Class 5 – SOLAECE Course

ECG in Athletes and Its Role in the Prevention of Sudden Cardiac Death

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The author does not report any conflict of interest regarding this presentation

Study methodology in detecting patients in risk

- ✓ **Periodical evaluations:** every 2 years.

- ✓ **I) Personal and family clinical history**

- ✓ **II) Physical examination: mandatory for any candidate.**

- ✓ **III) Non-invasive supplementary tests:** (Only in selected cases? Without consensus among USA and European researchers.)

Study methodology for screening candidates to athletes in risk.

I) Personal and familial clinical history

1) Ask questions about SCD in first-degree relatives under 45 years old;
2) Ask questions about the knowledge in the family about HCM, LQTS, Marfan-type somatic habit, sindactily, etc;
3) Personal history of murmur in childhood;
4) Personal history of dizziness, syncope, palpitations, intolerance to exercise, precordialgia, dyspnea, etc.
5) Dizziness or syncope during or after exercise, may indicate the presence of: HCM, dromotropic disorder, MVP, aortic stenosis or arrhythmia;
6) Precordialgia intra- or post-strain may indicate early coronary atherosclerosis;
7) Excessive/progressive dyspnea may indicate valvular diseases, pulmonary disease, or structural anomalies;
8) Palpitations during or after exercise may be a sign of arrhythmia.
9) Ask questions about the current or past use of legal (tobacco, alcohol) and illegal drugs;
10) Recent history of virus infection may lead to symptoms compatible to myocarditis;
11) History of congenital heart disease or cardiac surgery;
12) Any background that may imply greater risk for congenital heart disease. E.g.: Maternal rubella, exposition to toxics used or environmental.

Significance of personal and familial clinical history.

II) Pre-participation physical examination

1) Anthropometrical evaluation: weight, height, BP and percentage of body fat;

2) Identification and characterization (intensity, location and time of cycle) of murmurs and arrhythmias, standing and in supine position;

3) Recognize phenotypes: e.g. Marfand, Noonan and Holt-Oram syndrome, supravalvular aortic stenosis, Williams syndrome.

4) Measurement of BP in superior and inferior limbs, and assessment of femoral, radial and foot pulses to exclude Aorta coarctation.

5) Auscultation must be performed in decubitus and standing to identify murmurs influenced by dynamic obstruction in the LV outflow tract; detection of extracardiac clicks and sounds;

6) Musculo-skeletal aptitude. Try to detect medical conditions or skeletal muscles that may predispose to injuries or diseases during a competition.

Value of physical examination.

III) Non-invasive methods in pre-participation evaluation in young candidates to athletes

✓ Although they may increase the diagnostic power of history and physical examination, they are not advised as a routine "screening";

✓ The three most used are: ECG, echocardiogram and ergometer test;

✓ Other specific tests include: Holter, transesophageal cardiostimulation, high resolution ECG, Tilt-Test, Looper, RR variability, QT dispersion, microvolt T wave alternans, etc.

Non-invasive supplementary tests

1) Eletrocardiogram;

2) Ergometer test: it is an electric recording of the heart while the organism is undergoing physical stress. The most used ergometers are the bicycle ergometer and the treadmill. It should be performed in all preventive medicine check-ups, but regrettably, it is not so. But it is convenient for all athletes or at least sportsmen with a regular activity, to do this test. Only thus it is possible to know how the heart behaves regarding physical strain.

   In sedentary people older than 30 years old, who begin a program of exercises, it should be mandatory. It is known that 70% of people with chest angina and who hadn't experienced myocardial infarction, present rest ECG without abnormalities, and that many myocardial ischemias appear in a strain test even before the person has displayed any symptom.

3) M-module, two-dimensional echocardiogram with transthoracic doppler.

ECG and exercise stress test value.

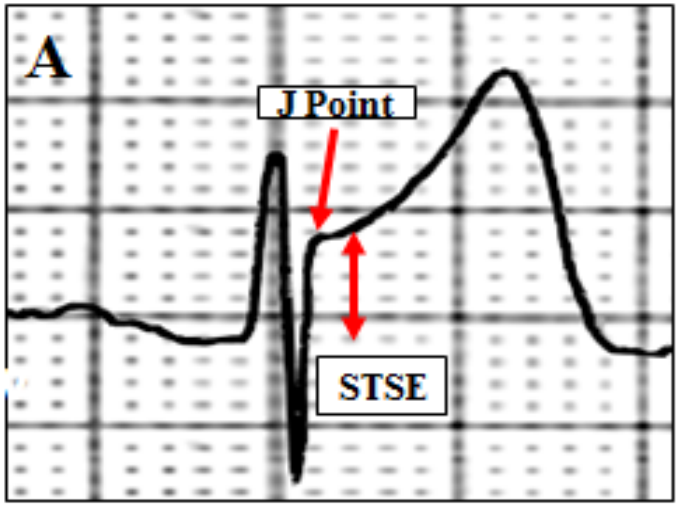
Common and uncommon ECG abnormalities in athletes (Corrado 2007)

#Group 1 common ECG abnormalities	##Group 2 uncommon ECG abnormalities
Sinus bradycardia: > 50% of cases	Left atrial enlargement
Phasic Sinus Arrhythmia or respiratory sinus arrhythmia	Extreme QRS left axis deviation on frontal plane; LAFB type
AF in endurance sports practice	Right QRS axis deviation on frontal plane LPFB type
First-degree AV block. Observed in 5% to 30% of cases (in non athletes, 0.65%).	Ventricular pre-excitation
Second-degree AV block type I or Wenckebach: 10% (in non athletes <1 in 30,000 or 0.003%), and it disappears during exercise and atropine.	Complete RBBB and LBBB pattern
Notched QRS in ascending ramp of S wave in V1 or incomplete RBBB in 15% of athletes	Pathological Q wave ≥40 ms
Benign Early Repolarization Pattern	Malignant Early Repolarization Pattern
Isolated voltage criteria for LVH/LVE	Long or short QT intervals
Possible pattern of RVH/RVE: RV1+SV5 >10.5 mm between 18% and 69% of the cases.	Inverted T waves in 2 consecutives leads
	ST segment elevation convex to the top in right precordial leads followed by negative T wave.

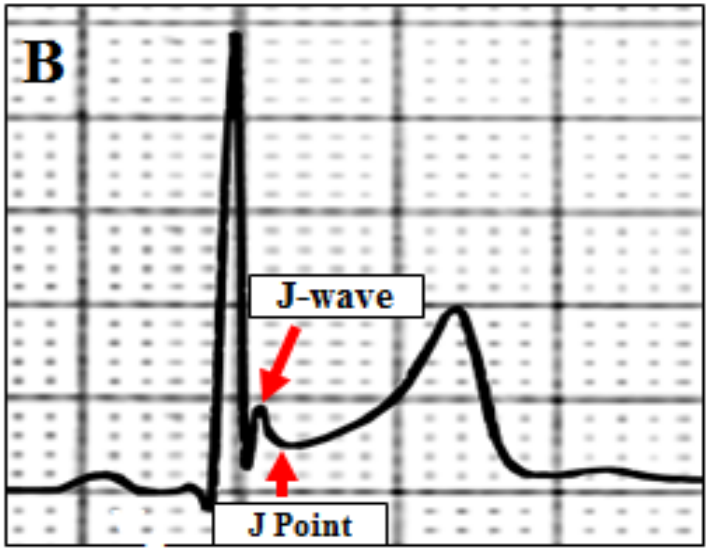
#: Training-related no increase
Training-unrelated, increased cardiovascular risk. Required additional testing <4 of cases.

Benign Early Repolarization Pattern
versus
Malignant Early Repolarization Pattern:
How to recognize the murderer easily?

A and B - Classical definition of early repolarization pattern: ST segment elevation

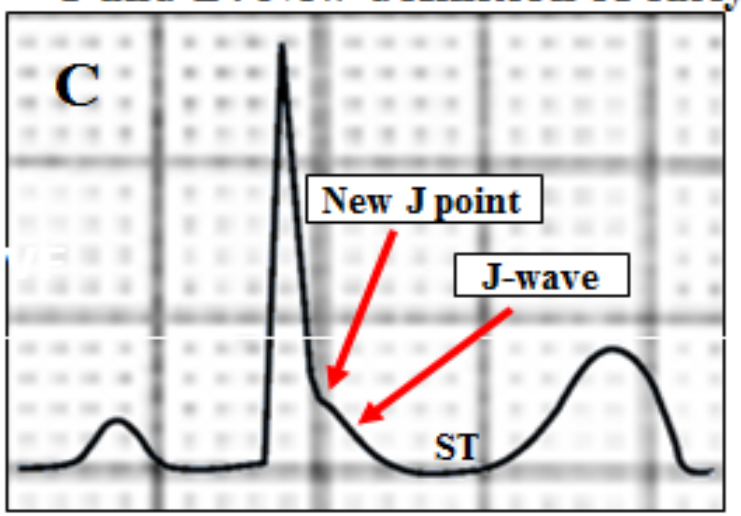


Without J-wave and STSE

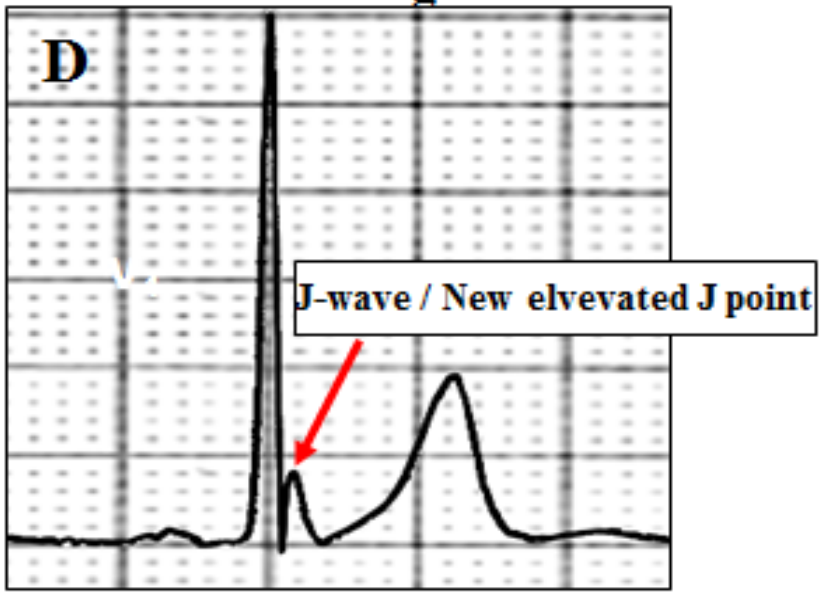


With J-wave and STSE

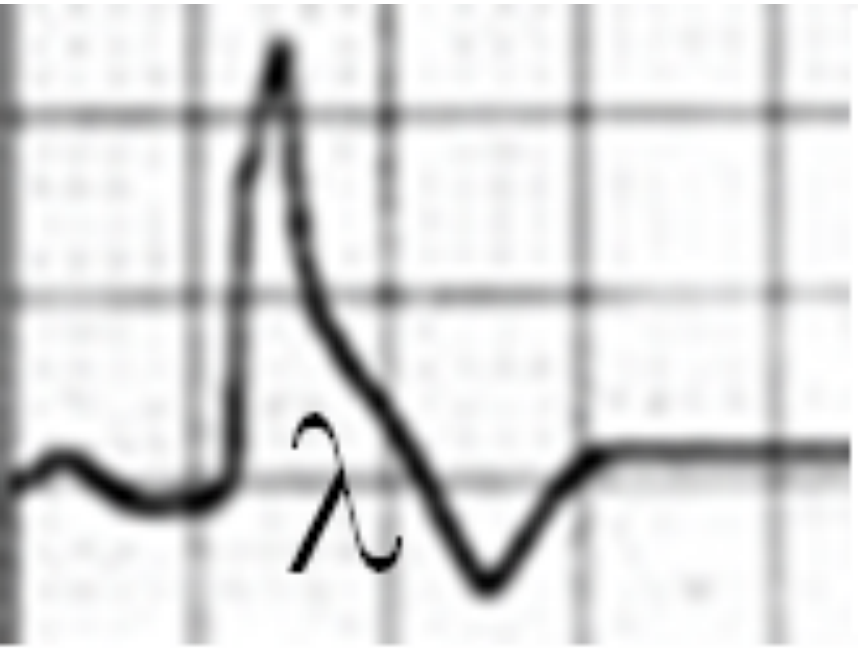
C and D: New definition of early repolarization: without ST segment elevation



J-wave after P point without STSE



J-wave or new elevated J point without STSE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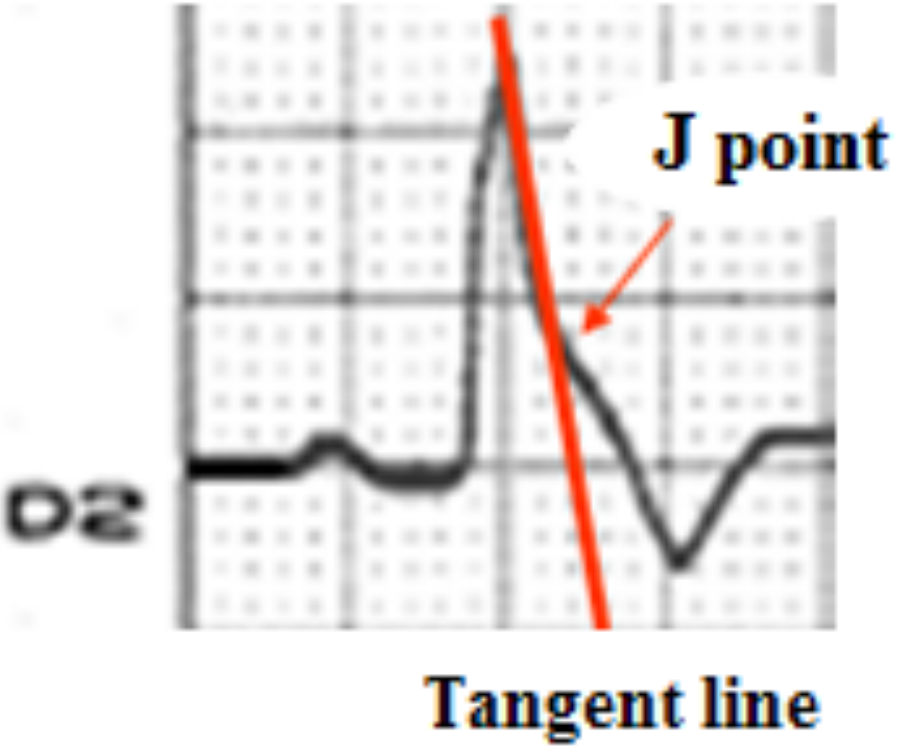
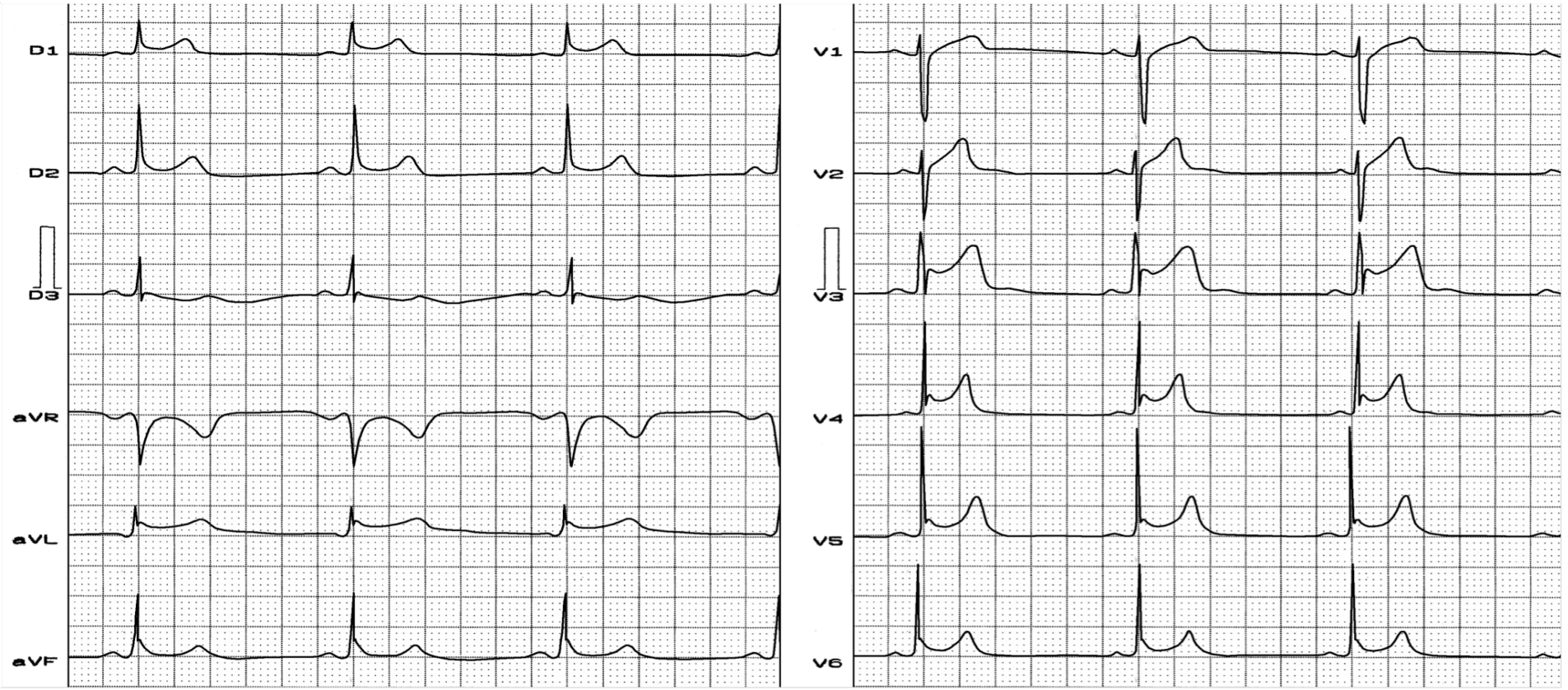
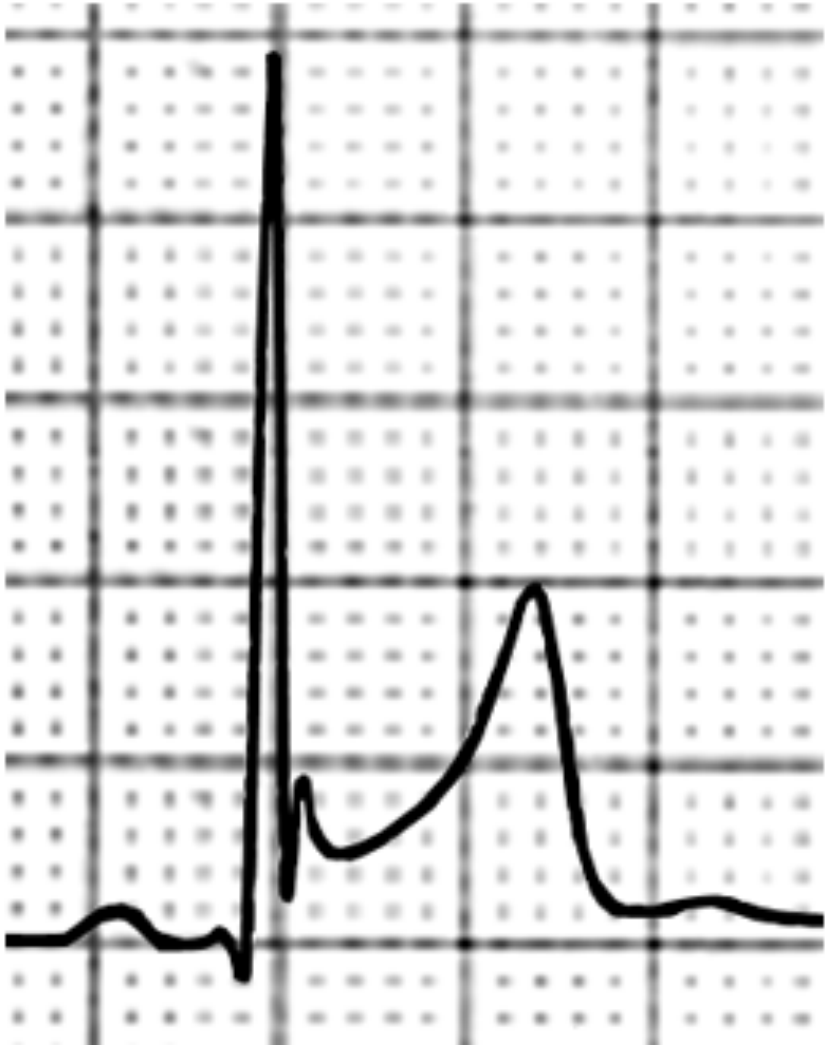

J point
Tangent line

Name: DAS **Age:** 24 y.o. **Sex:** Male **Race:** Black **Weight:** 82 kg
Height: 1.91 m **Biotype:** Athletic **Profession:** professional basketball player



ECG diagnosis: sinus bradycardia (HR 50 bpm). J point and ST segment with elevation >4 mm in precordial leads from V_3-v_5 of superior concavity. Notch or slurring of terminal portion of the QRS complex (J point). ST segment elevation >4 mm in precordial leads V_3, V_4 and V_5.
Conclusion: sinus bradycardia, early repolarization syndrome.

