

ECG in the Athlete

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Italian Olympic Committee – Rome, Italy*



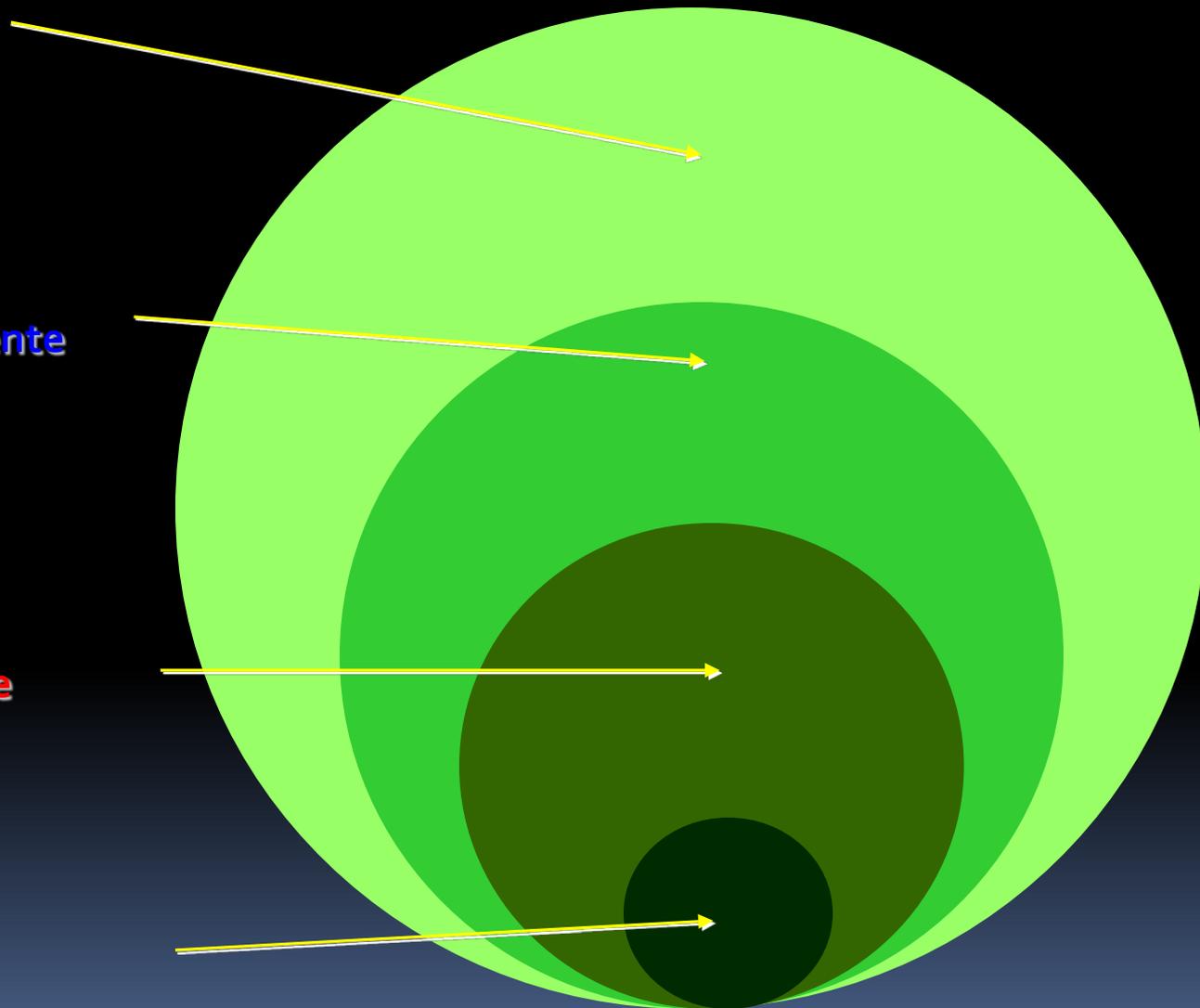
Obiettivo della Legge

**60 Milioni
Italiani**

11 Milioni
fanno sport occasionalmente

6 Milioni
S'allenano regolarmente e
gareggiano

~ 2.000
Atleti di alto livello



Dati CONI-ISTAT 2011

COMITATO ORGANIZZATIVO CARDIOLOGICO
PER L'IDONEITÀ ALLO SPORT
ANCE - ANMCO - FMSI - SIC - SIC SPORT



**Protocolli cardiologici per
il giudizio di idoneità
allo sport agonistico
2009**

Edizione del Ventennale

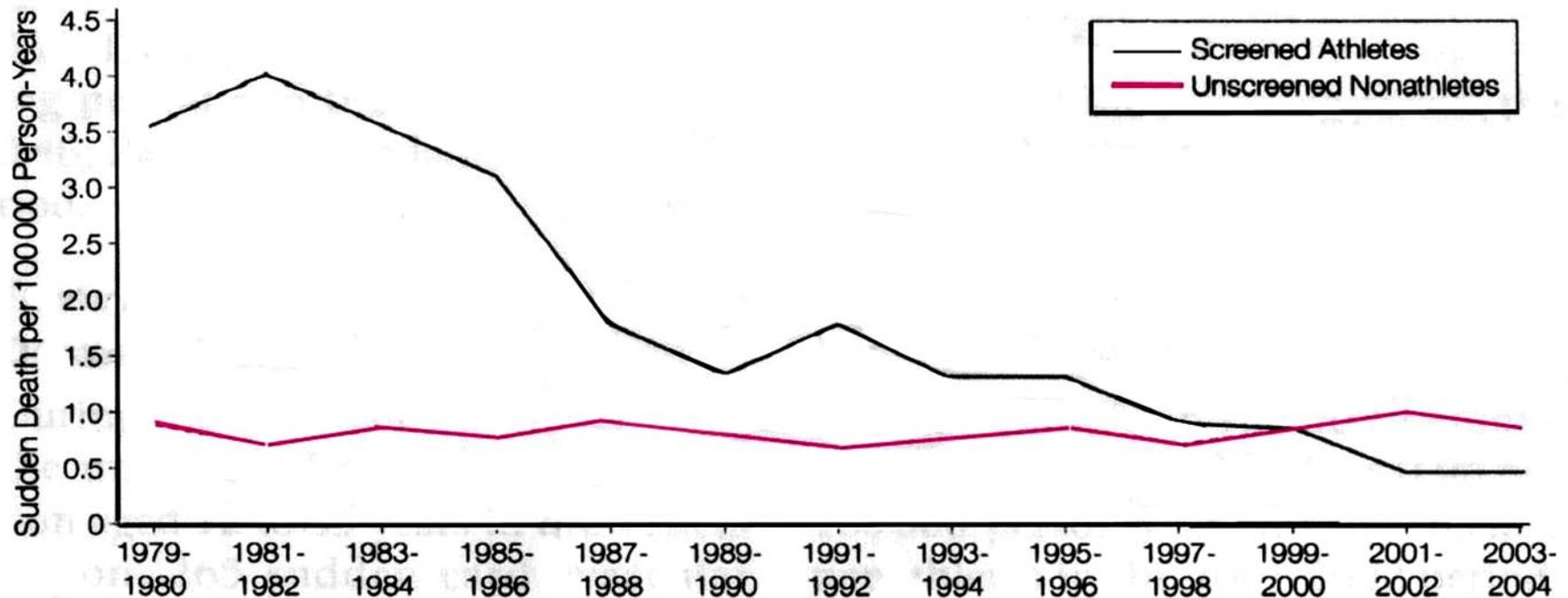


Casa Editrice Scientifica Internazionale

Trends in Sudden Cardiovascular Death in Young Competitive Athletes After Implementation of a Preparticipation Screening Program

D. Corrado et al.

Figure. Annual Incidence Rates of Sudden Cardiovascular Death in Screened Competitive Athletes and Unscreened Nonathletes Aged 12 to 35 Years in the Veneto Region of Italy (1979-2004)



During the study period, the annual incidence of sudden cardiovascular death decreased by 89% in screened athletes (P for trend $< .001$). In contrast, the incidence rate of sudden cardiovascular death did not demonstrate consistent changes over time in unscreened nonathletes.

How to interpret 12-lead ECG in athlete

- To provide cardiologists and sports medical doctors with a *appropriated and updated* approach to interpretation of ECG in the athlete. They have to be deeply informed about the clinically relevant information which can be obtained from ECG in the athlete.
- To *distinguish between normal physiologic patterns* that should cause no alarm and those that require action and/or additional testing to exclude (or confirm) the *suspect of an underlying cardiovascular conditions* at risk of sudden death during sports.
- For every ECG abnormality, it should be focused on the *ensuing clinical work-up* required for differential diagnosis and clinical assessment.

