P wave, f waves: atrial fibrillation.

From the third beat to the sixth, wide and regular QRS with rate of 110 bpm: AIVR: Accelerated IdioVentricular Rhythm.

The eighth beat is a premature ventricular contraction (PVC).

In such case, AIVR indicates digitalis intoxication. The dose of serum digoxin was 3 ng/mL. The levels above 2.5 ng/mL in adults are considered to be toxic.

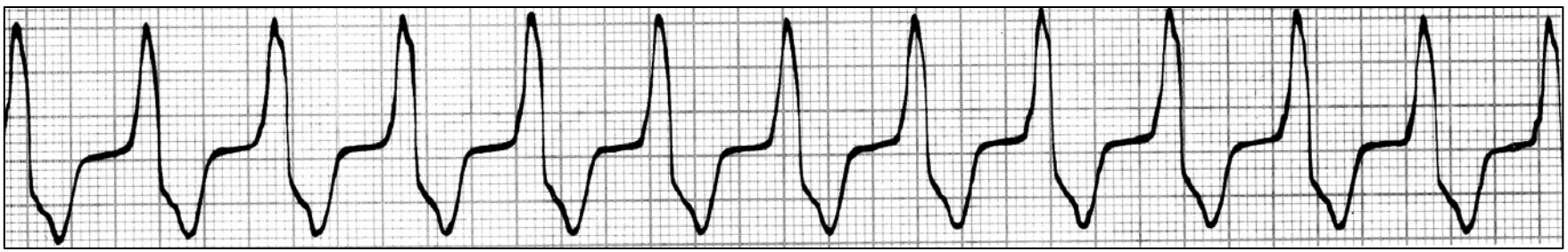
Example of AIVR in digitalis intoxication.



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.

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.