Typical ECG of early repolarization syndrome in an athlete with bradycardia.



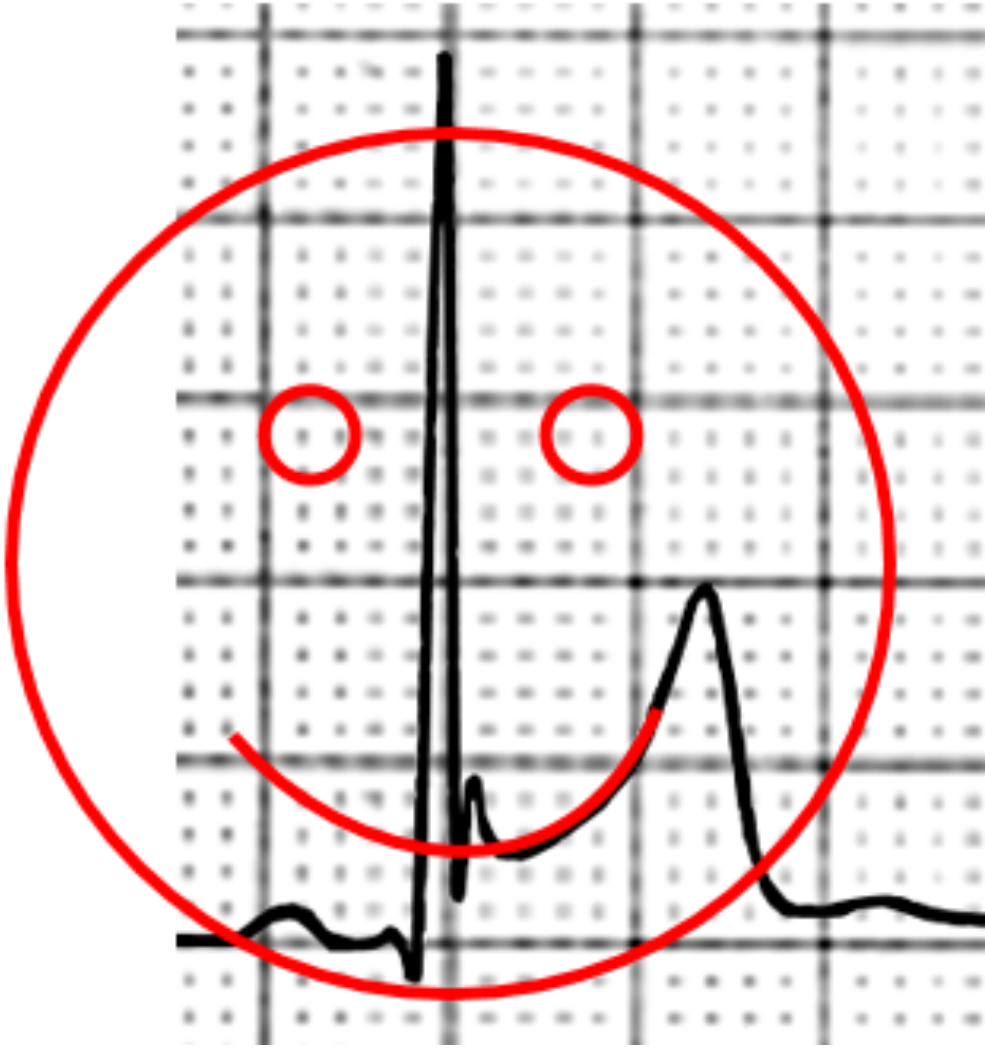
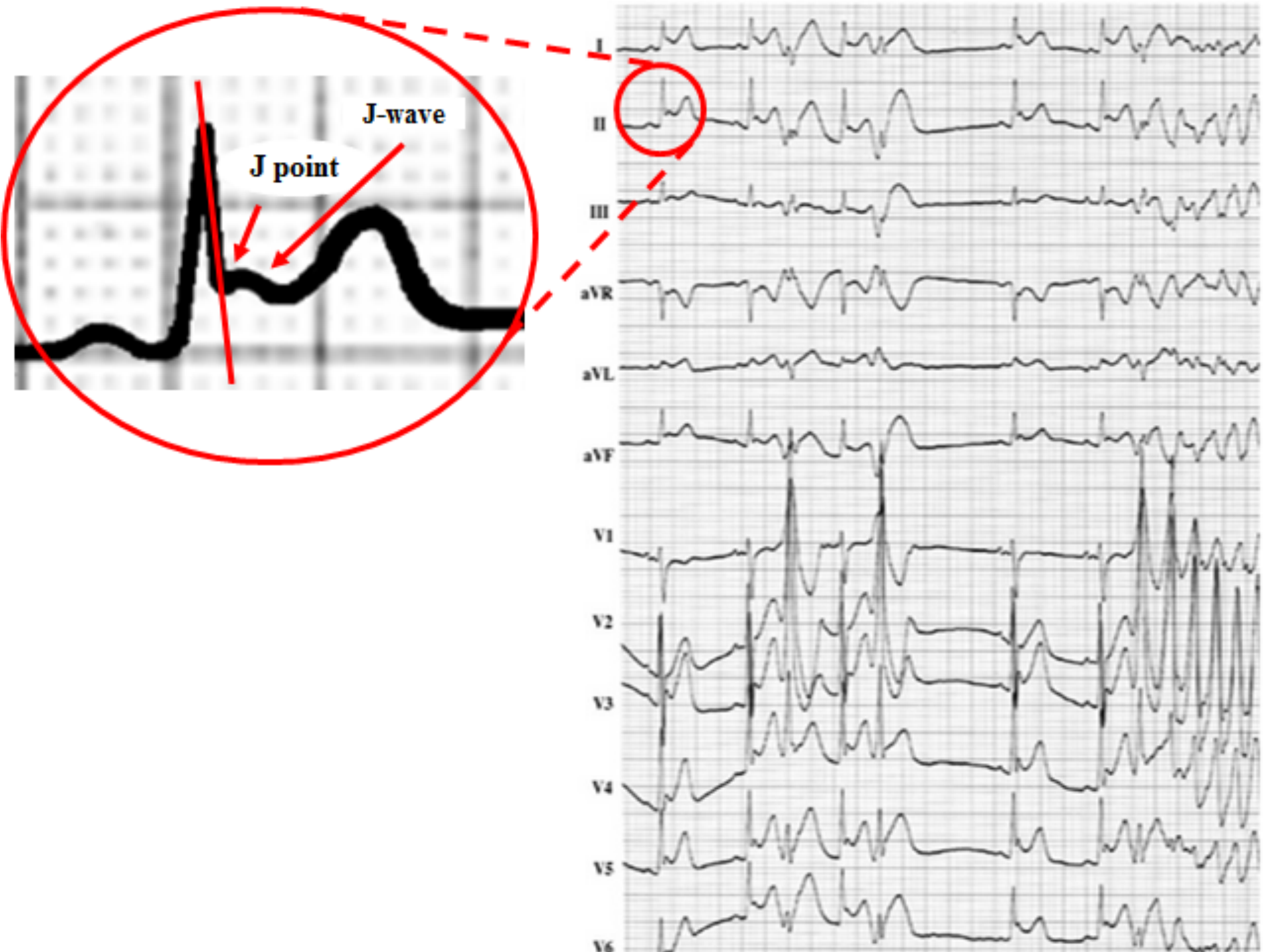
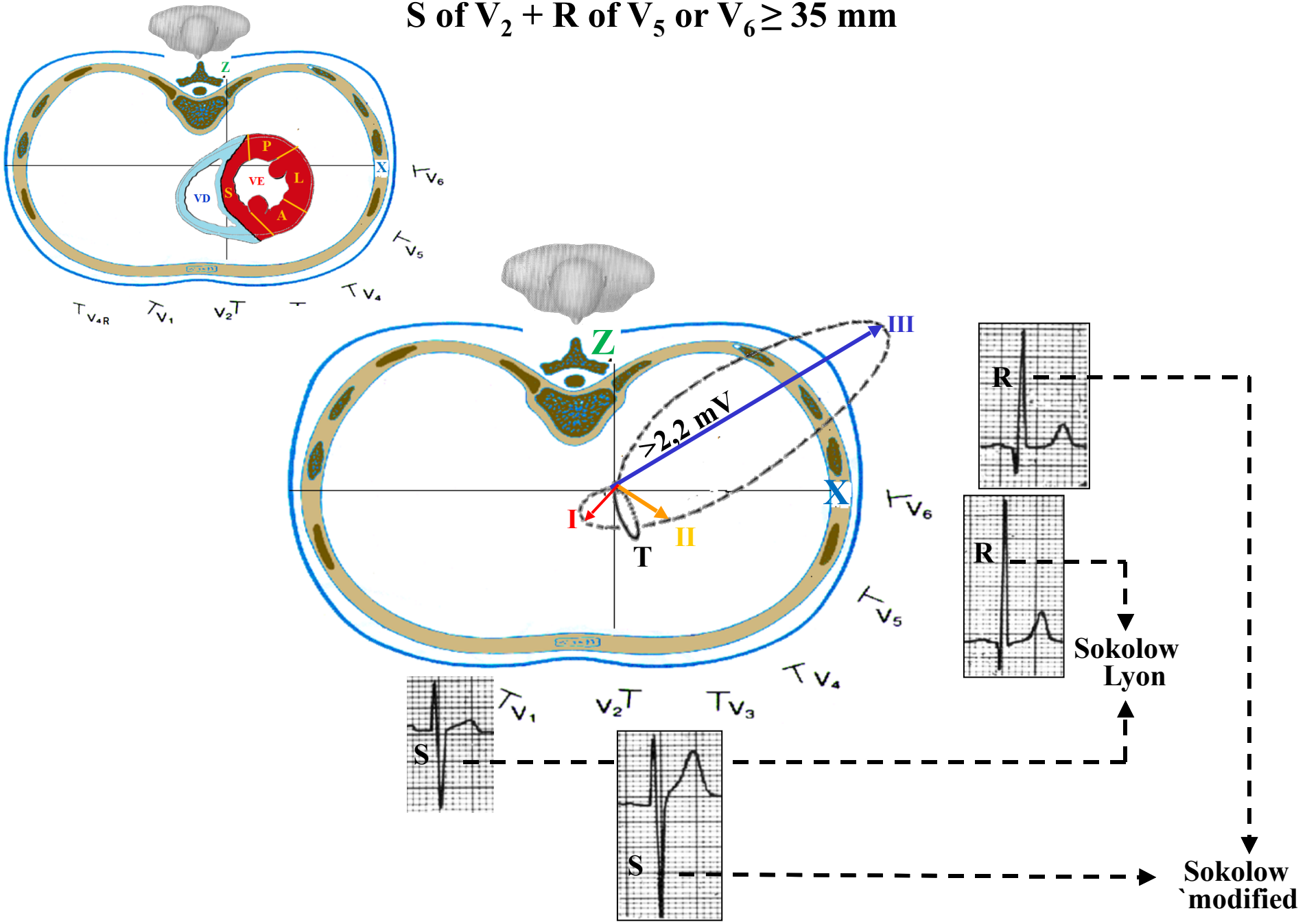


Figure 1 shows in lead V4, ST segment of “upper concavity”, followed by wide positive T wave, resembling a “smiley”.


J-wave
J point
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

Voltage criterion for LVE: Sokolow-Lyon index modified for LVE

S of V_2 + R of V_5 or $V_6 \geq 35$ mm



Most frequent characteristics of ECG in athletes

1. **1) Rhythm:**
   - Sinus, junctional or rarely ventricular.
   - Variable pacemaker or rhythm of left atrium.
   - Junctional rhythm is present in 0.31% (in the general population in 0.02%).
   - Phasic or respiratory sinus arrhythmia: present in 60% (in the non-athlete population in 2.4%).
   - Long sinus pauses: they are frequent (> 2 seconds);
2. **Heart rate:**
   - Sinus bradycardia in more than 50% of the cases.
   - HR of 30 to 40 bpm in rest are not rare.
   - In highly trained athletes, there are descriptions of HR of 25 bpm. **Etiology:** vagal hypertone; decrease in resting sympathetic tone; and intrinsic component of bradycardia;
3. **P Wave:**
   - Increase of voltage and notches are described;
4. **PR Interval:**
   - 1st degree AV block: 5% and 30% (in non athletes, 0.65%). When the PR interval does not reach the value as a criterion for 1st degree AV block, it is relatively prolonged. The PR interval normalizes or even gets smaller after exercise;
   - 2nd degree AV block:
   - a) Mobitz Type I or Wenckebach: it is observed in 10% (in non athletes <1 in 30,000 or 0.003%), and it disappears invariably during exercise and atropine;
   - b) Mobitz Type II;
   - AV dissociation;
   - Complete or 3rd degree AV block: 5 each 12,000 athletes.

5. **QRS:** SAQRS: tendency to vertical position; possible presence of voltage criteria for LVH: SV1+RV5 > 35 mm (Sokolow and Lyon index); possible pattern of RVH: RV1+SV5 >10.5 mm between 18% and 69% of the cases. RVH manifests by a diastolic pattern translated by minimal degrees of IRBBB. IRBBB is observed in 15% of athletes; absence of progression of increase of voltage of r or R wave with QR pattern from V1 to V3: pattern of pseudo-infarction in anterior wall. Pattern of CRBBB: observed in 13.5% of the cases;
6. **Ventricular repolarization: ST segment**

   Pattern of early repolarization variant; it is described in four patterns:
   - J point and ST segment elevation followed by peaked T wave from V4 to V6 and in the inferior wall (2.4% to 44%);
   - J point and ST segment depression (rare);
   - J point and ST segment elevation followed by inverted T wave;
   - Disappearance of ST segment elevation after exercise.
7. **Ventricular repolarization: T wave:** Juvenile pattern of T wave. Inverted and asymmetrical T wave in left leads: I, aVL, V5 and V6, secondary to physiologic LVE. Negative or biphasic T waves from V1 to V3 and/or in the inferior wall. Frequent "normalization" of T wave before strain. This type of response is not observed in hypertrophic cardiomyopathy or in coronary insufficiency. Myocardial perfusion imaging associated to exercise stress test always negative. Characteristic reversion of ECG "alterations" in cases of interruption of competitive activity
8. **U wave:** After the T wave, a rounded deflection is observed mostly in precordial leads V3 and V4, called U wave. Usually, it has the same polarity as T wave. It is always, in normal cases, positive in D1, D2 and from V2 through V6. Its amplitude is inversely proportional to HR, being greater in athletes with bradycardia, and smaller in children with tachycardia.

12. Junctional rhythm.

13. Atrial fibrillation (AF) and atrial flutter are facilitated by atrial remodeling, atrial ectopy, and an imbalance of the autonomic nervous system. Endurance sports practice has an impact on all of these factors and may therefore act as a promoter of these arrhythmias. In an animal model, long-term intensive exercise training induced fibrosis in both atria and increased susceptibility to AF. While the prevalence of AF is low in young competitive athletes, it increases substantially in the aging athlete, which is possibly associated with an accumulation of lifetime training hours and participation in competitions. A recent meta-analysis revealed a 5-fold increased risk of AF in middle-aged endurance athletes with a striking male predominance. Beside physical activity, height and absolute left atrial size are independent risk factors for lone AF and the stature of men per se may explain part of their higher risk of AF. Furthermore, for a comparable amount of training volume and performance, male non-elite athletes exhibit a higher blood pressure at rest and peak exercise, a more concentric type of left ventricular remodeling, and an altered diastolic function, possibly contributing to a more pronounced atrial remodeling. The sports cardiologist should be aware of the distinctive features of AF in athletes. Therapeutic recommendations should be given in close cooperation with an electrophysiologist. Reduction of training volume is often not desired and drug therapy not well tolerated. An early ablation strategy may be appropriate for some athletes with an impaired physical performance, especially when continuation of competitive activity is intended.

Atrial Fibrillaton in Athletes

Lone atrial fibrillaton: AF in absence of any clinical evidence of cardiac or extra cardiac factor, such as latent hypertension, coronary artery disease, chronic obstructive lung disease, valvular disease, cardiomyopathy, diabetes, hyperthyroidism. obesity, metabolic syndrome, sleep apnea, alcohol consumption, endurance sports, anger, hostility, subclinical atherosclerosis, inflammation. etc

≤ 60 years AF is exceptional in children, uncommon in young adults, but 3 to 4% of subjects over 60 years of age.

Possibilites

Long-term endurance exercise

Athlete men with AF

The proportion of sportsmen among patients with lone AF is much higher than that reported in the general population

1. lower incidence of mild hypertension
2. Vagally mediated AF

Echocardiogram: greater atrial and ventricular dimensions; higher ventricular mass. (**Mont 2002**)

Idiopatic, cryptogenetic, essencial, primary truly lone AF

30% of patients with paroxysmal AF

It is the effect of an underlying, 'masked' disorder.

It has a favourable prognosis

Embolic risk of 2 to 5% per year.

Genetic causes; familial genetic predisposition

Epidemiological studies have provided unequivocal evidence that arrhythmia has a substantial heritable component.

Autosomal dominant pattern,

1. Mutations in K+ channel genes: congenital SQTS mutations *KCNE2*, *KCNJ2*, and *KCNQ1*
2. Common AF associated to 4q25 polymorphisms (**Ritchie 2012**)
3. Brugada syndrome
4. Sinus node dysfunction (see next slide)

Atrial Fibrillation genetic mutations background: Mechanistic Subclassification of Lone Atrial Fibrillation (Robert 2010)

AF subclassification	Culprit Gene	Functional effect
Enhanced atrial action potential repolarization	KCNQ1 KCNE2 KCNJ2 (Xia 2005) KCNE5 (Rayn 2008)	Enhanced slow component of the delayed rectifier K+ current (Iks). Enhanced KCNQ1 (Das 2009; Chen 2003; Lundby 2007) - KCNE2 K+ (Yang 2004) current . Enhanced inward rectifier current (I_{k1}) Enhabced I_{ks}
Delayed atrial action potential repolarization	KCNA5 SCN5A(Makiyama 2008; Watanabe 2009; Darbar 2008)	Decreased ultrarapid component of the delayed rectifier potassium current (I_{kur}). (Yang 2009; Olson 2006) Hiperpolarizing shift in Na, 1.5 inactivation.
Conduction velocity heterogeneity	GJA5 (Gollob 2006)	Decreased Gap Junction conduction (Delmar 2000)
Cellular hyperexcitability	SCN5A (Li 2009)	Depolarizing shift in Na, 1.5 inactivation.
Hormonal modulation of atrial electrophysiology Adrenocholinergic stimulation (Yamazaki 2009)	NPPA	Increased circulating levels of mutant atrial natriuretic peptide (Hodgson-Zingman 2008)
Cholinergic	Unknown	Enhanced cholinergic sensitivity. A greater abundance of Kir3.x channels and higher $I_{(K,ACh)}$ density in LA than RA myocytes result in greater ACh-induced speeding-up of rotors in the LA than in the RA, which explains the ACh dose-dependent changes in overall AF frequency and wavelet formation (Sarmast 2003; Rudy 2004).
Locus on chromosome 10q22-q24 Gene Map locus on chromosome 4(4q25)	ATFB1 ATFB5 (Gudbjartsson 2007; Benjamin 2009; Kääb 2009; Ellionor 2010; Husser 2010)	Type 1 familial AF (Brugada R 1997)
Locus on chromosome 16q22	ZFHX3	(Gudbjartsson 2009)
ATP-binding cassette sub-family A member 1	Chromosome: 9; Location: 9q31.1	cholesterol efflux regulatory protein

Arrhythmias in the hearts of athletes and comparative incidence with the general population

Arrhythmia	General population	Athletes
Sinus Bradycardia	23.7	50-85
Phasic Sinus Arrhythmia	2.4-20	13.5-69
Atrial Variable Pacemaker	Not available	7.4-19
1st degree AV block	0.65	6-33
2nd degree AV block	<1 in 30,000 or 0.003%,	10%
Mobitz Type 1	0.003	0.125-10
Mobitz Type II	0.003	Not reported
3rd degree AV block	0.0002	0.017
Junctional Rhythm	0.06	0.31-7.0

Table comparing the incidence of arrhythmias in the general population and in athletes.