PERSPECTIVE

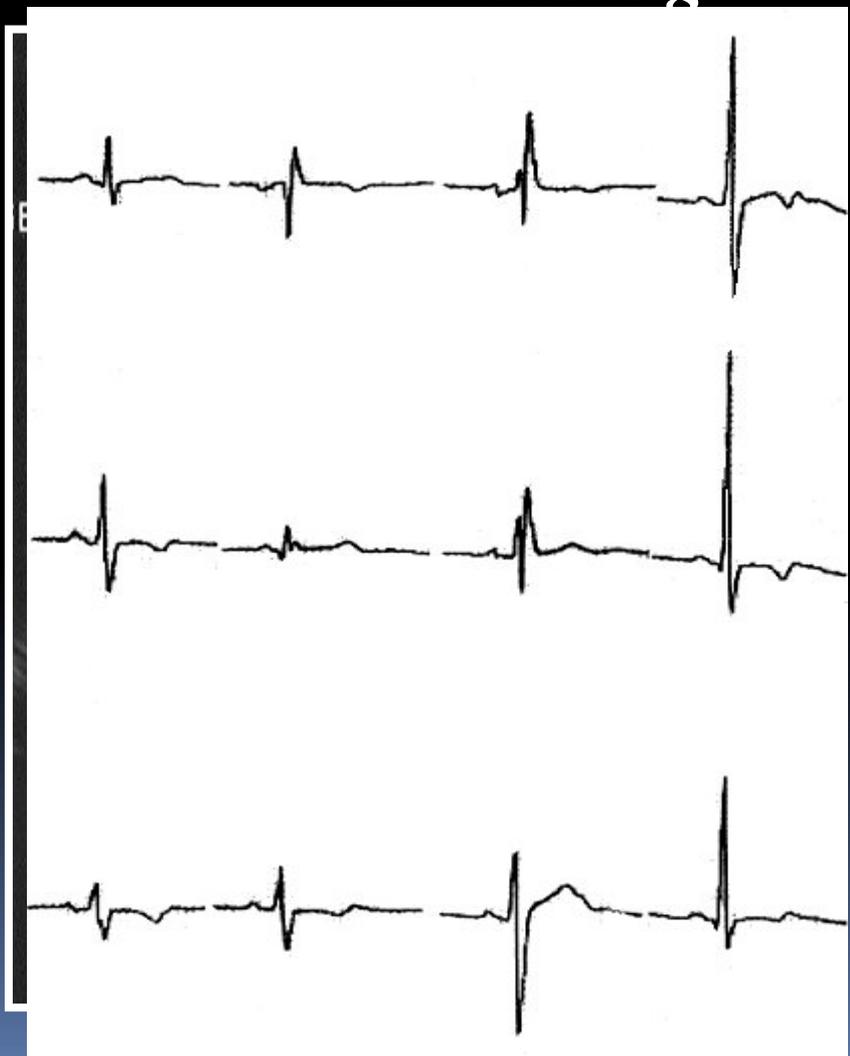
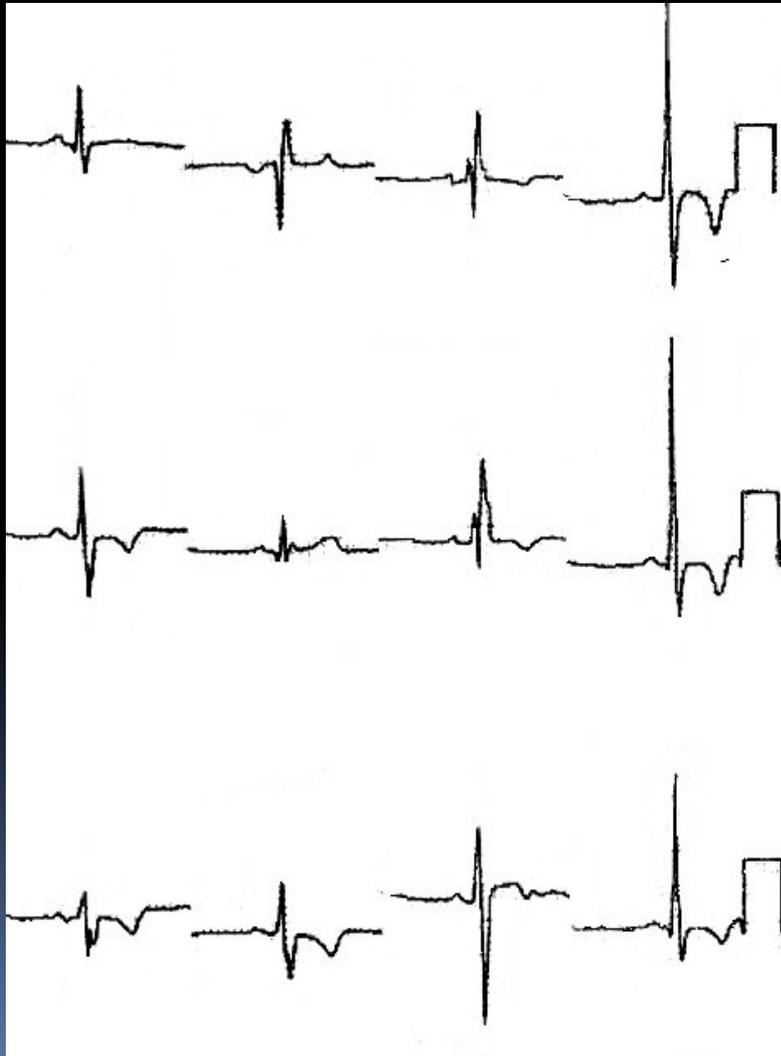
How to interpret 12-lead ECG in the athlete

- Appropriate interpretation of athlete's ECG requires the distinction of two main groups of abnormalities based on:
 - prevalence,
 - relation to exercise training and detraining,
 - inherent cardiovascular risk,
 - and need for further clinical investigation to confirm (or exclude) an underlying cardiovascular disease