Arrhythmias observed in athletes and ECG
Most frequent characteristics of ECG

9) High resolution ECG

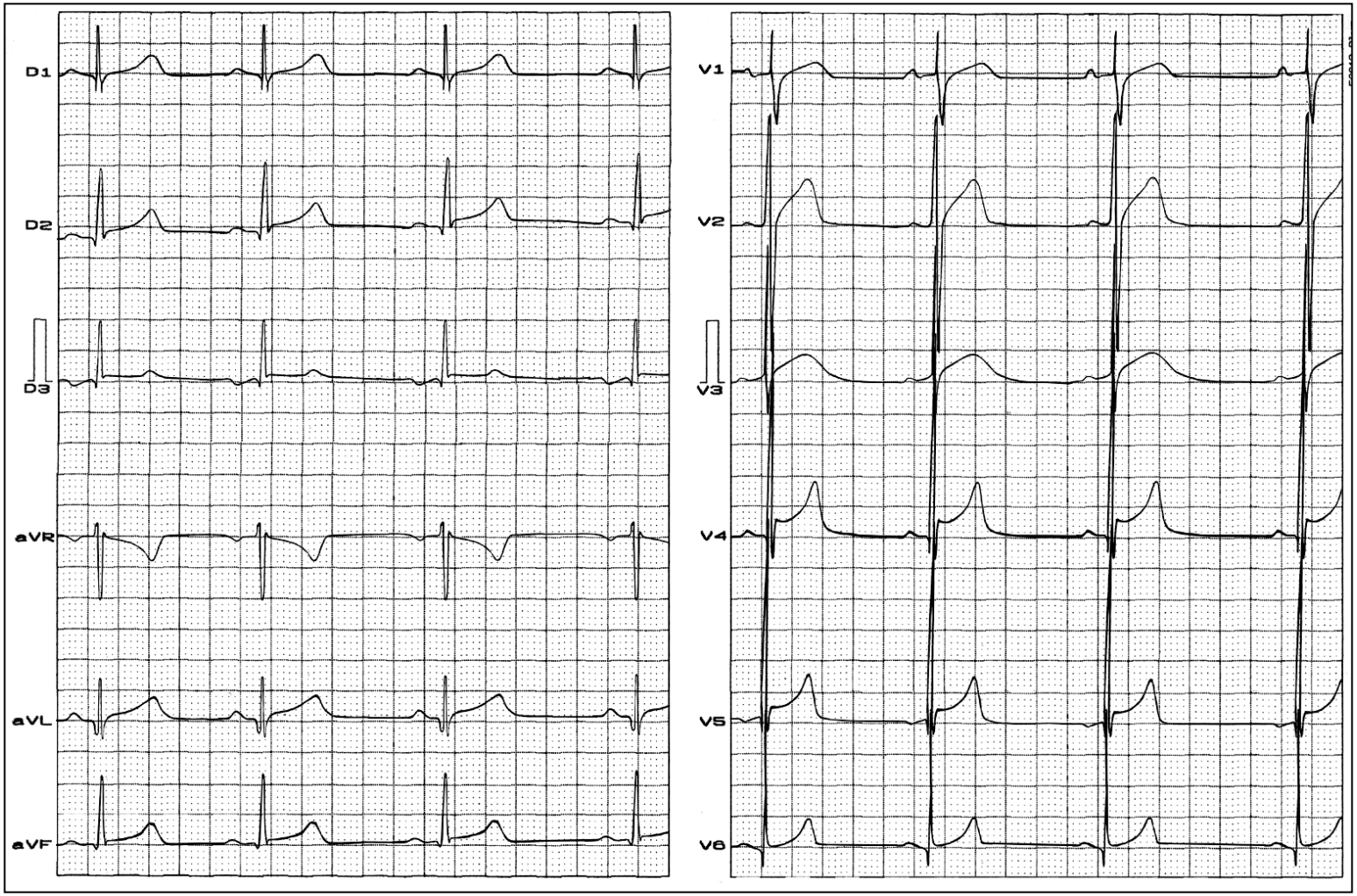
• Presence of late potentials in 10% of the cases against 1.4% in the population of athletes (**Borbola 1992**);

10) VCG

• Increase of anterior forces in almost all cases;
• Dislocation of QRS loop to the front and left in the HP;
• T loop not matching QRS loop;

High resolution ECG and VCG in athletes.

Name: ALK **Age:** 27 y.o. **Sex:** Male **Race:** White **Weight:** 70 kg
Height: 1.71 m **Biotype:** Athletic **Profession:** Athlete

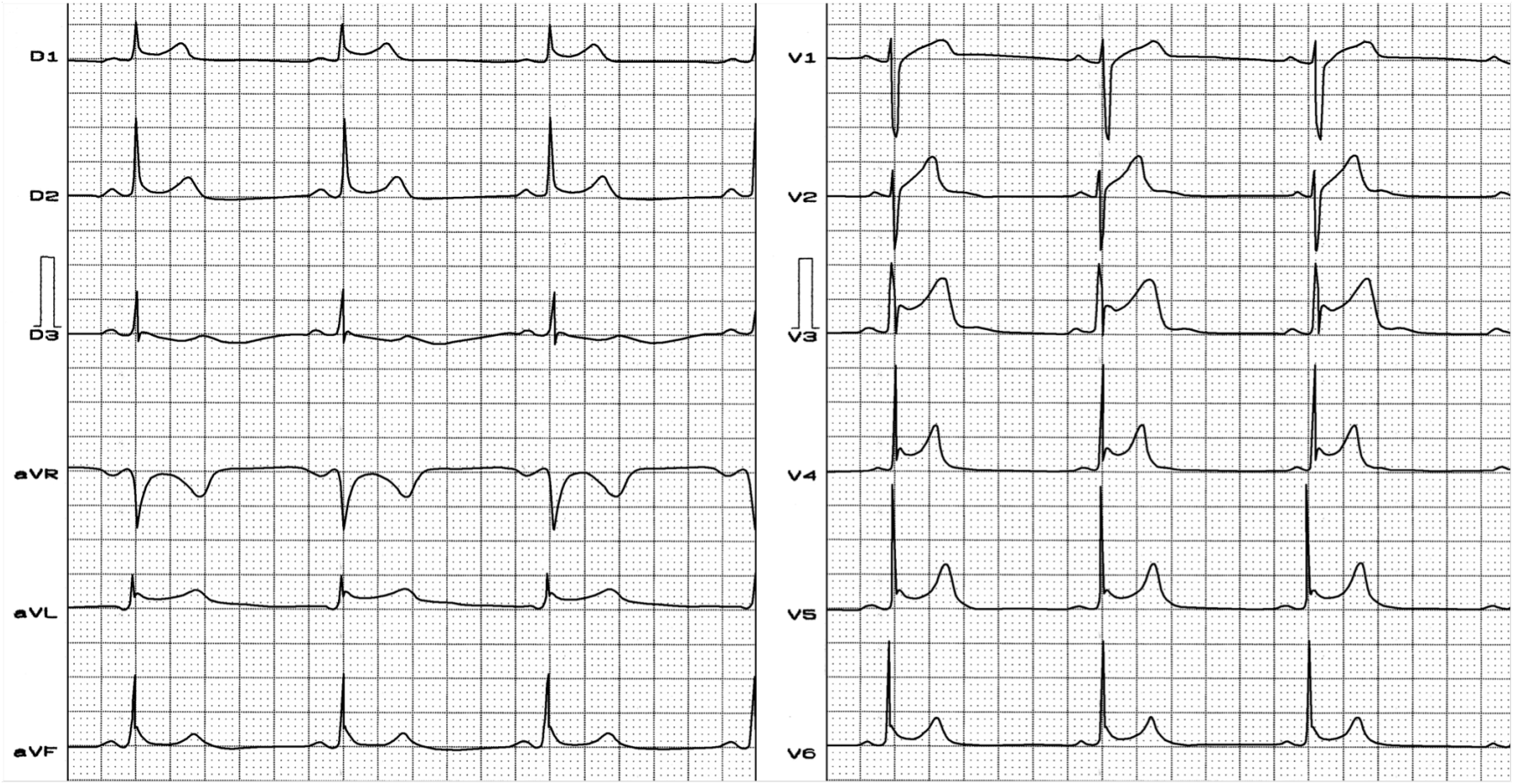


Clinical diagnosis: normal variant in elite athletes in the modality of hurdles.
ECG diagnosis: Rhythm: sinus bradycardia, phasic sinus arrhythmia. HR: between 48 and 55 bpm. Discrete ST segment elevation, upwardly convex from V4 to V6 and inferior leads. Prominent q wave in these leads.

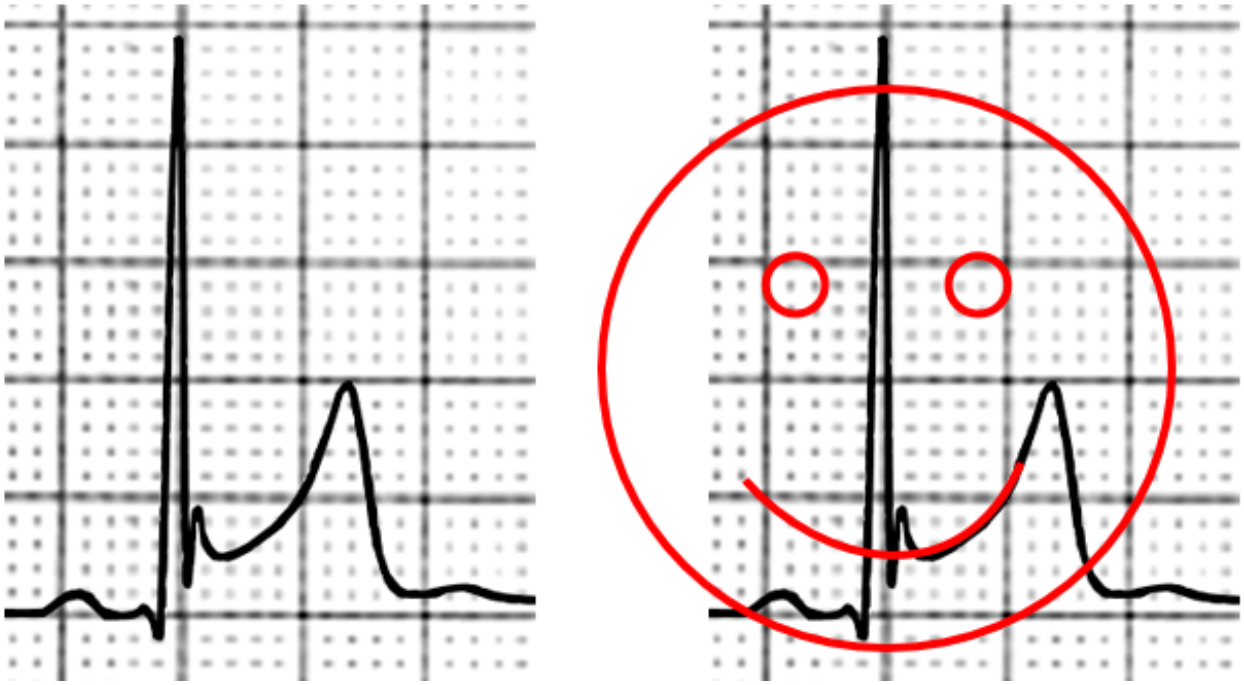
Typical ECG of an elite athlete, showing bradycardia and early repolarization syndrome.

Early repolarization pattern: ECG-VCG

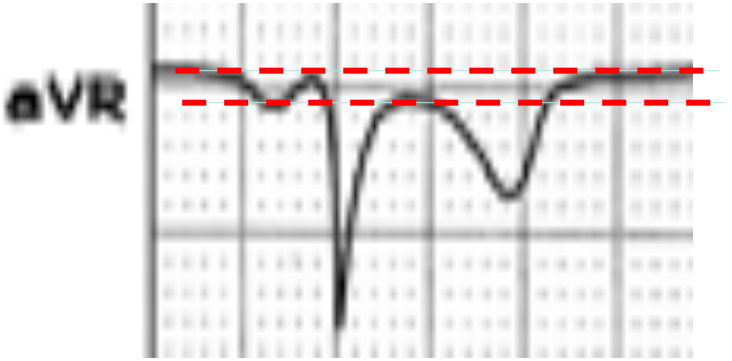
Male, 16 years old, mulatto, professional soccer player



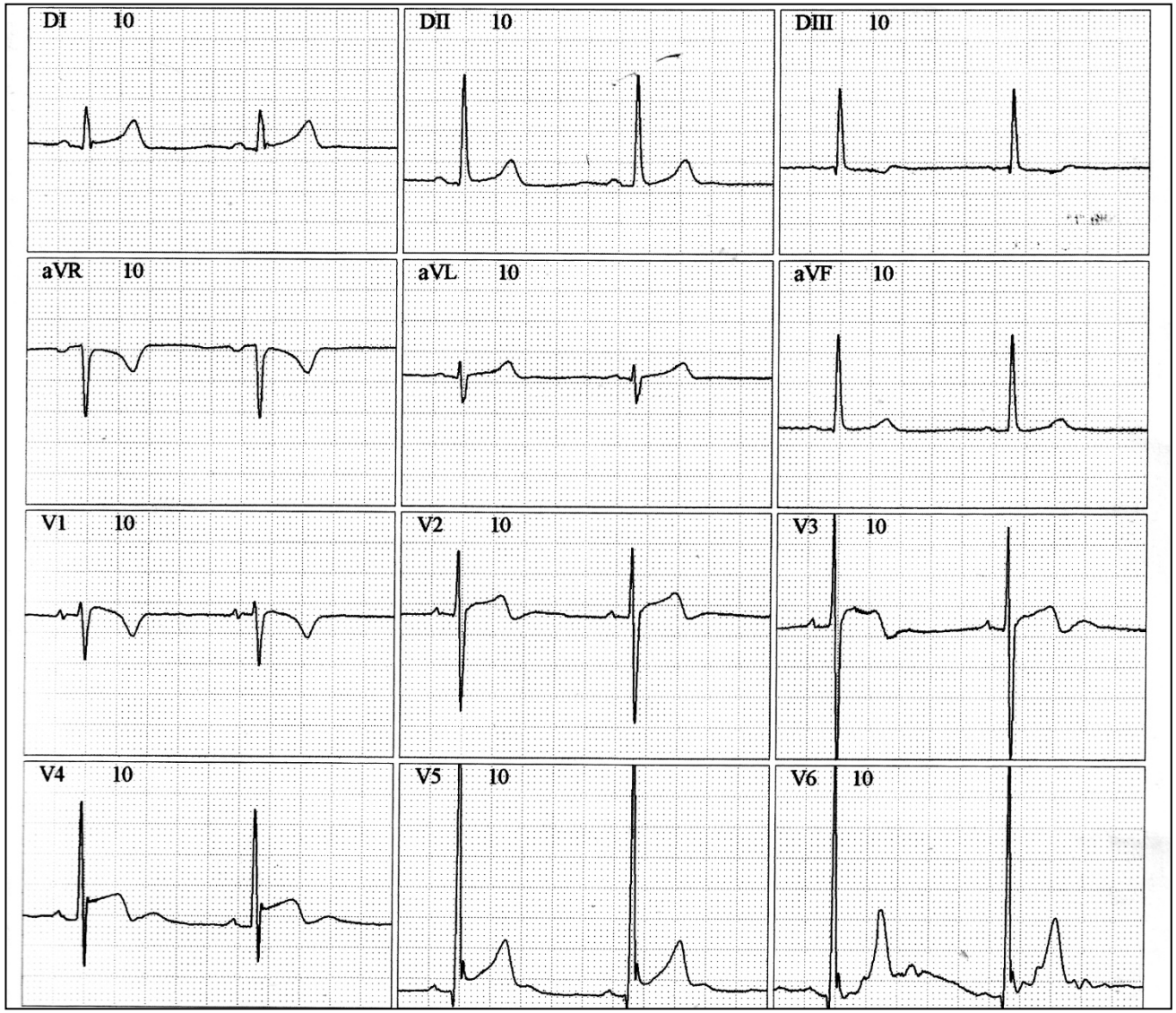
Electrocardiographic diagnosis: typical ECG pattern of benign early repolarization: sinus bradycardia, P, QRS and T directed to the same place, ST segment elevation concave to the top from V2 to V5 followed by tall pseudo-symmetrical T waves concordant with precedent polarity of QRS complexes. Absence of mirror image or reciprocal changes (with exception of aVR).



The figure shows the V4 precordial lead with STSE concave to the top followed by large positive T wave that resembles a "smiling face".



Mirror image or reciprocal changes only in the aVR lead



Clinical diagnosis: Professional soccer player with athlete's heart._

Name: VLAS **Gender:** M

Age: 16 y.o. **Ethnic group:** Mulatto

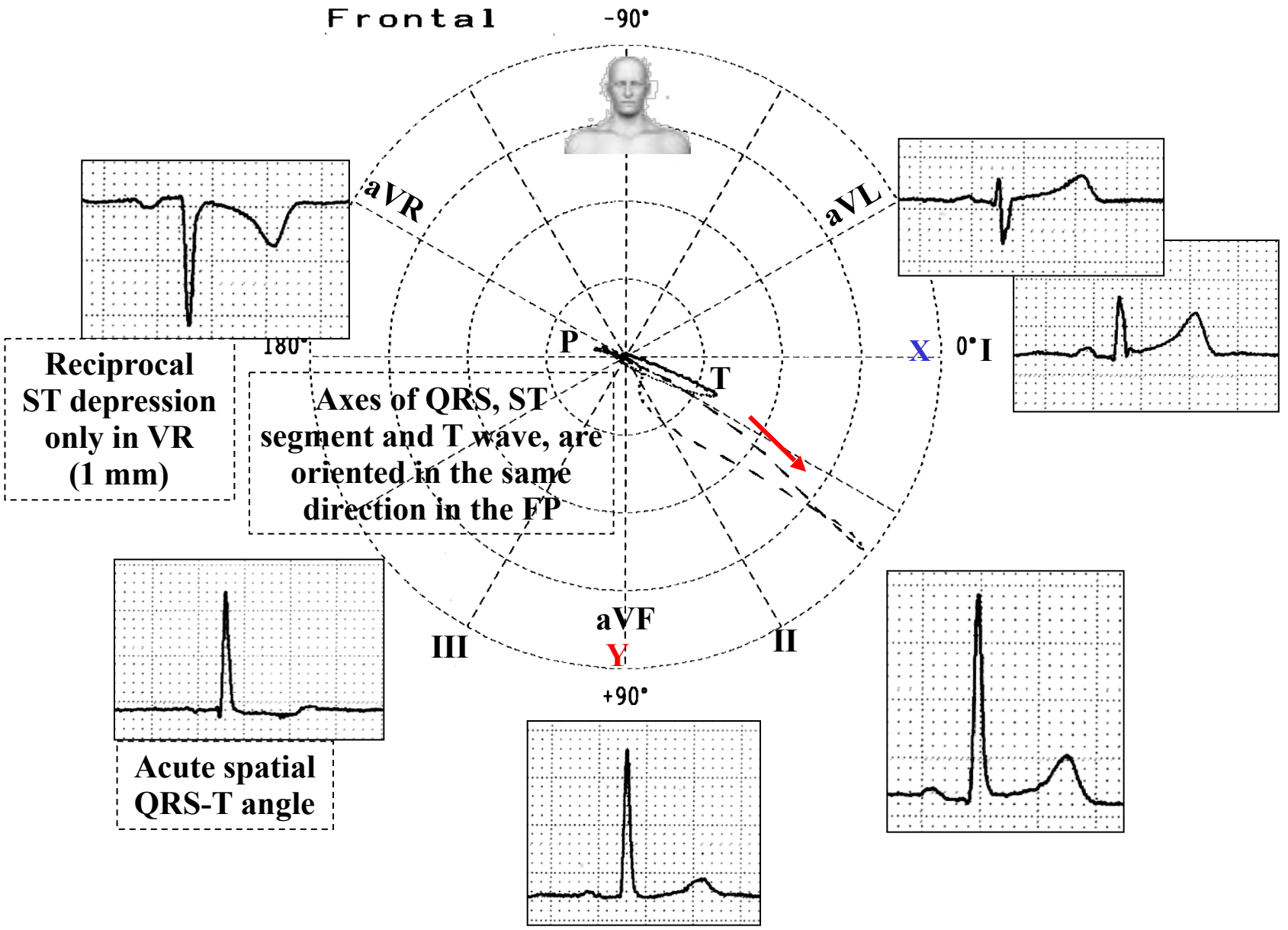
Weight: 65 Kg **Height:** 1.73

Biotype: Normal **Date:** 04/11/2008

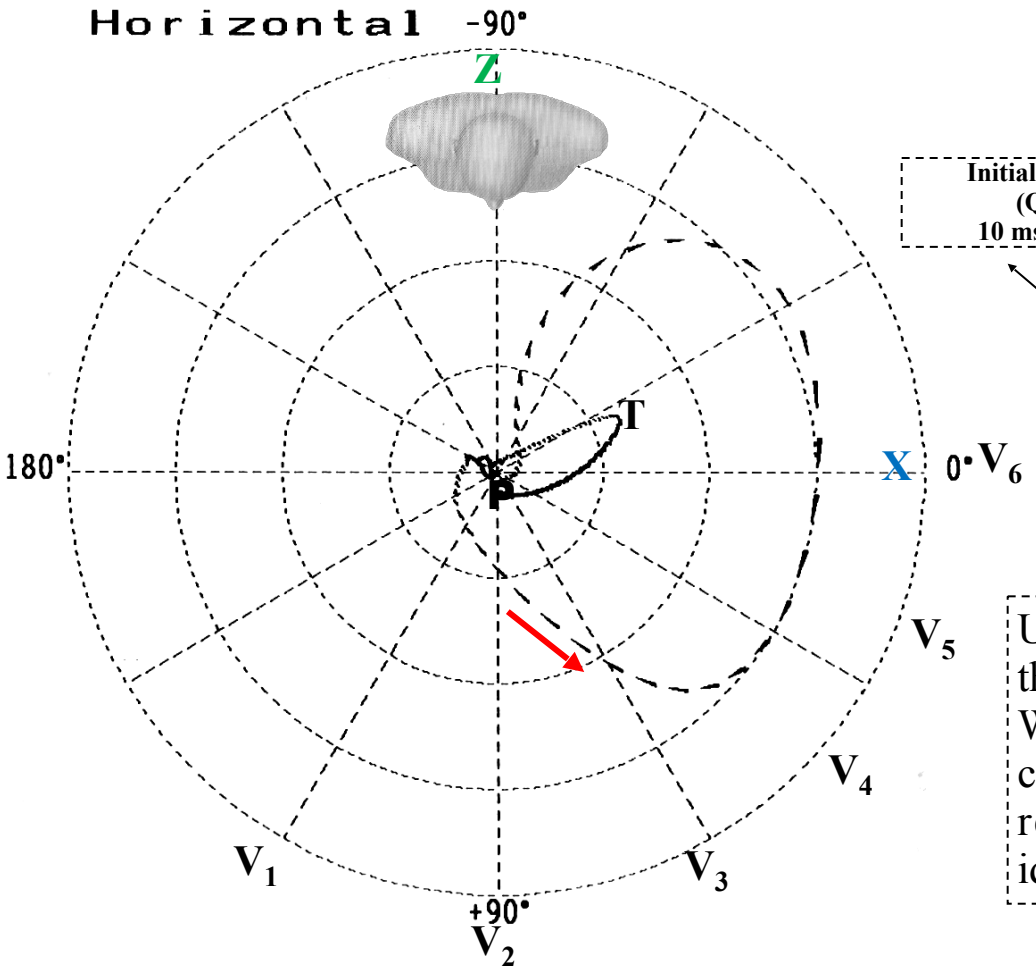
ECG diagnosis:

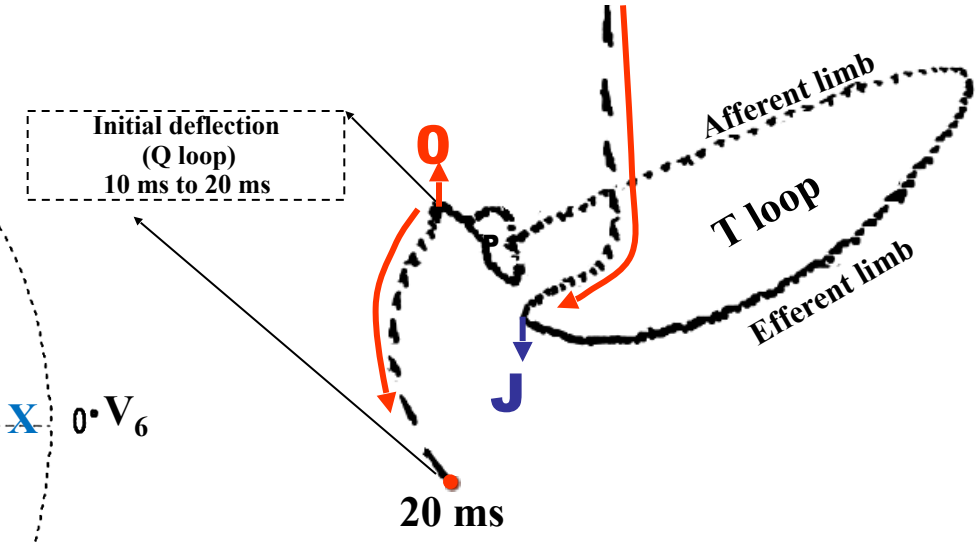
- _HR: 53 bpm: Predominant sinus bradycardia is observed in early repolarization pattern. ERV is seen in individuals with high vagal tone, such as athlete's heart. QRS duration of 90 ms.
- QRS axis +40º
- At least two adjacent precordial leads show ST segment elevation, with values ≥1 mm (2 mm). Notching, irregular or slurring contour of the terminal QRS complex (J point)._
- Relatively deep but narrow q waves may appear in the left precordial leads
- Positive Sokolow index. High QRS voltage is more frequent in male athletes, but its correlation with left ventricular hypertrophy is low **(our case)**. This young man is a professional soccer player.
- Prominent J wave and ST-segment elevation, concave to the top, predominantly in left precordial leads, ending in a positive large T wave from V2 to V4 or V5.
- Prominent U waves are observed because sinus bradycardia is present.

ECG/VCG frontal plane correlation



VCG horizontal plane correlation



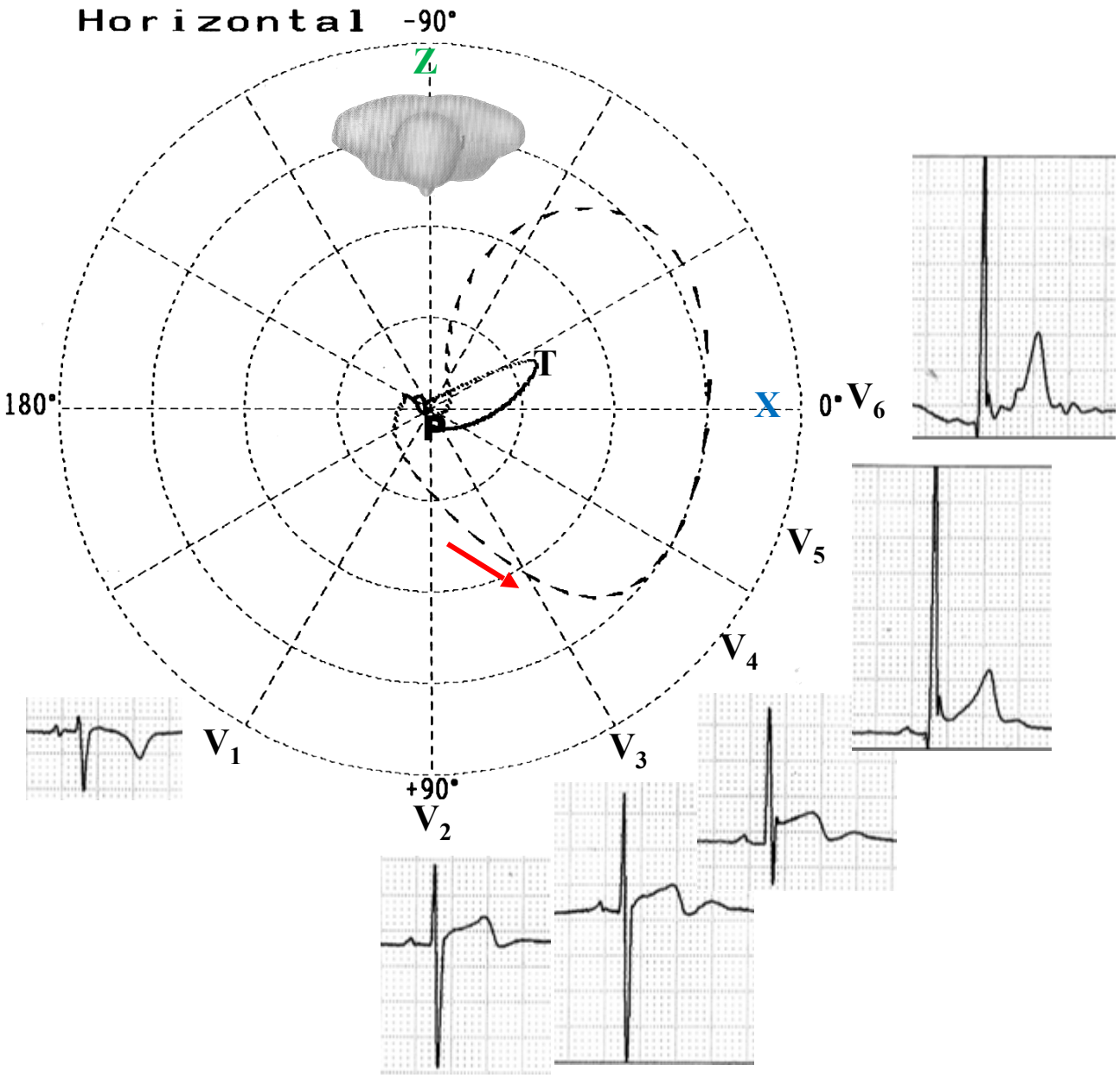


Usually 0 & J points are coincident. In this case, they are not: ST-segment elevation
When ST is elevated, the J and 0 points do not coincide: we observe this phenomenon in early repolarization pattern, Brugada syndrome, idiopathic VF, and short QT syndrome

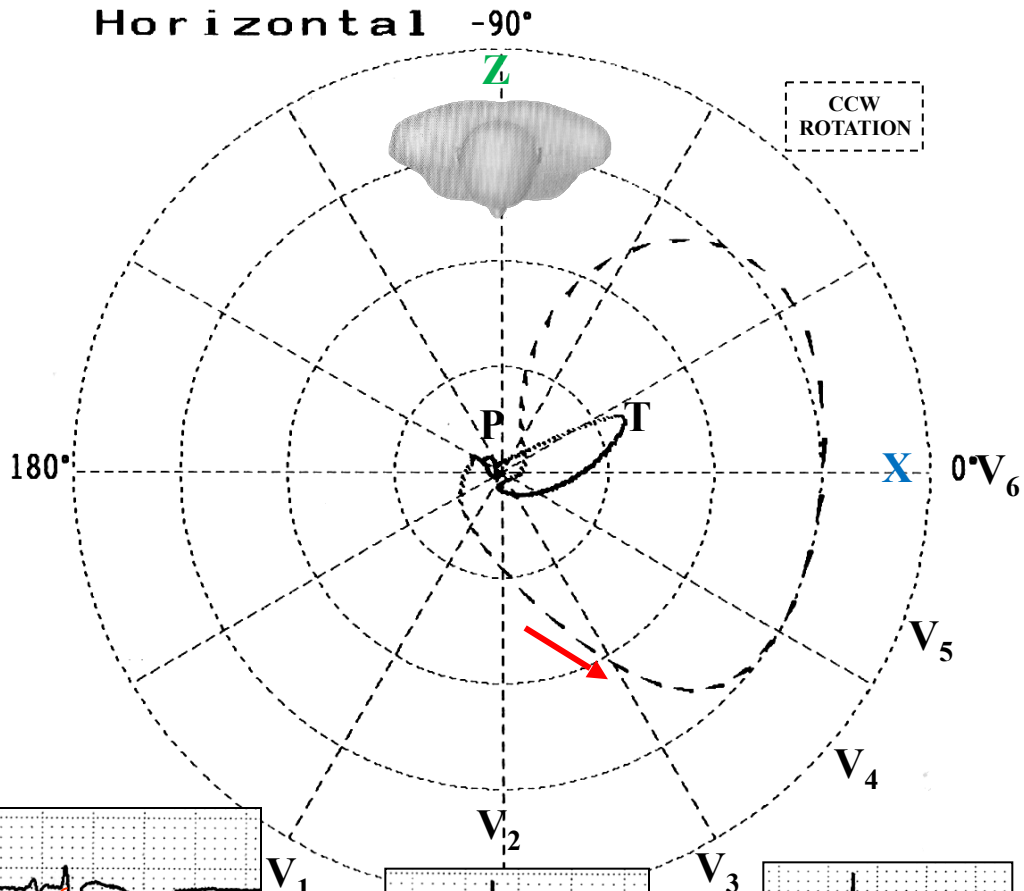
0 point: it corresponds to the end of biatrial chamber activation, QRS loop onset (because PR segment does not exist; it is only a point) and the end of ventricular repolarization (T loop).

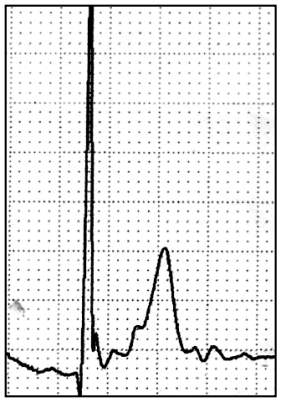
J point: in vectocardiography, it corresponds to 3 elements: end of ventricular depolarization (QRS complex); beginning of repolarization (ST segment) when it does not present depression or elevation, and T wave onset.

ECG/VCG correlation on horizontal plane

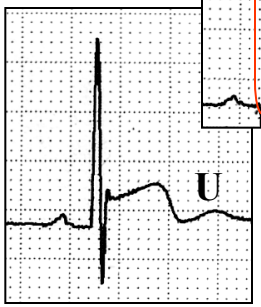


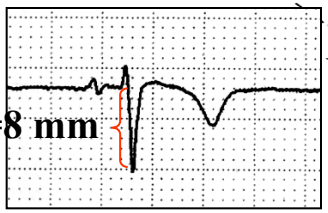
ECG/VCG horizontal plane correlation



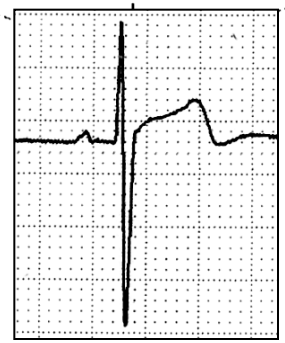


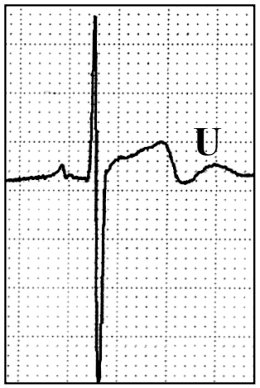
R=36.5 mm





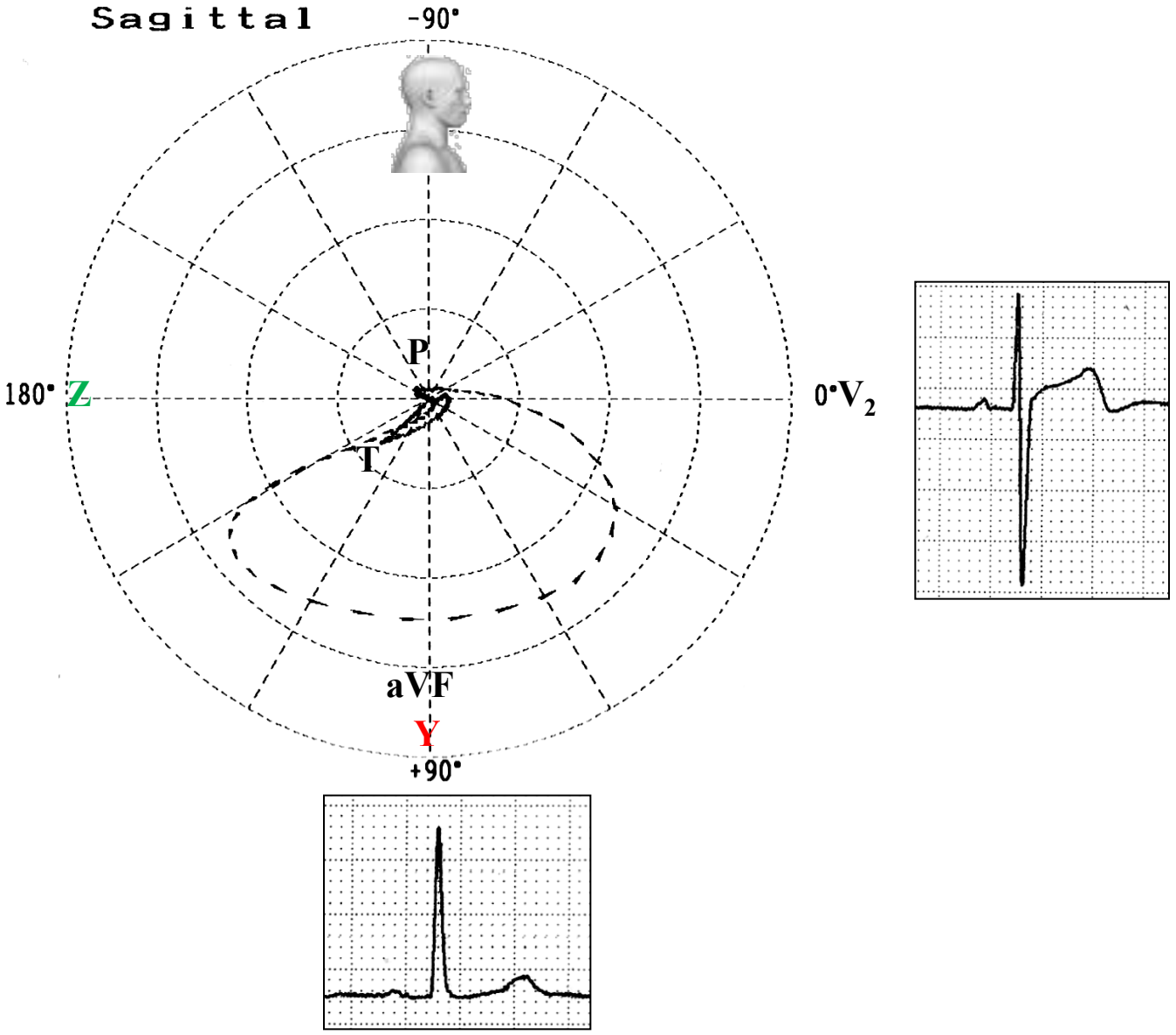
S=8 mm



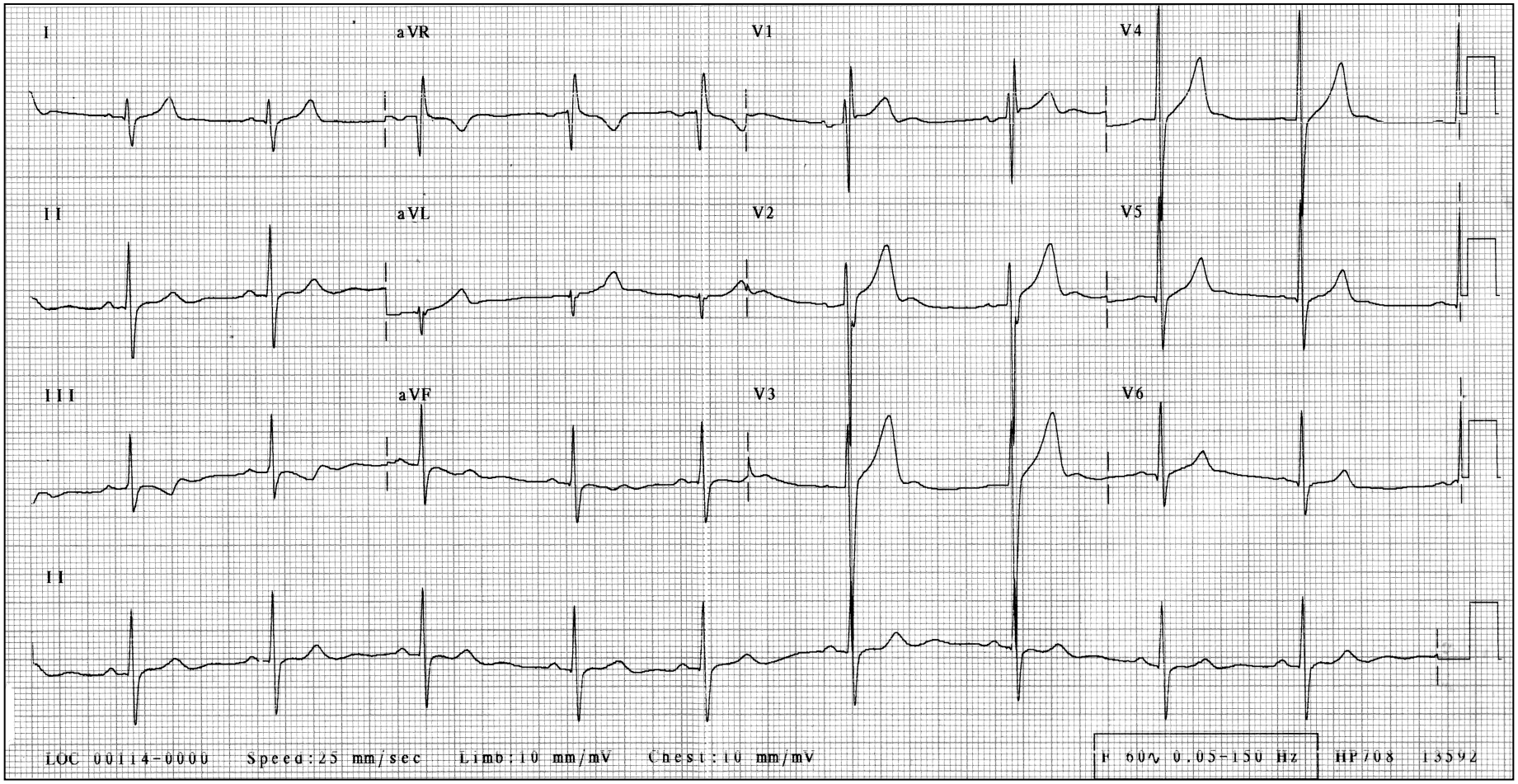


SV_1+RV_5 = 44 mm (maximum value 35 mm) (positive Sokolow & Lyon criteria for left ventricular hypertrophy)