same asymptomatic athlete
(20-year male soccer player)

Peak

Detraining



ECG abnormalities in the athlete

(Group 1)

Common (up to 80%)

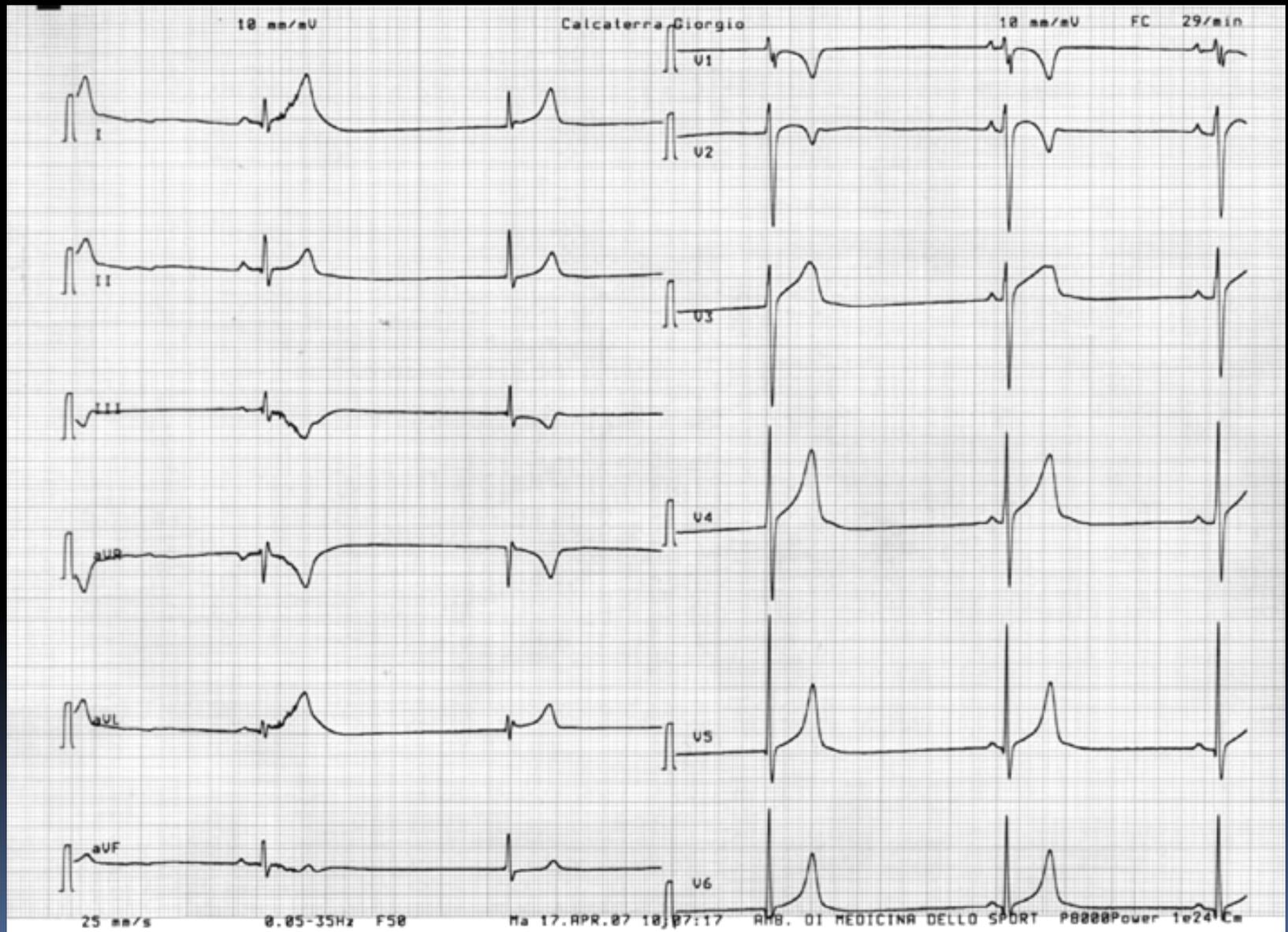
- Sinus bradycardia;
- First degree AV block;
- Notched QRS in V1 or incomplete RBBB;
- Early repolarization;
- Isolated QRS voltage criteria for left ventricular hypertrophy