ECG/VCG right sagittal plane correlation

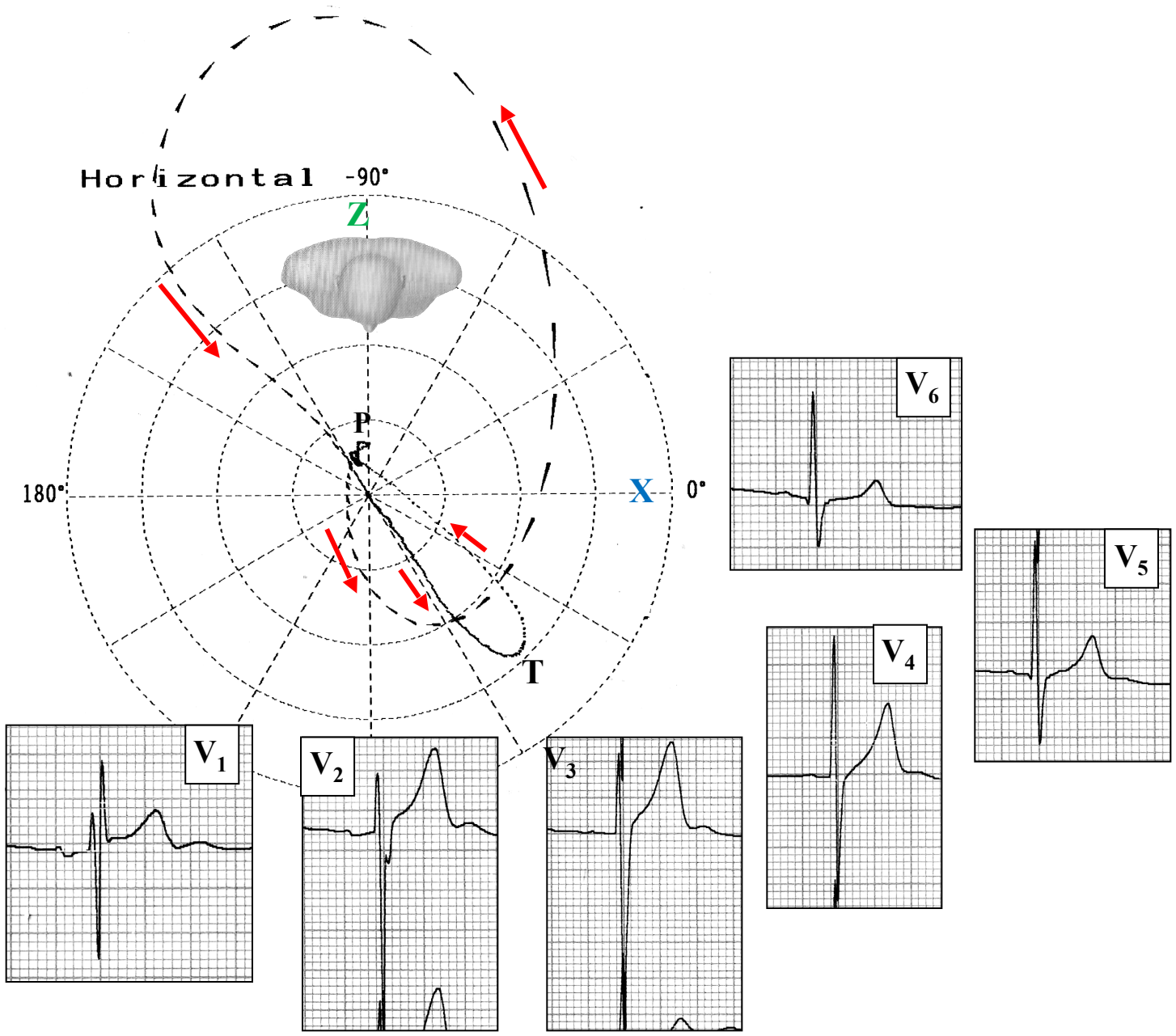


Name: ASF; **Sex**: Male; **Age**: 18 y.o.; **Race**: Afro-Descendent; **Weight**: 97 Kg; **Height**:1.93 m;
Biotype: Asthenic; **Date**: 07/30/2008; **Professional soccer player**: forward

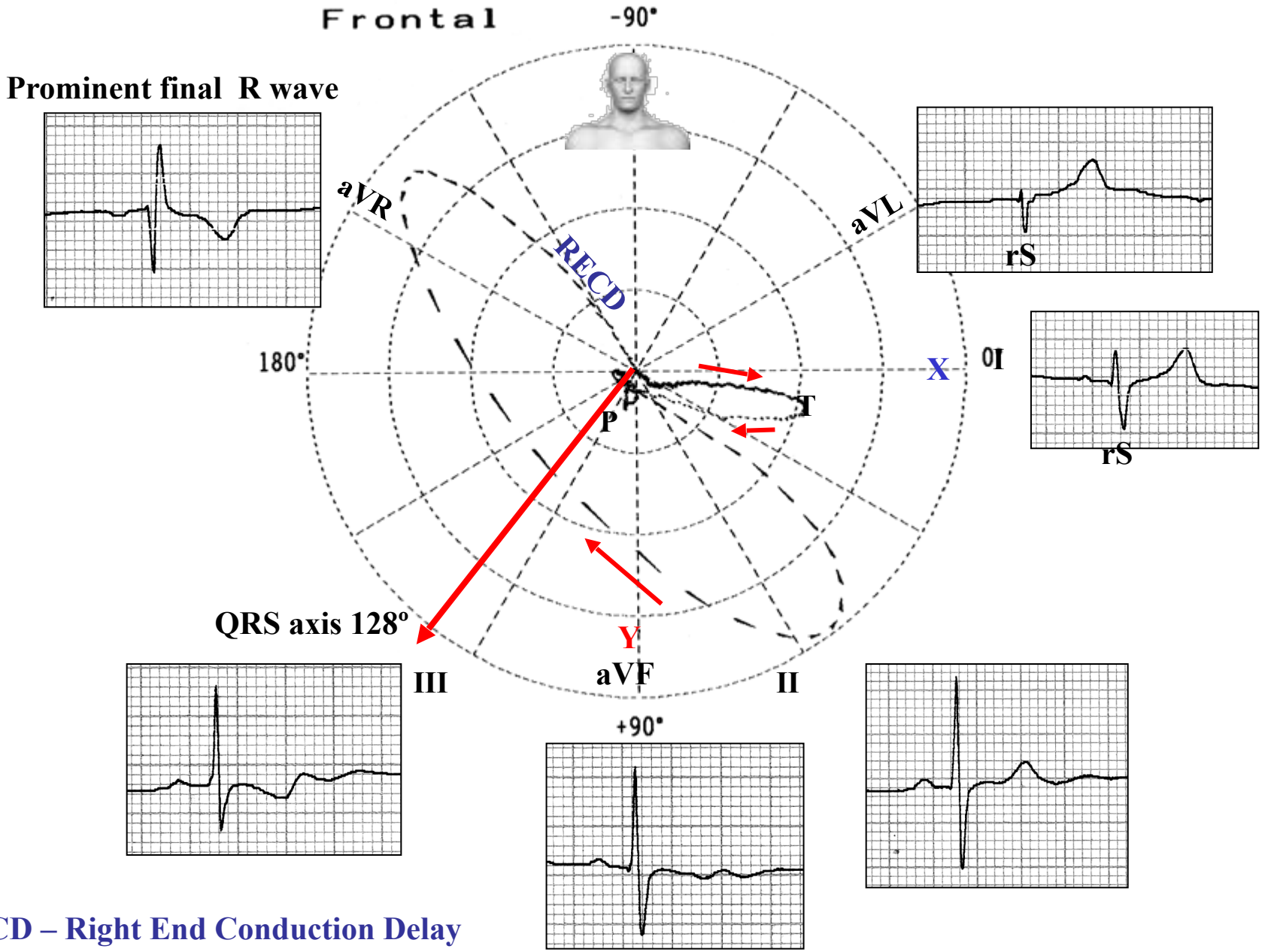


Clinical Diagnosis: Familial hypertension (BP: 150x105 mmHg). Recent diagnosis.
ECG diagnosis: HR: 58 bpm, P axis: 66 bpm, PR interval: 168 ms, QRSd: 97 ms, QRS axis: +128º, QT: 406 ms, QTc: 399 ms, T axis: +5º.
What is the diagnosis?

ECG/VCG horizontal plane correlation

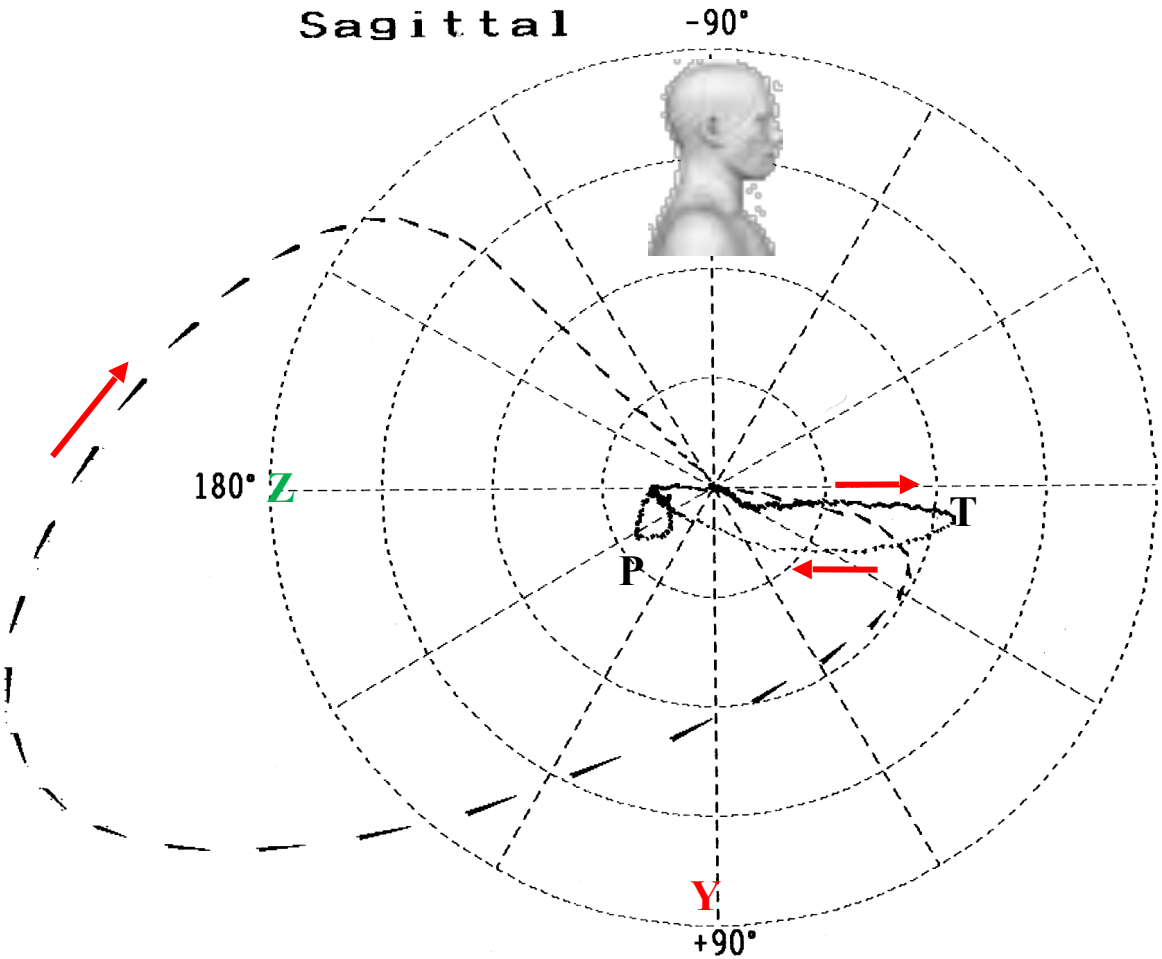


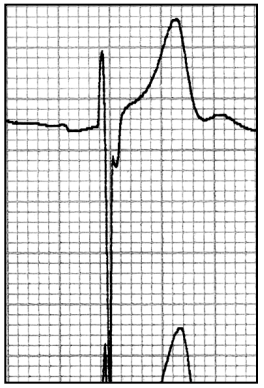
ECG/VCG frontal plane correlation

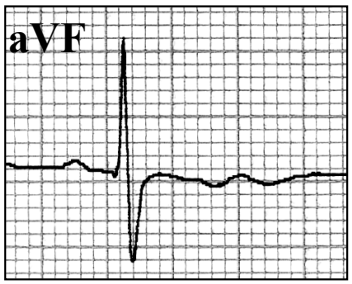


RECD – Right End Conduction Delay

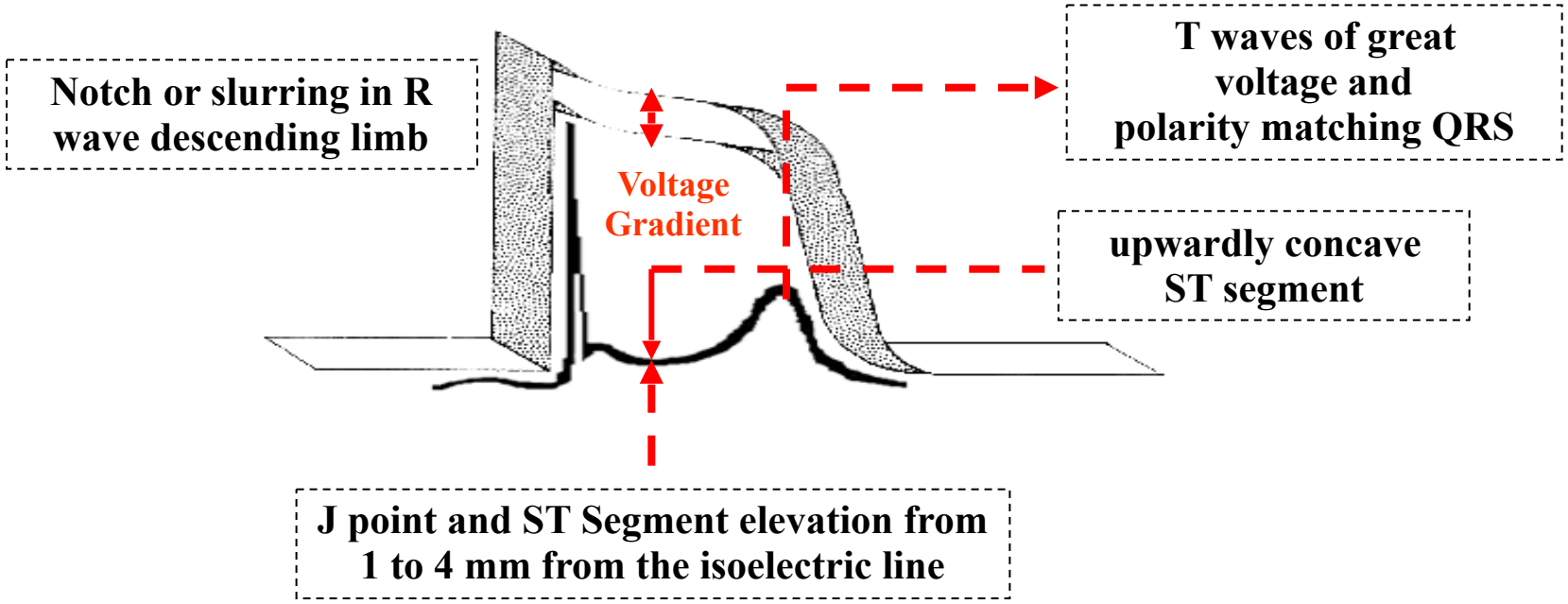
ECG/VCG right sagittal plane correlation





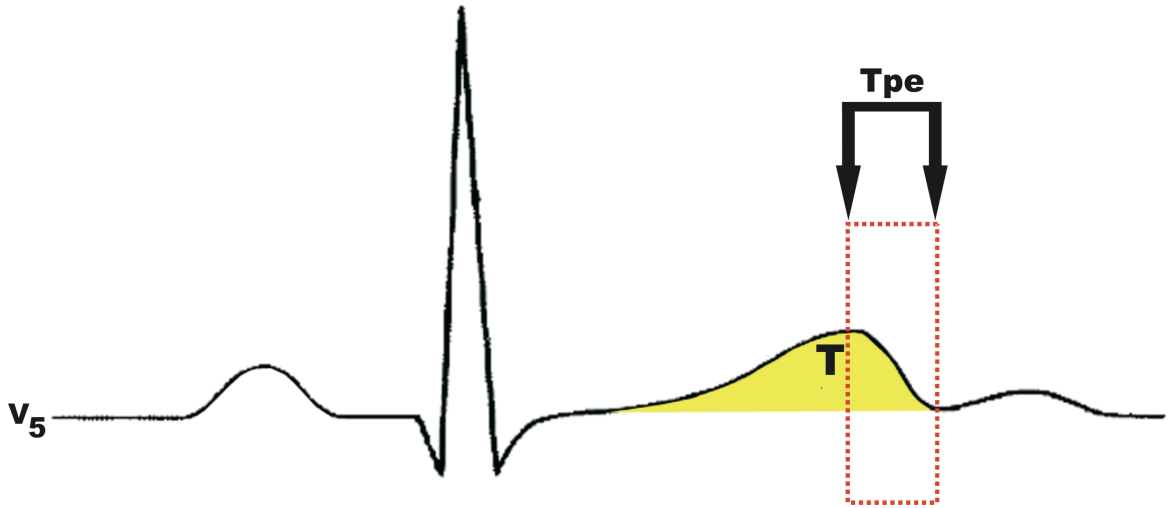


In early repolarization, there is a voltage gradient; however, no dispersion of duration of action potentials in ventricular wall thickness. For this reason, these patients showed ST segment elevation with no tendency to develop arrhythmias.



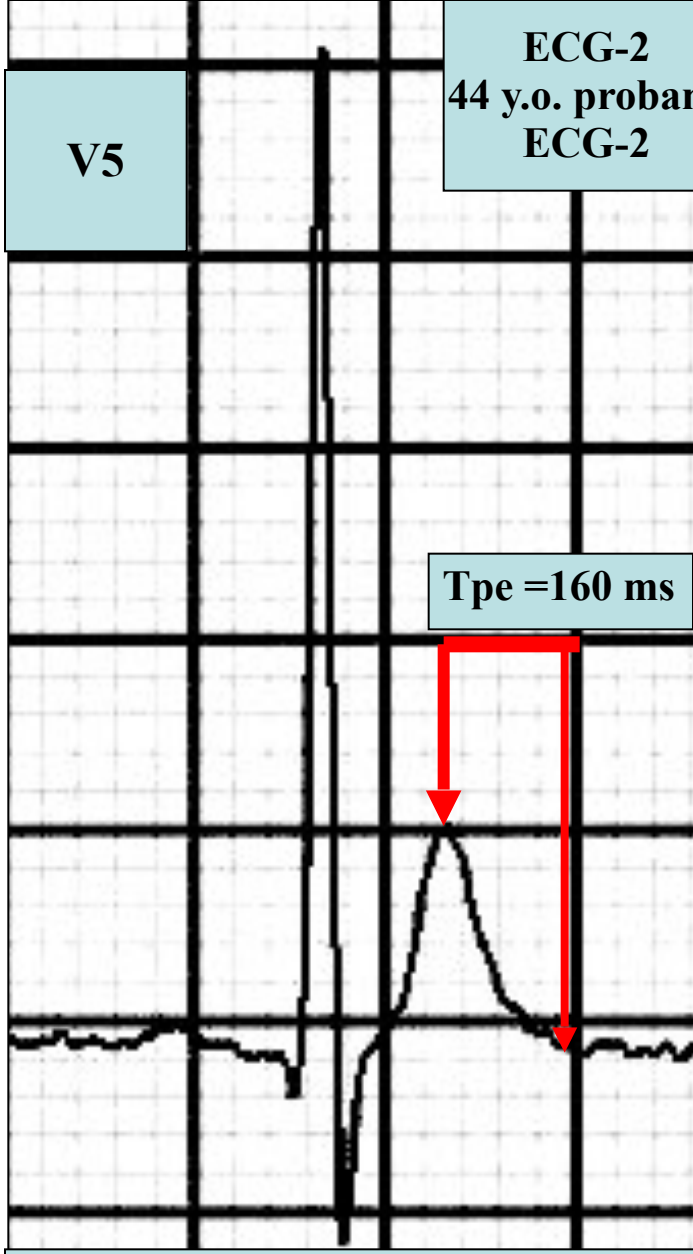
Theoretical electrophysiological explanation for ST segment elevation in ECGs in athletes.

The normal value of Tpeak/Tend interval (Tpe) is 94 ms in men and 92 in women when measured in the V5 lead. Tpe prolongation to values ≥120 ms is associated to a greater number of events in patients carriers of channelopaties. In experimental models of SQTS, increased transmural dispersion of repolarization (TDR) and its electrocardiographic counterpart T-wave peak to T-wave end interval (TPE) appeared to be critical for induction of polymorphic ventricular tachycardia (Anttonen 2008)



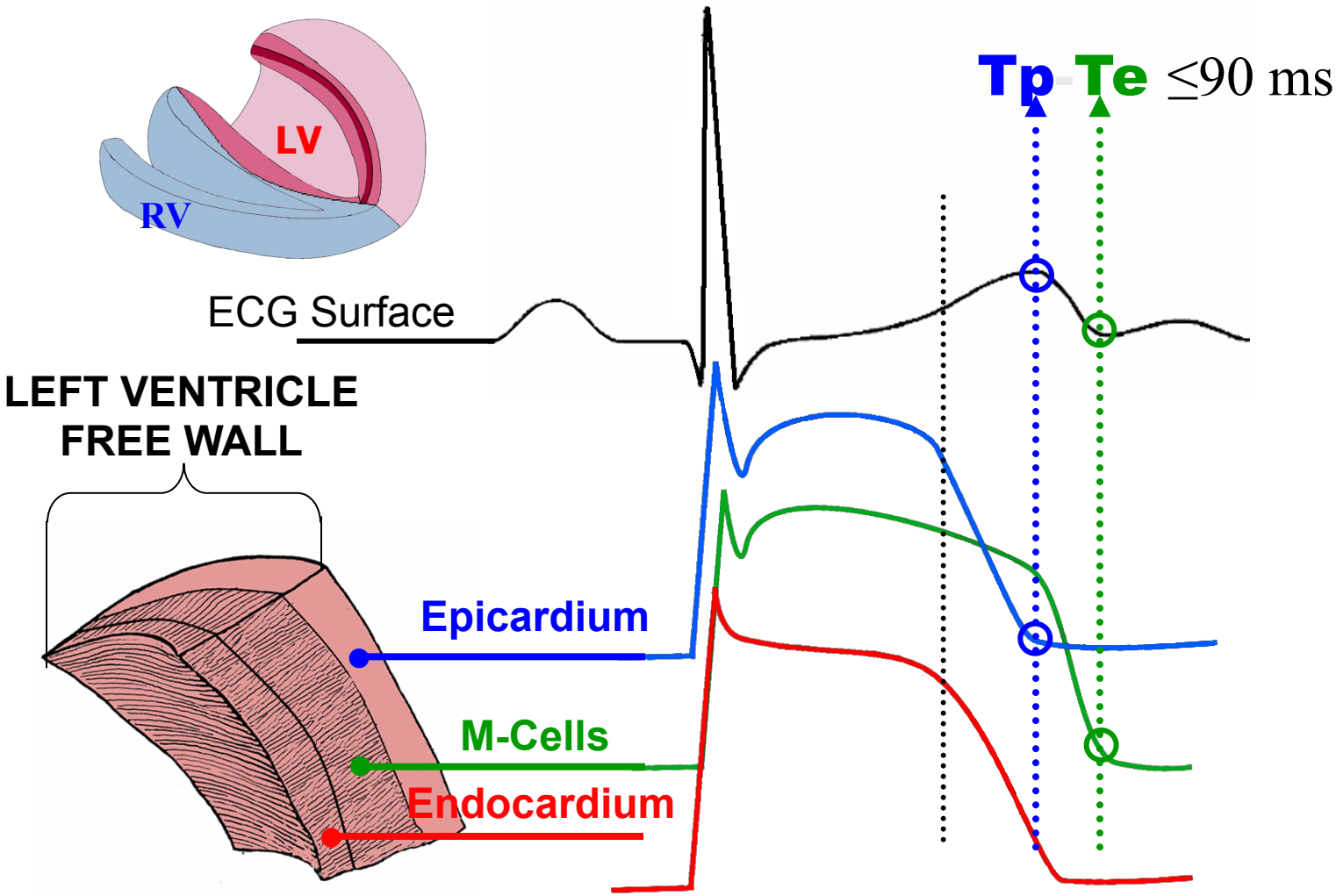
Tpeak/Tend interval (Tpe)

Interval elapsed from the apex to the end of T wave (Tpeak-Tend interval or Tpe). Tpe may correspond to transmural dispersion of repolarization and consequently, the amplification of this interval is associated to malignant ventricular arrhythmias.



Very broad Tpe interval indicati significant transmural dispersion repolarization

T-peak to T-end interval (TpTe)



Distance between the T-peak/T-end . Normal value ≤ 90 ms. It is prolonged from 90 to 130 ms in the global transmural dispersion cases. In this circumstance the following is observed:

- *QT interval prolongation from 350 to 450 milliseconds*
- *T-peak to T-end interval (TpTe) prolongation*
- *T-wave notches appeared in very limited precordial leads.*

ECG criteria that suggest benign Early Repolarization Pattern (ERP)

✓ HR: sinus bradycardia is frequent;

✓ Axes of QRS, ST segment and T wave, are oriented in the same direction in the FP;

✓ Deep and narrow Q waves followed by R wave of great voltage in left precordial leads;

✓ Notch or slurring of R wave descending branch;

✓ Transition area in precordial leads of sudden occurrence;

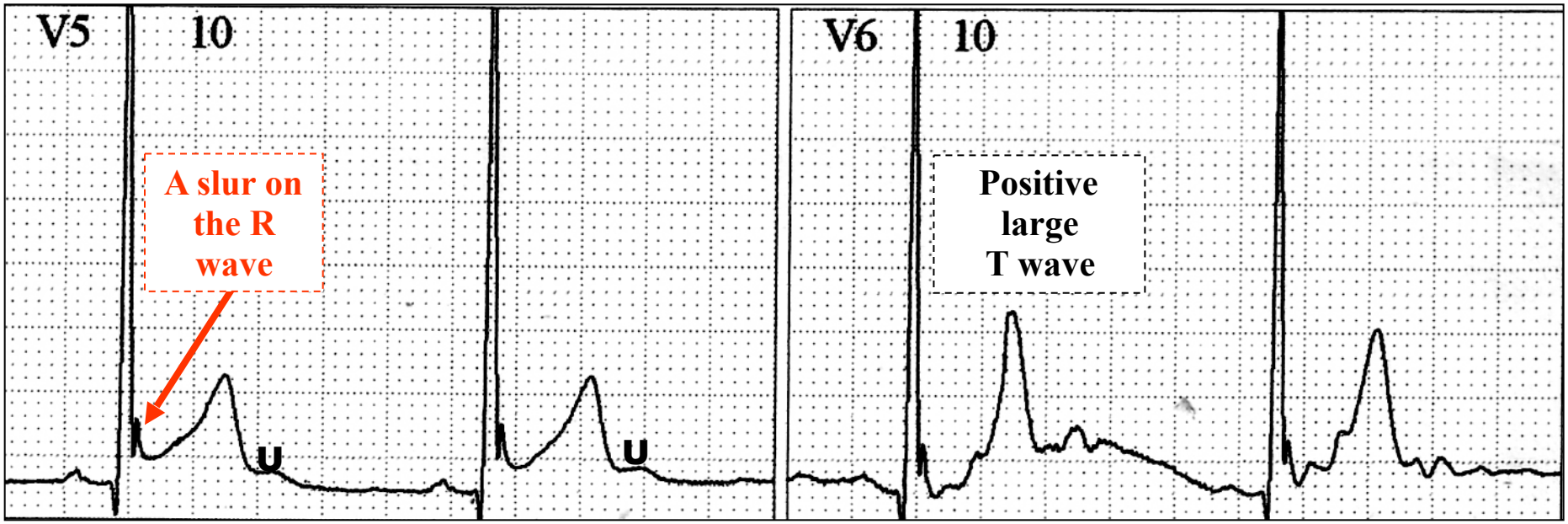
✓ J point and ST segment elevation, usually <2 mm (exceptionally it may be >5 mm) of superior concavity in middle and/or left precordial leads and possibly in inferior leads;

✓ Possible reduction in J point and ST segment elevation by sympathetic action and sympathomimetic drugs;

✓ Absence of reciprocal or mirror image (exception in VR lead);

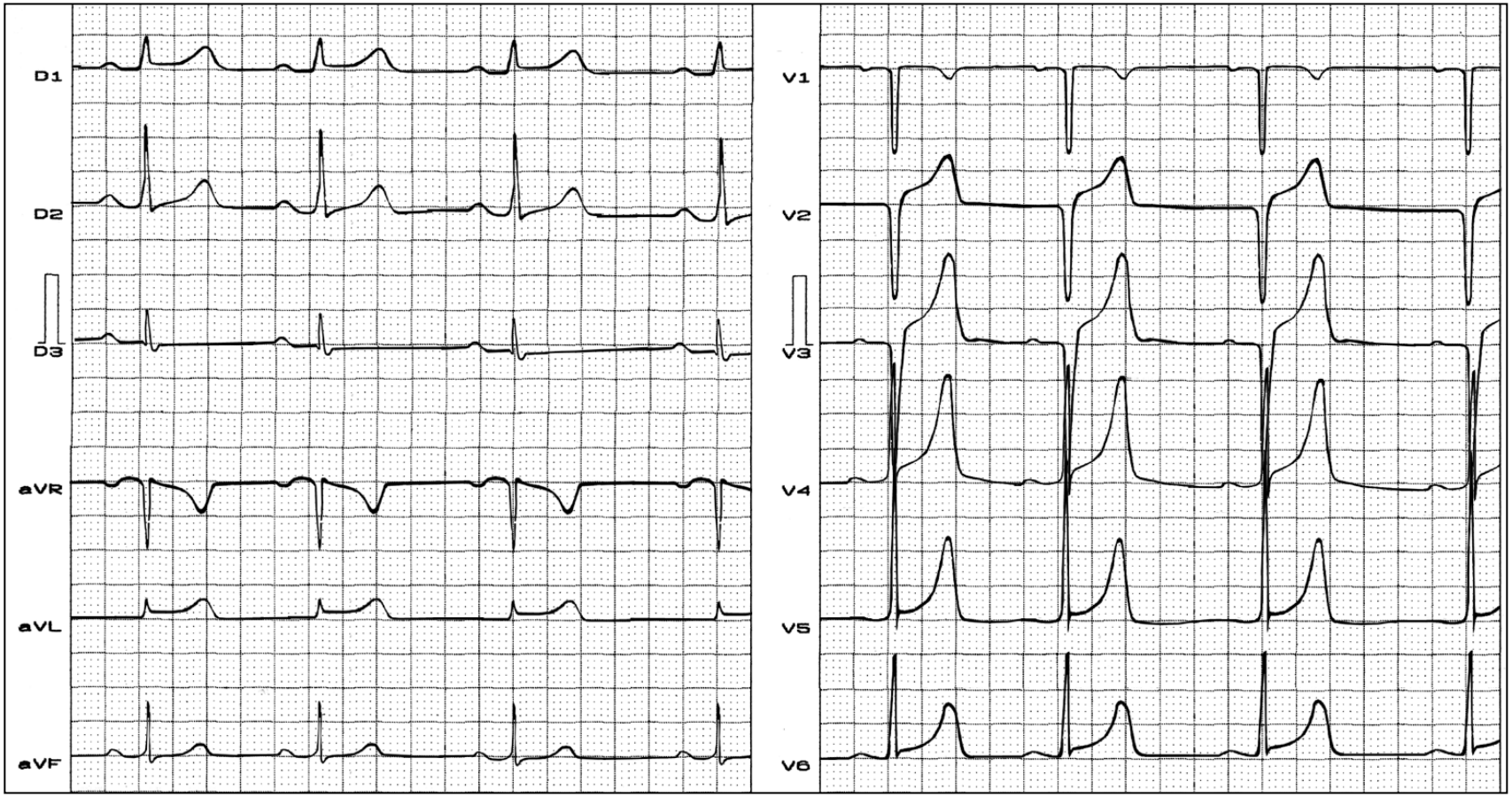
✓ Symmetrical T waves, with great width and polarity matching QRS;

Electrocardiographic criteria of early repolarization pattern.



- HR: 53 bpm: Predominant sinus bradycardia is observed in early repolarization pattern (ERP). ERV is seen in individuals with high vagal tone, such as athlete's heart. QRS duration of 90 ms.
- QRS axis +40º
- At least two adjacent precordial leads show ST segment elevation, with values ≥1 mm (2 mm). Notching, irregular or slurring contour of the terminal QRS complex (J point).
- Relatively deep but narrow q waves may appear in the left precordial leads
- Positive Sokolow index. High QRS voltage more frequent in male athletes, but its correlation with left ventricular hypertrophy is low **(our case)**. This young man is a professional soccer player.
- Prominent J wave and ST-segment elevation, concave to the top, predominantly in left precordial leads, ending in a positive large T wave from V2 to V4 or V5
- Prominent U waves are observed because sinus bradycardia is present.

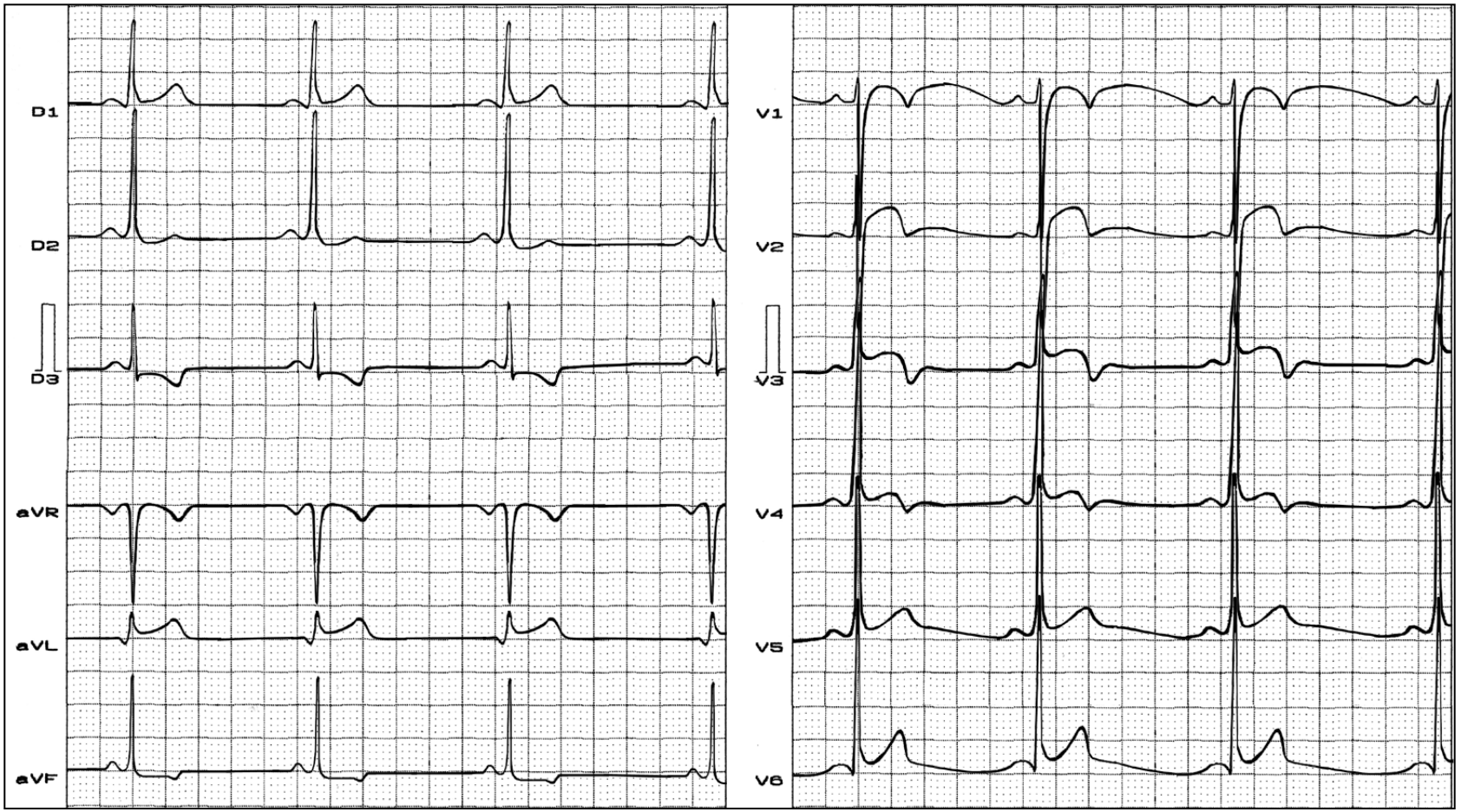
Name: BCA **Age:** 22 y.o. **Sex:** Male **Race:** Black **Weight:** 82 kg
Height: 1.96 m **Biotype:** Athletic Profession: professional basketball player **Date:** 2/09/2001



Clinical diagnosis: athlete's heart. Normal variant.
ECG diagnosis: sinus rhythm; HR: between 50 bpm and 57 bpm: phasic or respiratory sinus bradyarrhythmia; QS from V1 to V3: pattern of pseudo infarction in antero-septal wall. Peaked T waves from V3 to V6. Normal X-rays of chest and echocardiogram.

Pattern of pseudo anterior infarction in an athlete, professional player of basketball with normal heart.

Name: BCW; **Age:** 24 y.o.; **Sex:** Male; **Race:** Black; **Weight:** 86 kg; **Height:** 2.02 m**; Biotype:** Asthenic; **Profession:** professional basketball player; **Date:** 05/01/1999



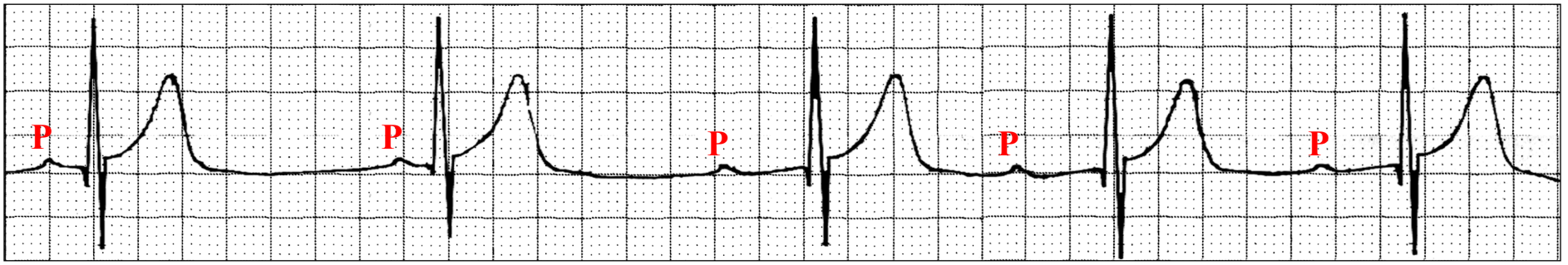
Clinical diagnosis: healthy patient. Tracing obtained in a periodical evaluation.

ECG diagnosis: sinus bradycardia, phasic sinus arrhythmia. Positive voltage criterion for LVE. SV_1 or V_2+RV_5 or V_6 >35 mm (Index of Sokolow Lyon). ST segment elevation from V_2 to V_6 and with negative T from V_1 to V_4. Early repolarization, pattern of pseudo injury and anterior subepicardial ischemia. Normal chest X-rays and echocardiogram.