(Group 2)

Uncommon (<5%)

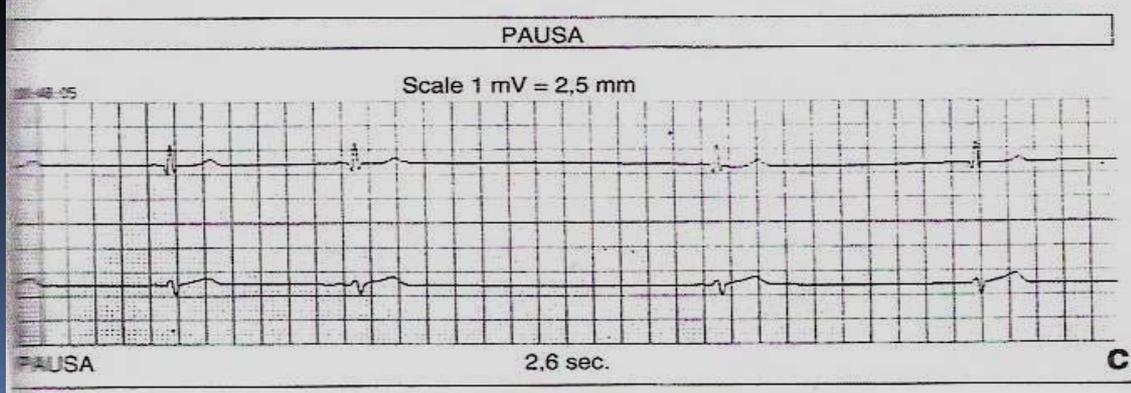
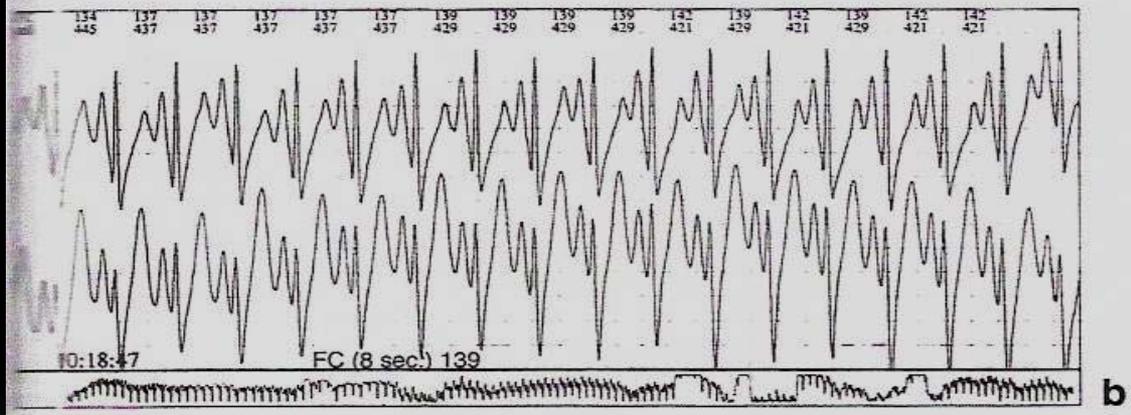
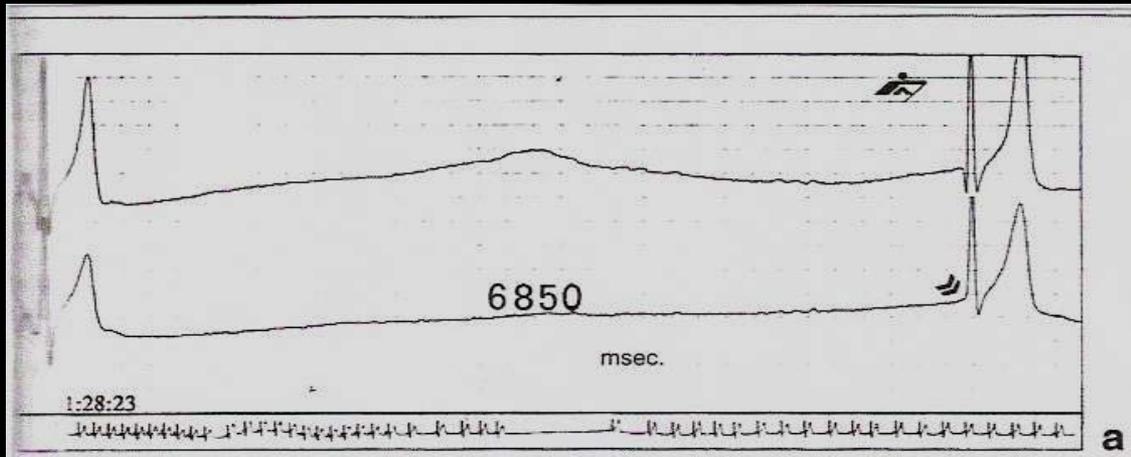
- T-wave inversion;
- ST-segment depression;
- Pathological Q waves;
- Left atrial enlargement;
- Left axis deviation/left Anterior hemiblock;
- Right axis deviation/left posterior hemiblock;
- Right ventricular hypertrophy;
- Complete LBBB or /RBBB;
- Long or short QT interval;
- Brugada-like early repolarization;
- Ventricular arrhythmias