Pattern of pseudo subepicardial injury and ischemia in anterior wall in an athlete, professional player of basketball with normal heart.

Holter recording 1ST degree AV block

Name: B.C. **Sex:** Male **Age:** 22 **Race:** Black **Weight:** 74 Kg.
Height: 1.82 m **Biotype:** Athletic **Date:** 01/04/2002
Time: 2:50:12 AM Patient sleeping. **Profession:** Marathon runner



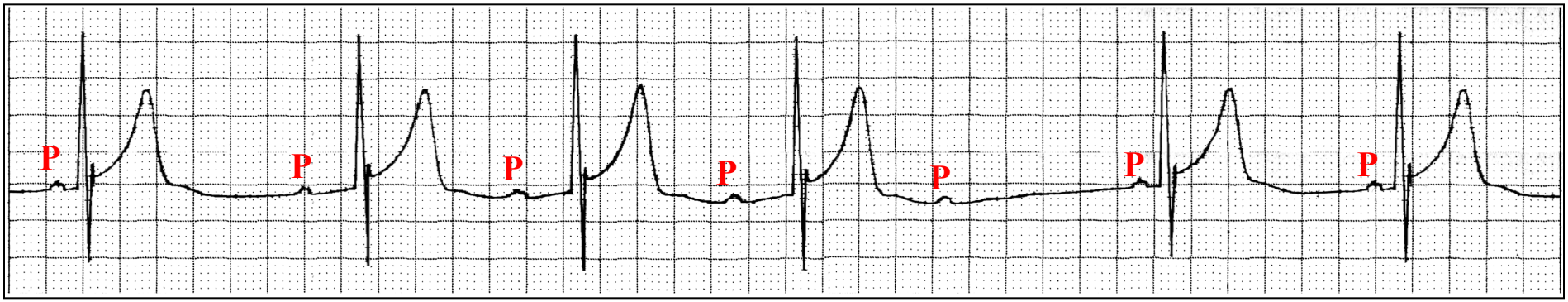
Heart rate of 38 bpm.
1st degree AV block usually observed for a few seconds, as in this case, where it is present only in the three last beats.

1st degree AV block is observed in average between 10% and 33% of athletes (**Smith 1964**), generally very briefly. In non-athletes it is around 0.65%.

1st-degree AV block in an elite athlete in Holter.

Holter recording

Name: A . S. **Sex:** Male **Age:** 26 **Race:** Black **Weight:** 64 Kg.
Height: 1.68 m **Biotype:** Athletic **Date:** 05/01/2003
Time: 3:42:30 AM Patient sleeping. **Profession:** long distance runner



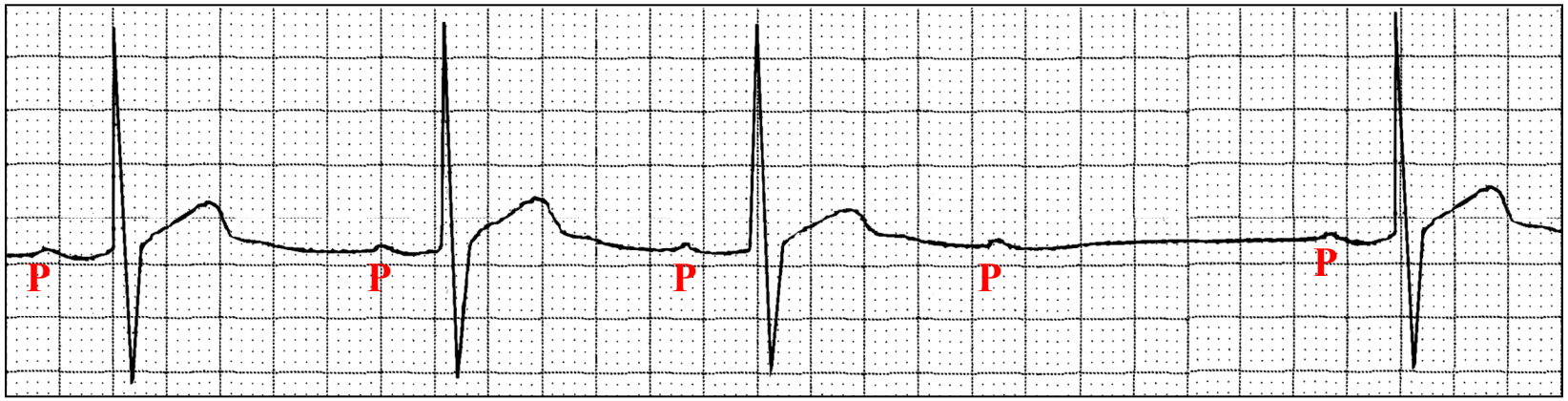
Gradual prolongation of PR interval until the 5th P wave is not conducted: 2nd degree AV block; Wenckebach or Mobitz Type I.

This modality of dromotropic disorder is observed in more than a 20% of elite athletes (**Viitasalo 1982**). In the general population, 2nd degree AV block Type I & II is observed and 1 each 30,000 people or 0.003 %

2nd-degree AV block, Wenckebach type or Mobitz Type I in an elite athlete.

Holter recording: 2nd degree AV block, Mobitz type II with narrow QRS

Name: E . J. **Sex:** Male **Age:** 26 **Race:** White **Weight:** 70 Kg.
Height: 1.72 m **Biotype:** Athletic **Date:** 25/01/2001 **Time:** 1:52:10 AM Patient sleeping
Profession: Long distance runner

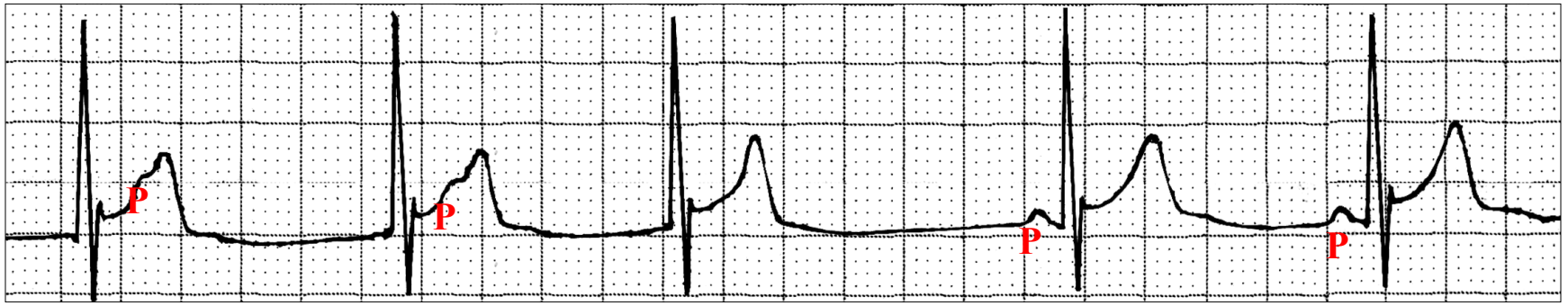


PR interval remains constant until a P wave is not conducted. This type of block is observed in 7% of the cases in athletes of enduro. Fixed or constant PR interval: it does not exist; progressive prolongation of PR, with the block occurring suddenly. In general, 2nd degree AV block type II with narrow QRS is observed in 35% of the cases and in the remaining 65%, the QRS is long.

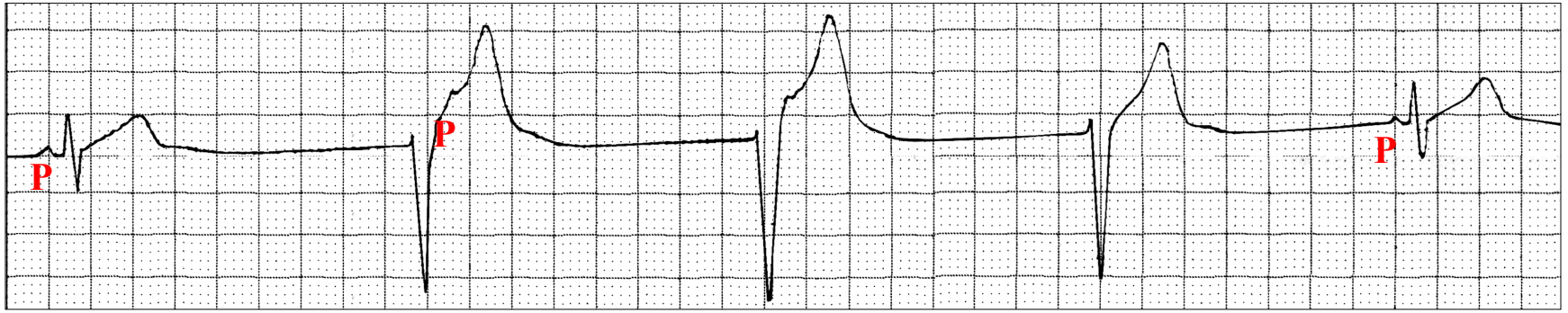
2nd-degree AV block, Mobitz type II with narrow QRS.

Holter recording

Atrioventricular dissociation (dissociation by interference) with junctional escape rhythm



Atrioventricular dissociation (dissociation by interference) with escape ventricular rhythm



Atrioventricular dissociation (dissociation by interference) with junctional escape rhythm and atrioventricular dissociation (dissociation by interference) with escape ventricular rhythm in an elite athlete in Holter.

Summary of the ECG elements common in athletes

1) Sinus bradycardia.
2) Sinus arrhythmia.
3) P wave with notches and of greater voltage.
4) 1st degree AV block: 6% to 36%.
5) 2nd degree AV block, Wenckebach type: Mobitz Type I (0.125% to 10%).
6) IRBBB or end conduction delay.
7) Voltage or axis criterion for RVE.
8) Voltage criterion for LVE.
9) Early repolarization variant
10) J point and ST segment elevation or depression.
11) QT interval in the superior borderline of normality.
12) T wave of increased voltage, peaked and inverted.
13) Atrial fibrillation and flutter (**Furlanello 1998**).
14) Junctional rhythm.

Features distinguishing athletic heart syndrome from HCM

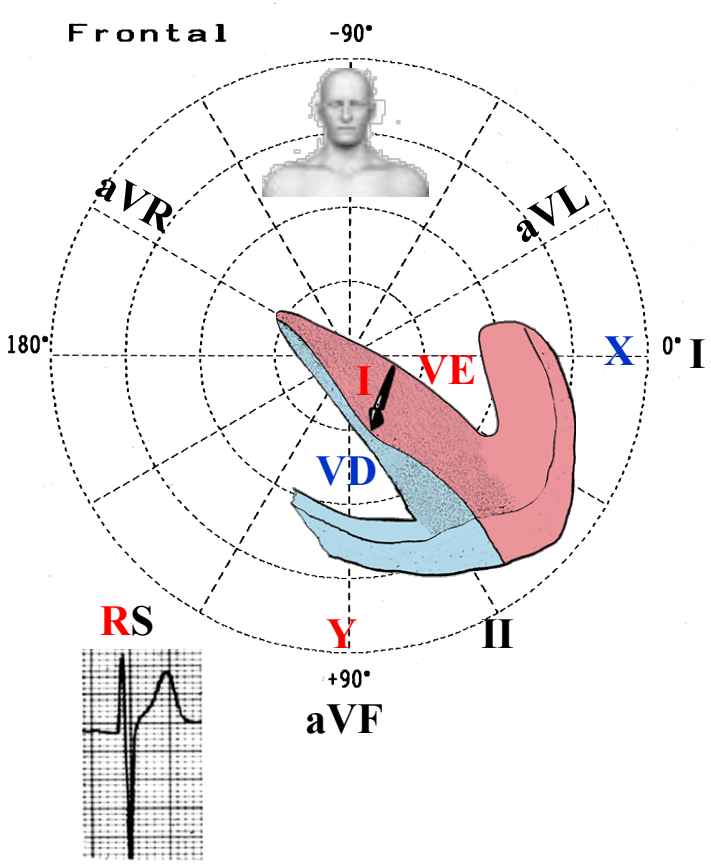
Final conclusions

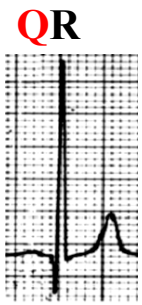
	Athlete's heart	HCM
LVE*	< 13 mm	> 15 mm
LV end-diastolic diameter†	< 60 mm	> 70 mm
Diastolic function	Normal (E:A ratio > 1)	Abnormal (E:A ratio < 1)
Septal hypertrophy	Symmetric	Asymmetric (in HCM)
Family history	None	May be present
BP response to exercise	Normal	Normal or reduced systolic BP response. Occasionally arrhythmias.
Deconditioning	LVE regression in ≈80% of cases	No LVE regression

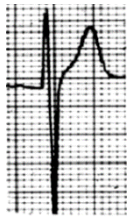
*A value of 13 to 15 mm is indeterminate.
†A value of 60 to 70 mm is indeterminate.
E: A ratio = ratio of early to late atrial transmitral flow velocity.

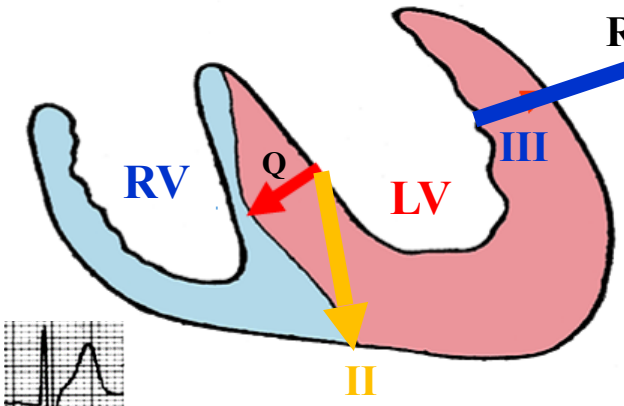
Consequences of the increase in the magnitude of Vector I of type IA diastolic LVE

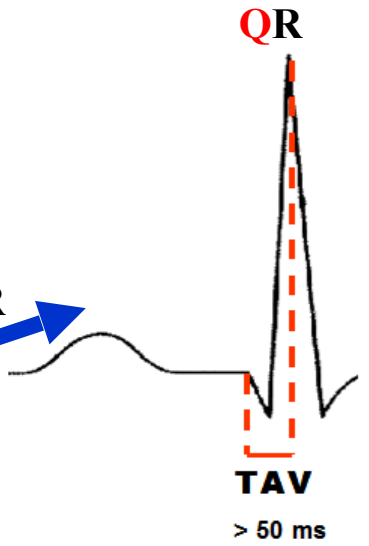
1. Discrete increase in QRS complex duration at the expense of delay in the time of appearance of R peak time (≥50 ms) in the left leads DI, aVL, V5 and V6.
2. Increase in the voltage of R of V1 and the depth of initial Q wave in the left leads I, aVL, V5 and V











QRS complexes in hypertrophic cardiomyopathy

- LVE with strain pattern with QRS/T angle near 180º and prominent R waves in intermediary precordial leads in 80%.
- In 10%, very wide R waves in V1 and aVR associated to deep and “clean” Q waves in V5 and V6 and/or in inferior leads, by > of septal vector 1 in 10%.
- LAFB (10%) with extreme shift of AQRS (beyond –30º).
- Increasing R wave from V2 to V4 and decreasing from V5 to V6, R wave of V4 of greater voltage than the other precordial leads (74%), absence of q waves in DI (87%) and V5 (91%), anterior shift of QRS loop in the HP (74%) and R vector of posterior and rightward orientation (91%); in familial forms, 50% QS pattern from V1 to V4. In sporadic ones is 15%.
- Important Q waves of pseudo infarction, with less duration (<40 ms) and deep. Q waves in young patients with absence of MI history.

Characteristics of QRS complexes in Hypertrophic Cardiomyopathy.

QRS complexes in hypertrophic cardiomyopathy

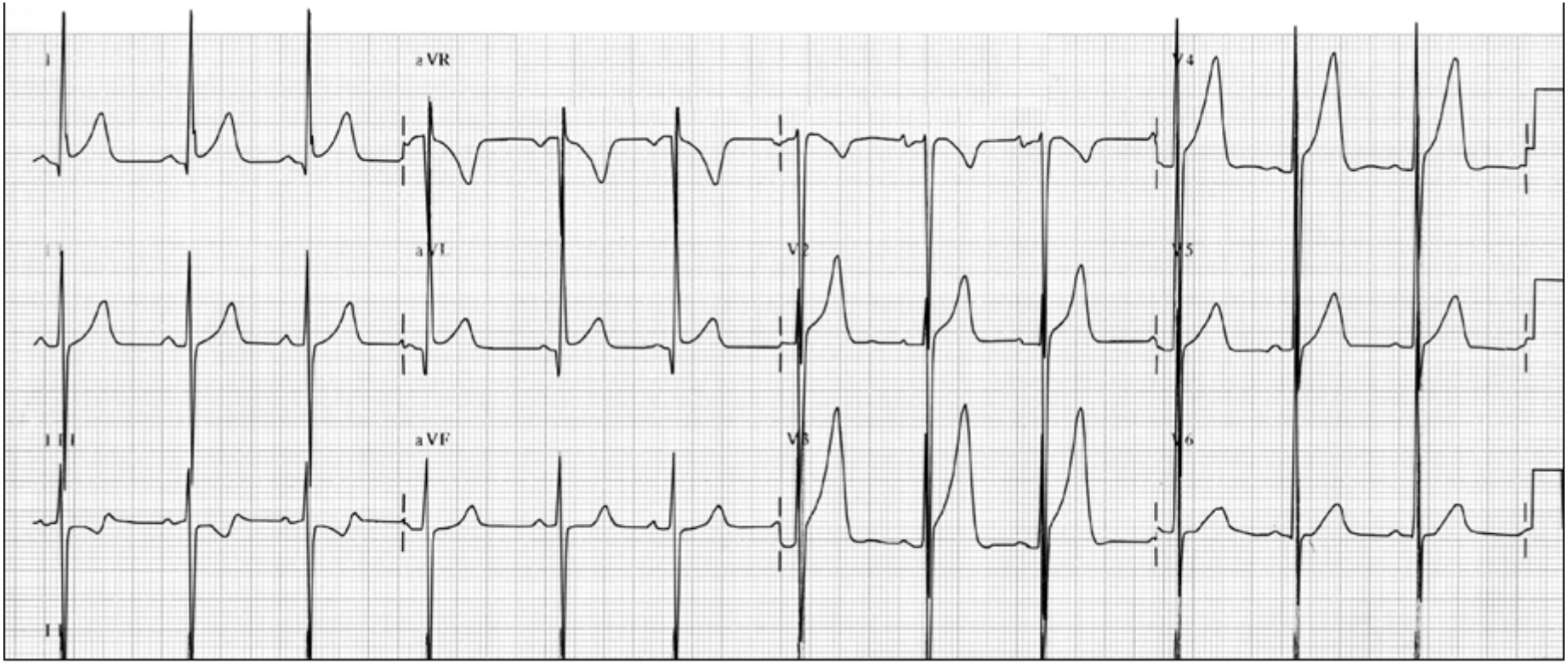
- **Complete LBBB**
  - After transvalvar myotomy/myomectomy surgery (80% of cases) (slide 27).

- **Complete RBBB**
  - After absolute alcohol injection in the first septal perforating artery of the anterior descending artery (ADA) in percutaneous transluminal septal ablation (**Pérez Riera 2002**).