Marathon Runner, 36 yrs, Sinus Bradycardia 29 bpm



Rower, 28 yrs
Prolonged nocturnal
Sinus pause
asymptomatic

After 3 months of detraining

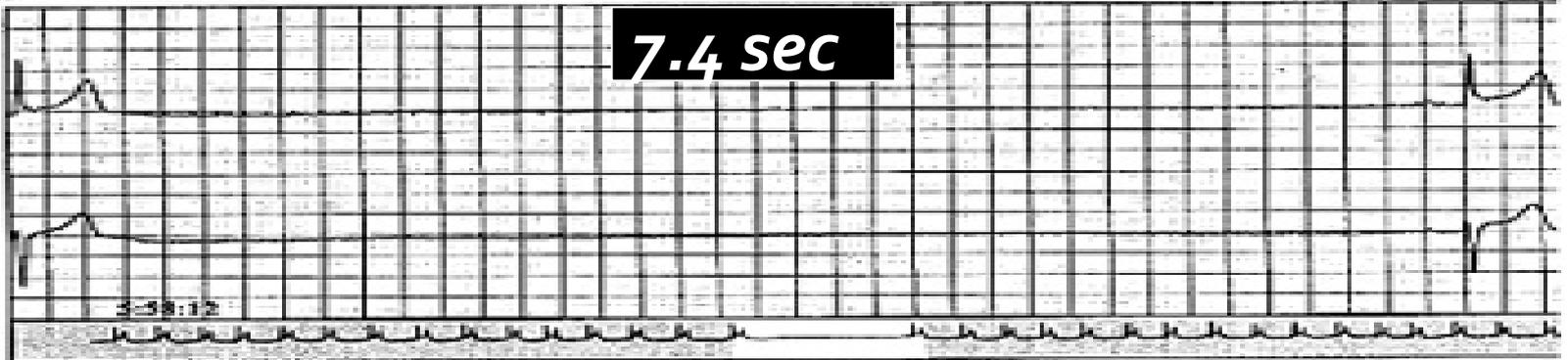


A

Striscia ECG AMPIEZZA 1.0

M. M. anni 26

18/03/1992

**B**

Striscia ECG AMPIEZZA 1.0

M. M. anni 26

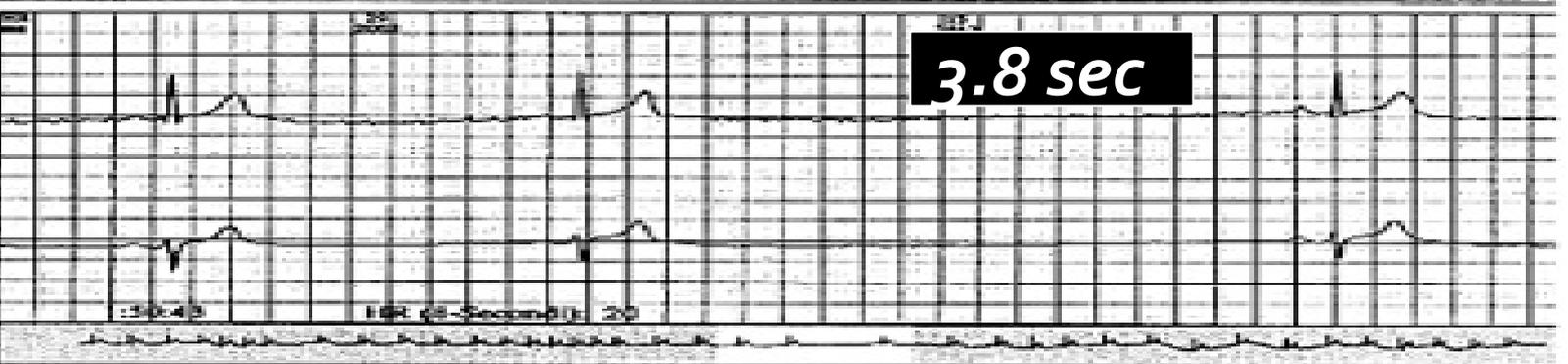
18/03/1992

**C**

Striscia ECG AMPIEZZA 1.0

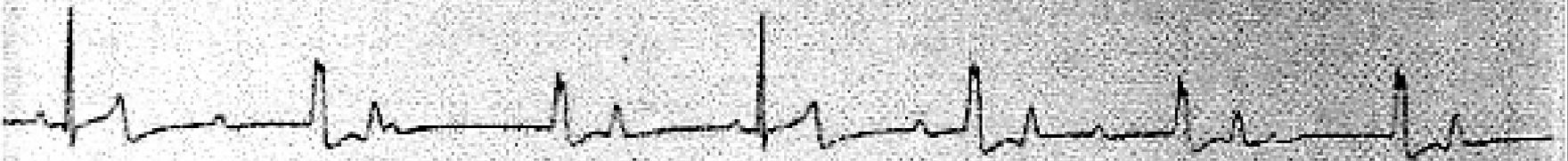
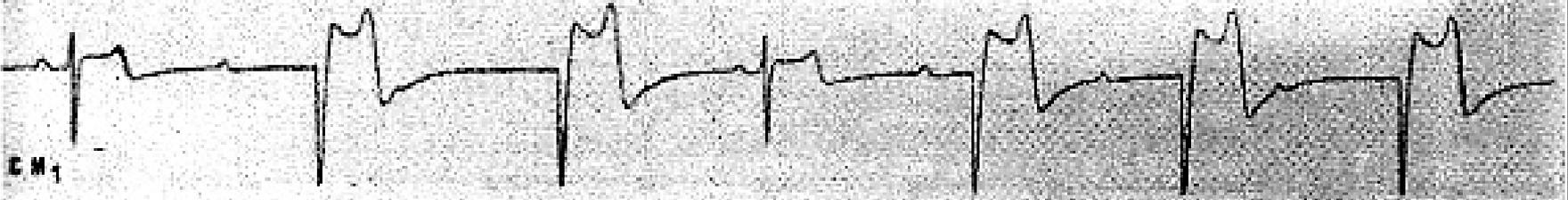
M. M. anni 26

23/03/1992

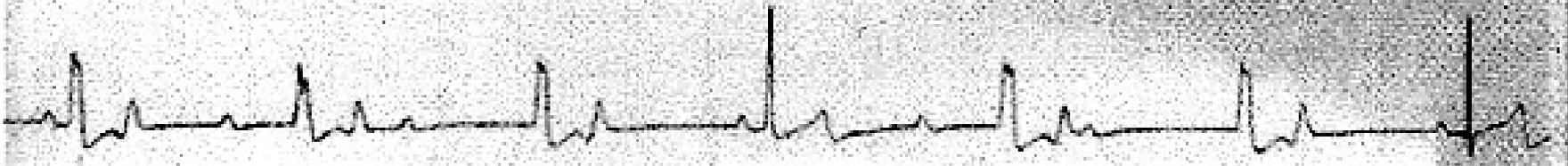
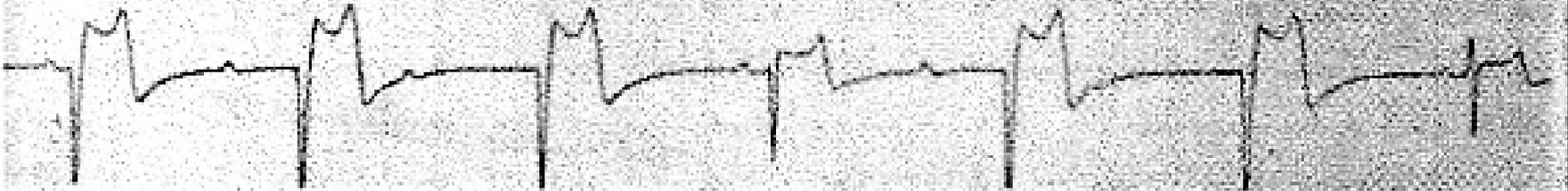