1) Pérez Riera AR, et al. Arq Bras Cardiol; 2002 79:471-475

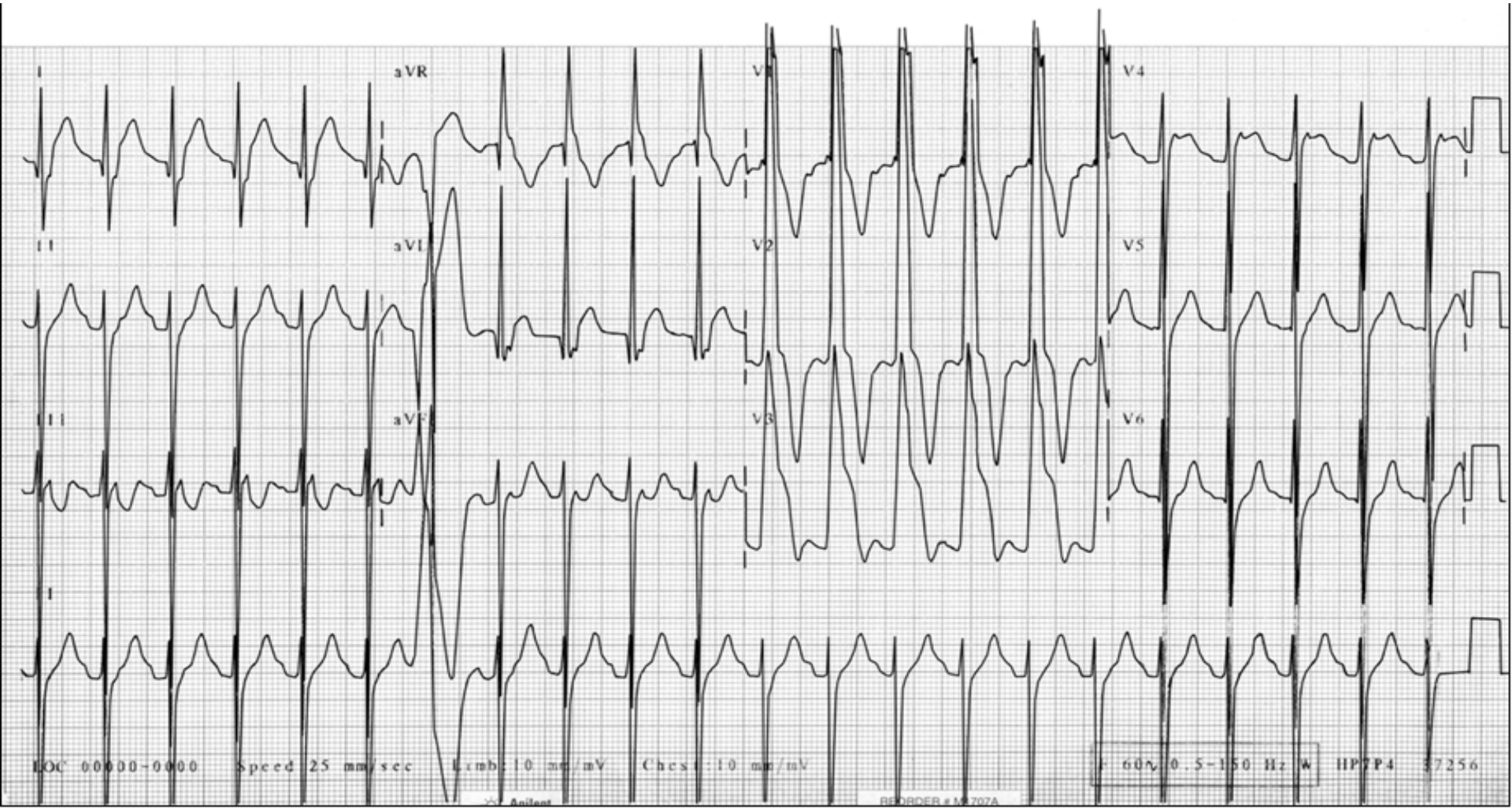
Characteristics of QRS complexes in Hypertrophic Cardiomyopathy.

ABB, female , age: 13 y.o. **Date: 23/01/2003**



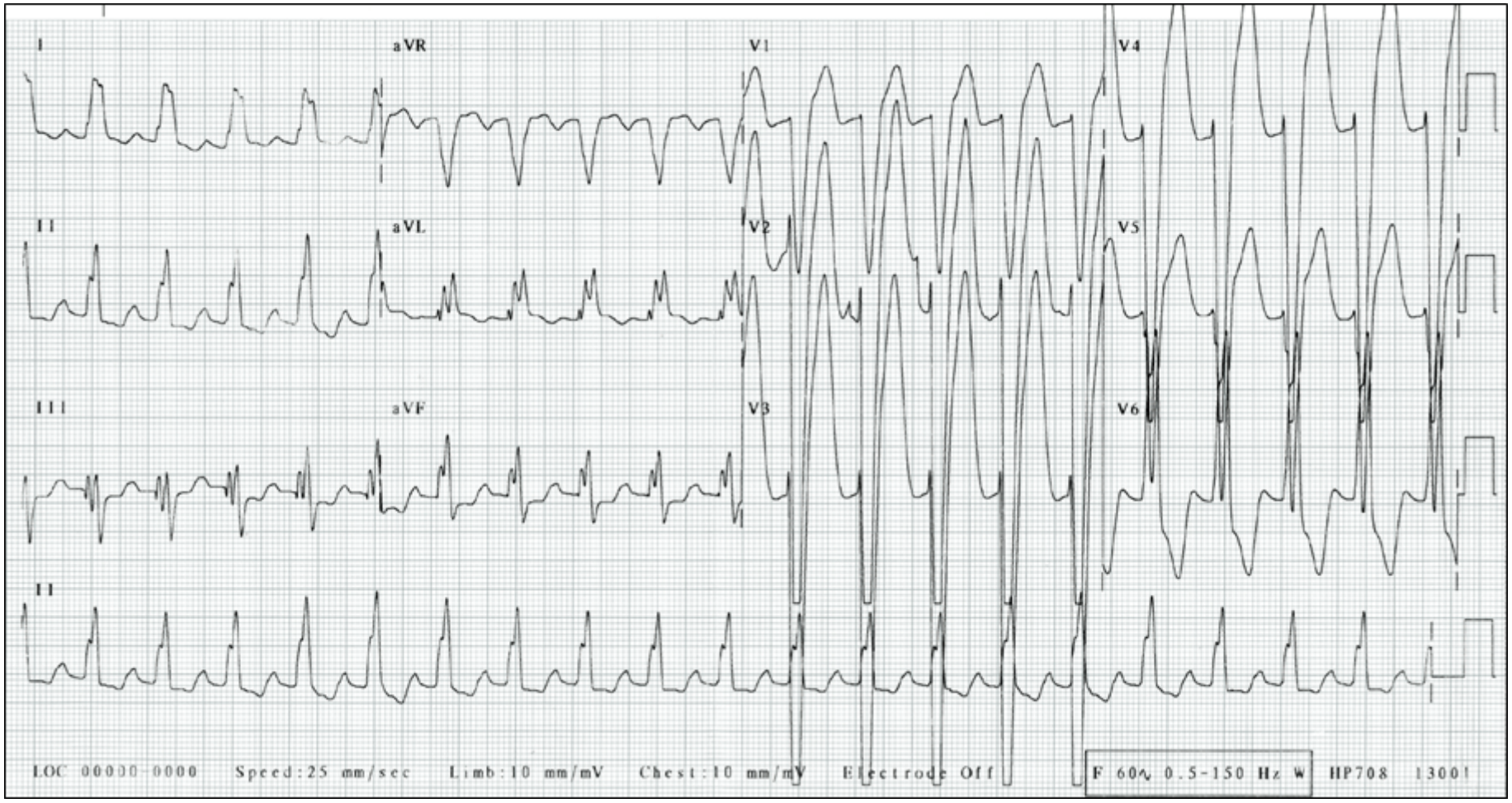
HOCM: ECG in pre-percutaneous transluminal alcohol septal ablation.

ABB, female , age: 13 y.o. **Date: 23/01/2003**



HOCM: ECG immediately post-percutaneous transluminal alcohol septal ablation.

ABB, female , age: 15 y.o. **Date: 11/01/2005**



HOCM: complete LBBB after transvalvar myotomy/myomectomy surgery.

Concept of Sudden Cardiac Death (SCD) and prevalence in athletes

SCD is defined as the death that is not traumatic, not violent, unexpected, which occurs within the first 6 h without a prior manifestation of cardiac disease (**Maron 1986**).

Prevalence

Estimated between young athletes of secondary school, as 1 in 200,000 per year (**Maron 1996, 1998**).

Concept and prevalence of sudden cardiac death in athletes.

The causes of arrhythmic sudden cardiac death in young athletes (average age: 17 years)

I. Entities with structural heart disease (98%)

1. Hypertrophic cardiomyopathy (HCM) whether in its obstructive form or in its non-obstructive form (36%);
2. Congenital anomalies of coronary arteries with increase in ventricular mass (19%);
3. Tumors or cardiac masses (10%);
4. Aorta rupture due to Marfan syndrome (5%). Mutation in the gene in fibrillin-1 (FBN1), in chromosome 15q21.1 and Marfan-like syndrome with no eye anomalies, mapped in chromosome 3p24;
5. Arrhythmogenic right ventricular dysplasia/cardiomyopathy (3%). Prevalence of 1 in 15,000;
6. Early atherosclerotic coronary artery disease (2%) by familial hypocholesterolemia and dominant mixed hyperlipidemia by alteration in chromosome 6;
7. Mitral valve prolapse syndrome (MVPS) (2%);
8. Myocarditis (2%);
9. Familial arrhythmogenic syndrome: ventricular and tachyarrhythmia association (syndrome of Wolff-Parkinson-White), progressive disease of conduction system and cardiac hypertrophy by involvement of regulatory subunit gamma-2 (PRKAG2) of AMP-activated by protein kinase (**Gollob 2002**);
10. 10) Aortic stenosis.

Description of the causes for arrhythmic sudden death in young athletes (average age: 17 years old), secondary to structural heart disease.

The causes of arrhythmic sudden death in young athletes (average age: 17 years)

II) Entities without structural heart disease (2%);

1) Drug abuse, e.g. anabolic agents;

2) Ventricular preexcitation of the Wolff-Parkinson-White syndrome type, with anomalous pathway of short refractory period, not detected previously;

3) Cardiac concussion or commotio cordis;

4) Channelopathies or primary electrical diseases.

Description of the causes for arrhythmic sudden death in young athletes (average age: 17 years old) without structural heart disease.

Channelopathies or primary electrical diseases

A) Of the sarcolemma or external channelopathies:

1) Congenital long QT syndrome;

2) Brugada disease;

3) Progressive familial heart block type I; progressive "idiopathic" disease of the His-Purkinje system or Lenègre;

4) Genuine idiopathic ventricular fibrillation (GIVF);

5) Mixed forms or with overlapped phenotypic aspects:

5a) Brugada disease and variant 3 of congenital LQTS;

5b) Brugada disease and Lenègre disease;

5c) Brugada disease and sinus node dysfunction;

5d) Association of Brugada disease, LQTS and progressive conduction disorder;

6) Some sudden unexpected nocturnal death syndromes (SUNDS);

7) Some sudden infant death syndromes (SIDS).

B) Of the channels of the endoplasmic reticulum or intracellular channelopathies:

1) Catecholaminergic polymorphic ventricular tachycardia (CPVT).

Description of the causes for arrhythmic sudden death in young athletes (average age: 17 years old) without structural heart disease by channelopathies or primary electrical diseases.

Structure of physiological ventricular hypertrophy in athlete's heart

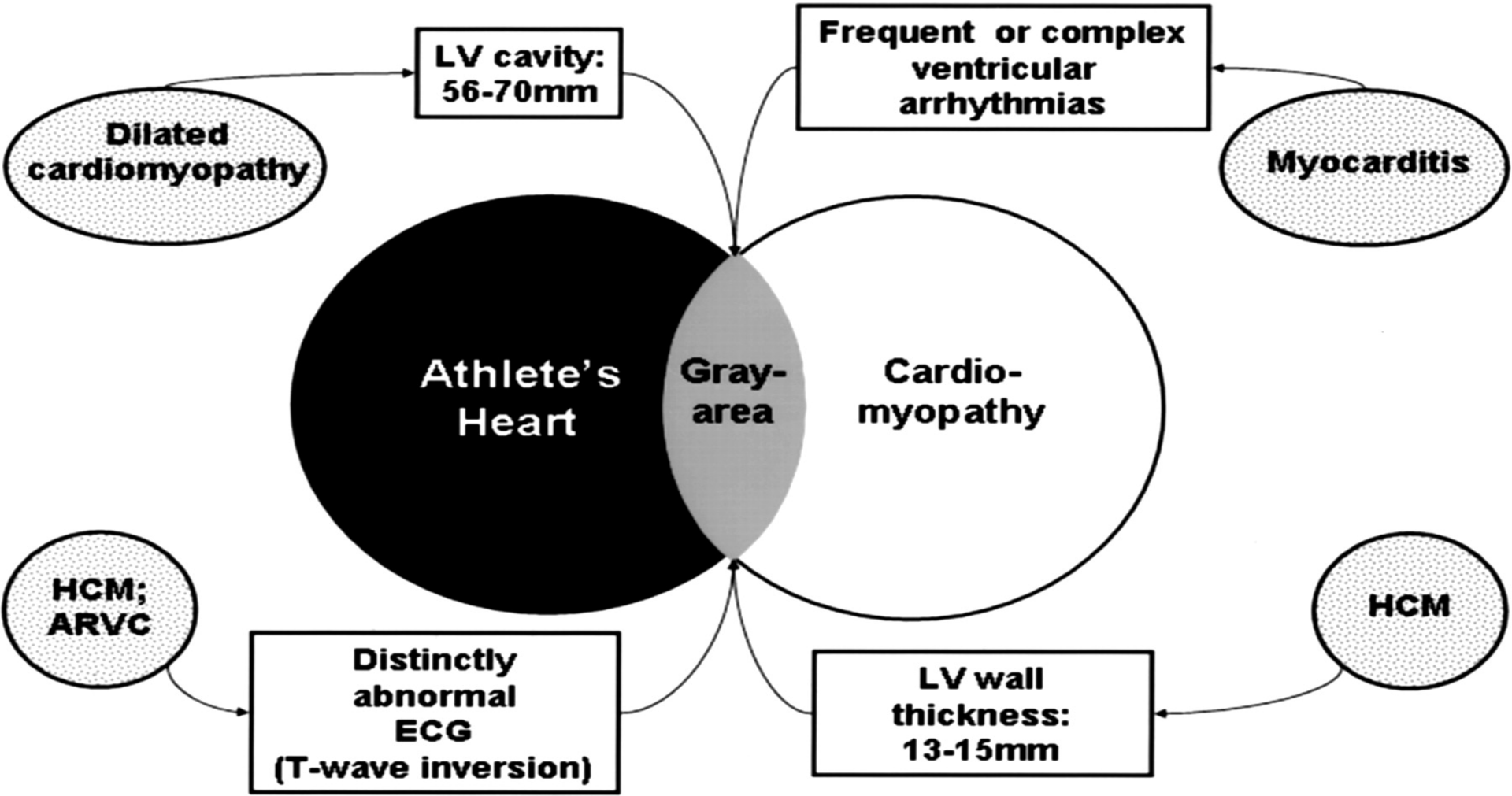
"Possibly, the differentiation between physiological ventricular hypertrophy of athletes and the pathological one (ventricular remodeling) may become a challenge".

Difference between physiological ventricular hypertrophy of athletes and the pathological one (ventricular remodeling).

Differences between physiological ventricular hypertrophy of the athlete and the pathological one (ventricular remodeling)

	Physiological ventricular hypertrophy	Pathological ventricular hypertrophy ventricular remodeling
Location:	Symmetrical, however, it may be asymmetrical.	Asymmetrical, however, it may be symmetrical.
Relative Ischemia:	Absent.	Present.
Myocytic/Non-myocytic Component Relationship:	Maintained.	Loss of balance in favor of the non-myocytic component (fibrosis).
Energetic Cycle:	Aerobiosis.	Anaerobiosis.
Renin-angiotensin-aldosterone mechanism	Normal.	Increased.
Norepinephrine	Normal.	Increased.
Atrial Natriuretic Peptide	Normal.	It may be increased.
Pump Function	Normal.	Depressed.
Heart Rate:	Tendency to sinus bradyarrhythmia by vagotony.	Frequent tachycarrhythmia and sympathotony.
LV Pd2:	Normal.	Increased.
Pulmonary Artery Pressure and Central Venous Pressure:	Normal.	It may be increased.
ANS:	Parasympathetic predominance.	Sympathetic predominance.

	Physiological ventricular hypertrophy	
Curve Of Dissociation of Hb:	Deviation to the right.	Deviation to the left.
Echocardiogram:	Proportional growth between the diameter and the thickness of walls. Normal LA.	Loss of walls thickness/diameter ratio. Increased LA.



Differences between physiological ventricular hypertrophy of athletes and hypertrophic cardiomyopathy (HCM) when both present wall thickness between 13 mm & 15 mm

The concentric or symmetrical form of HCM (5%), may be confused with the athlete's heart with physiological hypertrophy of its walls, since the increase is not asymmetrical. For the differential diagnosis, the following criteria could be used:

	Athlete	**HCM**
Female gender	Negative	Positive
Decrease of hypertrophy with less physical training	Positive	Negative
Family history or provable genetic mutation	Negative	Positive
Bizarre ECG pattern of LVE	No	Yes
LV cavity < 45 mm	No	Yes
LV cavity > 55 mm	Yes	No
LAE	No	Yes

Non-invasive supplementary tests

4) Transesophageal or biplanar echocardiogram.

5) Three-dimensional echocardiogram.

6) Intracardiac echocardiogram.

7) Ergometer test with spirometery.

8) High resolution ECG.

9) Holter.

10) RR variability.

11) Microvolt T wave alternans

12) Nuclear Magnetic Resonance.

13) Ultra-fast computed tomography.

14) Radioisotopic ventriculography.

Description of supplementary non-invasive tests, indicated in selected (suspected) cases.

Invasive supplementary tests

1) Programmed electrophysiological stimulation.
2) Cineangiography, coronary angiography, and ventricular angiography.
3) Endomyocardial biopsy.

Invasive tests.

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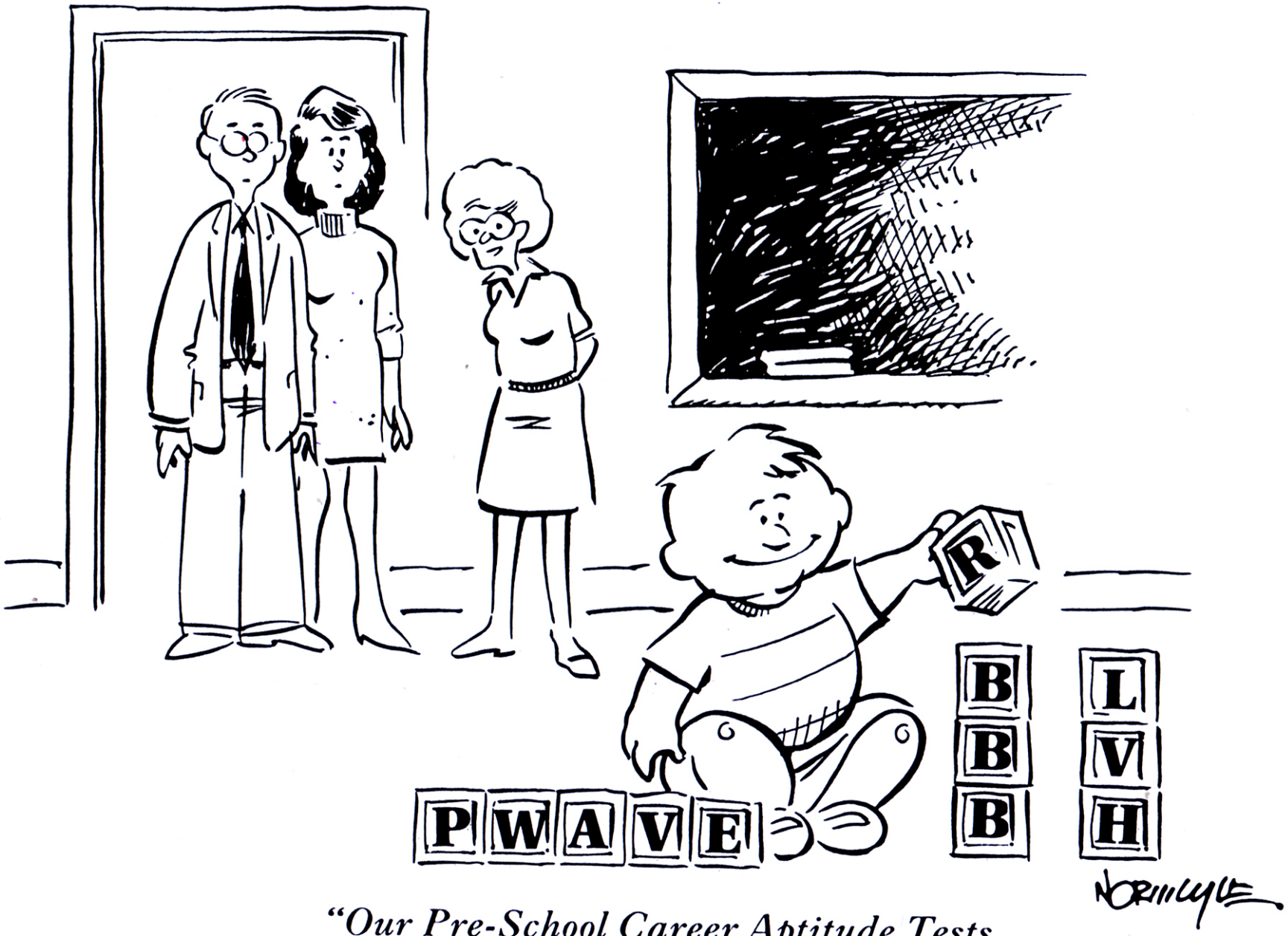

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