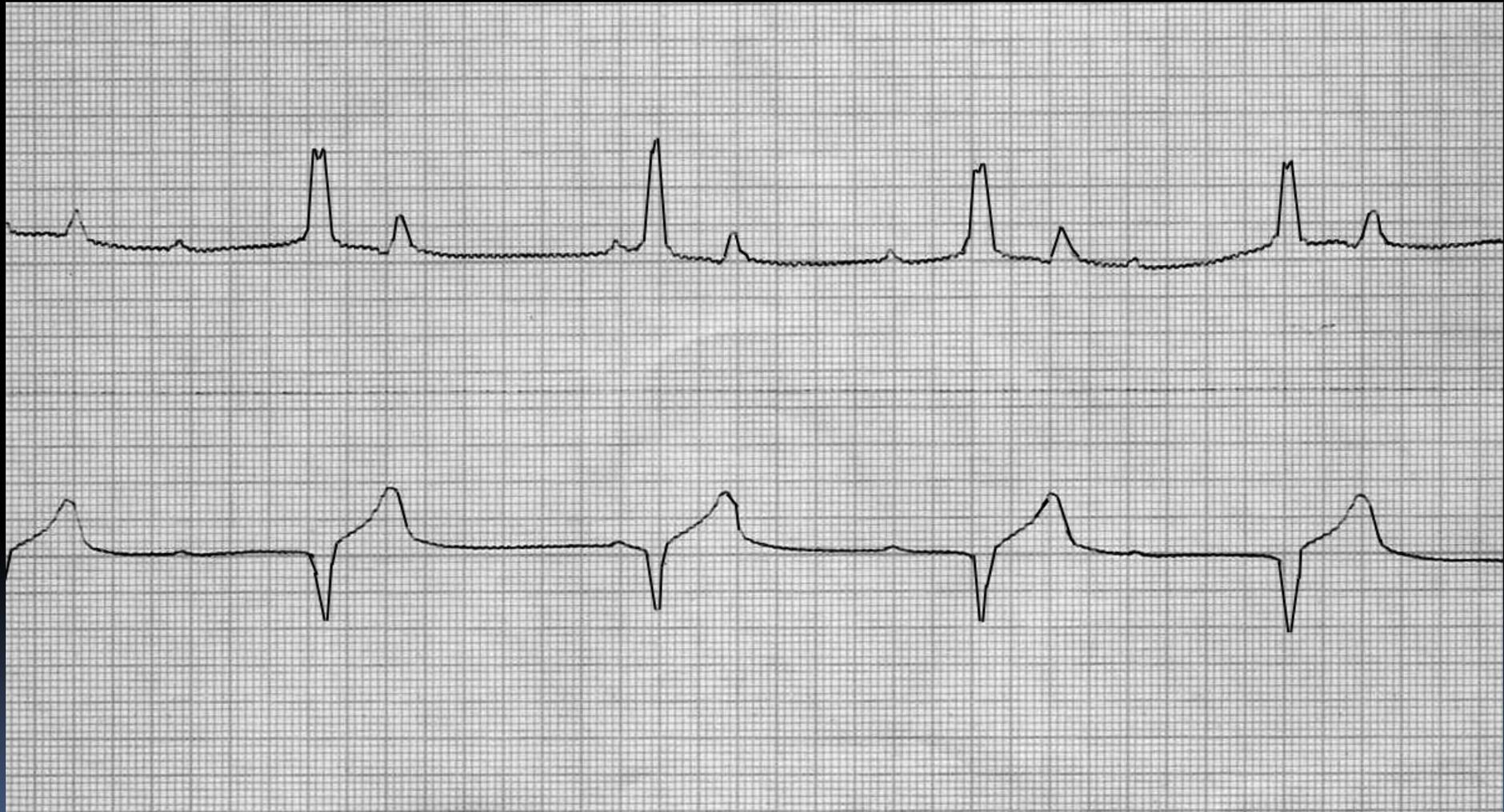
Asymptomatic water-polo player, 27 yrs

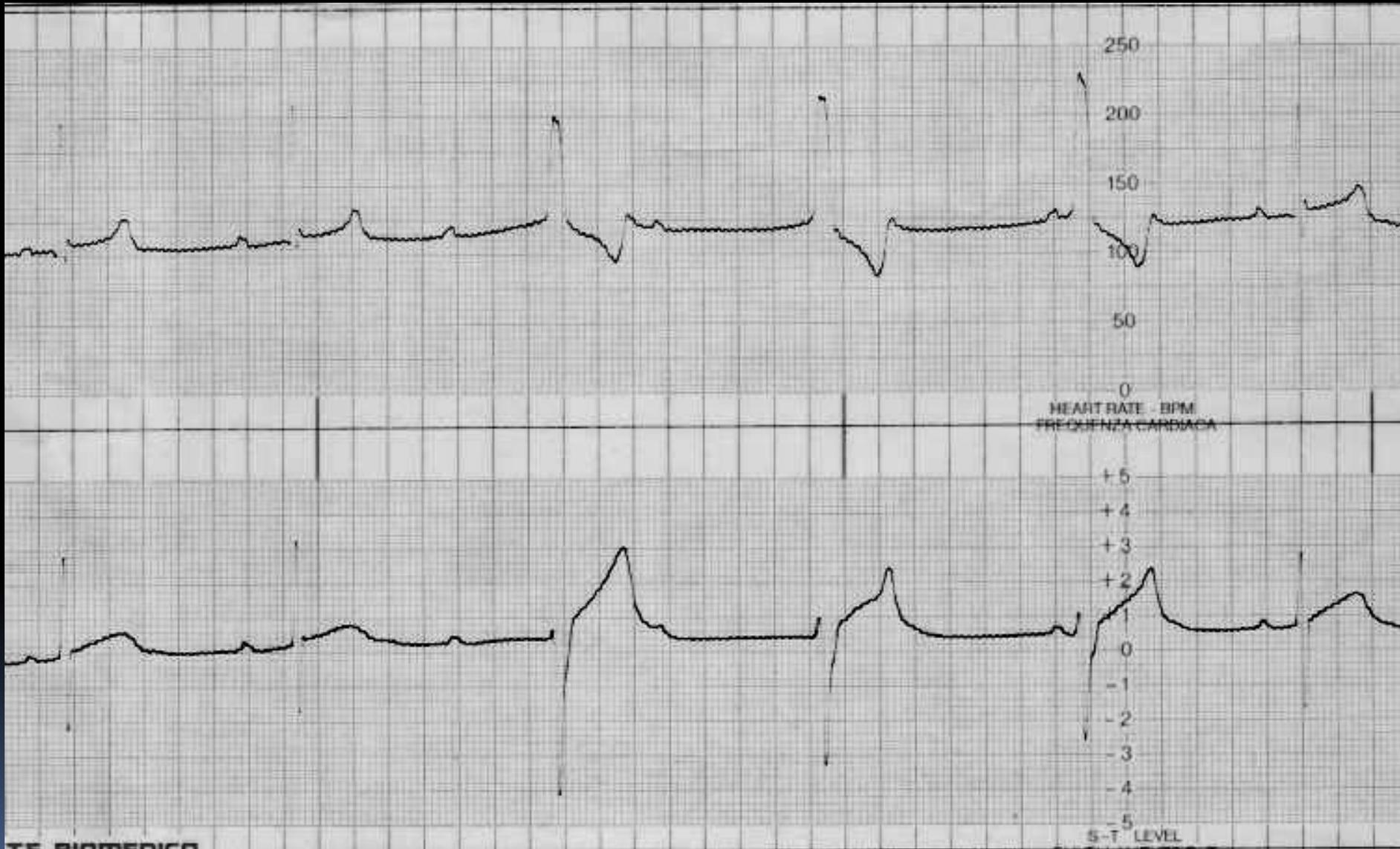
DURING SLEEP



Davis Cup International Tennis Player, 22 yrs !



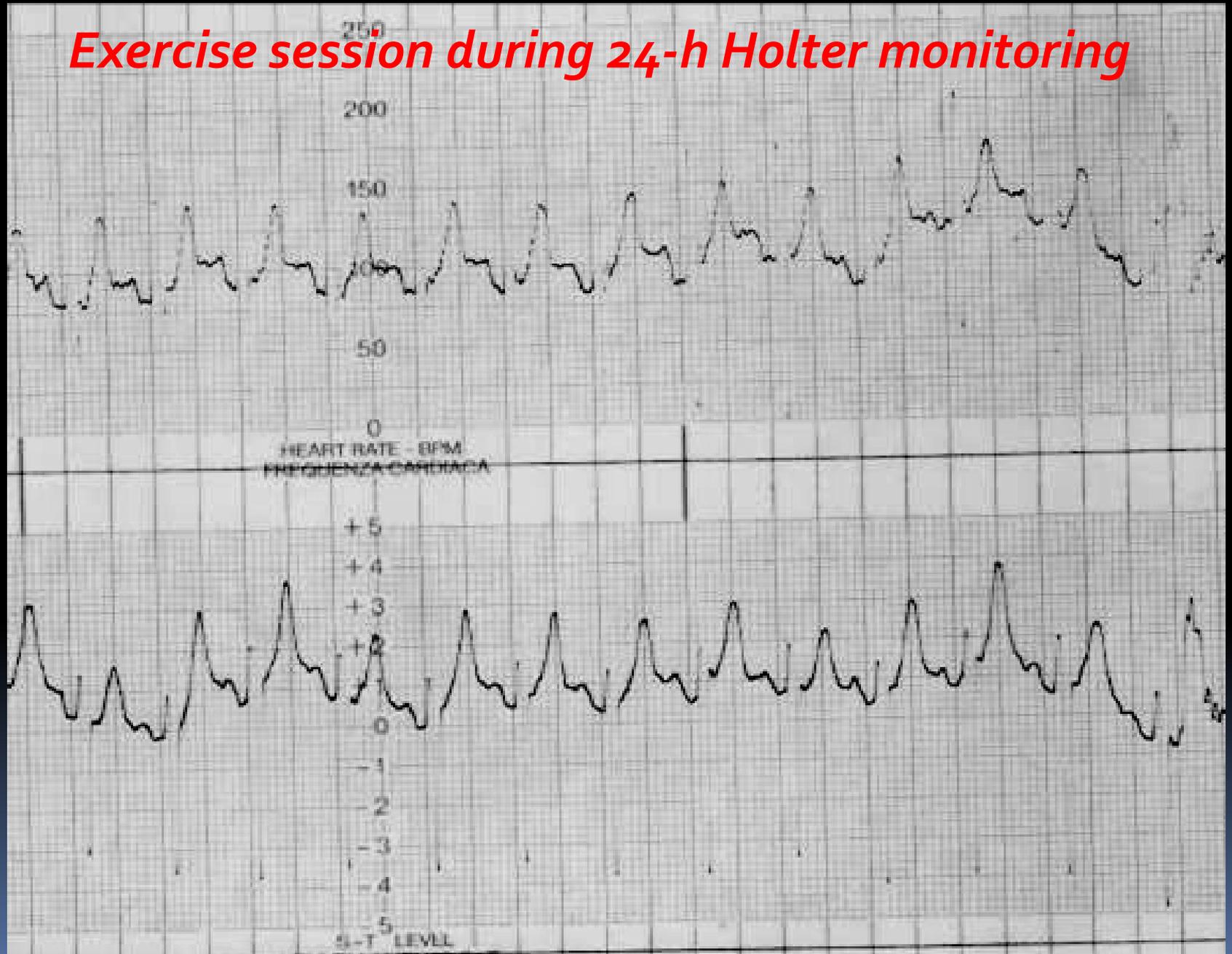




HEART RATE - BPM
FREQUENZA CARDIACA

S-T LEVEL

Exercise session during 24-h Holter monitoring



00197 - Roma, 4/XII/1980
Via dei Campi Sportivi, 46
Tel. 36851

- Prof. SERGIO DALLA VOLTA
Via Belzoni, 16
35100 PADOVA

Prot. 3720/AV/pm

Carissimo Dalla Volta,

da alcuni anni seguiamo un giovane tennista di alto livello, ~~di nome~~ ~~di cognome~~, il quale presenta a seconda del maggiore o minore grado di allenamento, un BAV di I, II o III grado, del tutto asintomatico.

Gli ultimi controlli sono quelli di cui ti allego i relativi referti. Noi vorremmo, a scopo di avere maggiori ragguagli ed anticipare eventuali obiezioni, avere conferma della nostra interpretazione funzionale attraverso uno studio elettrofisiologico endocavitario ed avrei molto piacere che ciò potesse avvenire presso il tuo Istituto.

Il ragazzo parte adesso per una tournée negli Stati Uniti e tornerà in Italia nei primi di gennaio; può essere a disposizione per eseguire tale indagine dal 15 al 22 gennaio p.v.

Ti sarei grato se potessi fissarmi un appuntamento e dirmi le modalità con le quali ciò potrà essere fatto.

Nell'attesa ti invio i più cordiali saluti e fervidi auguri di buon Natale e felice anno nuovo, unitamente ai tuoi valenti collaboratori.

Antonio Venerando



ISTITUTO DI MEDICINA CLINICA
dell'Università di Padova
Cattedra di Cardiologia
Direttore: Prof. S. Dalla Volta

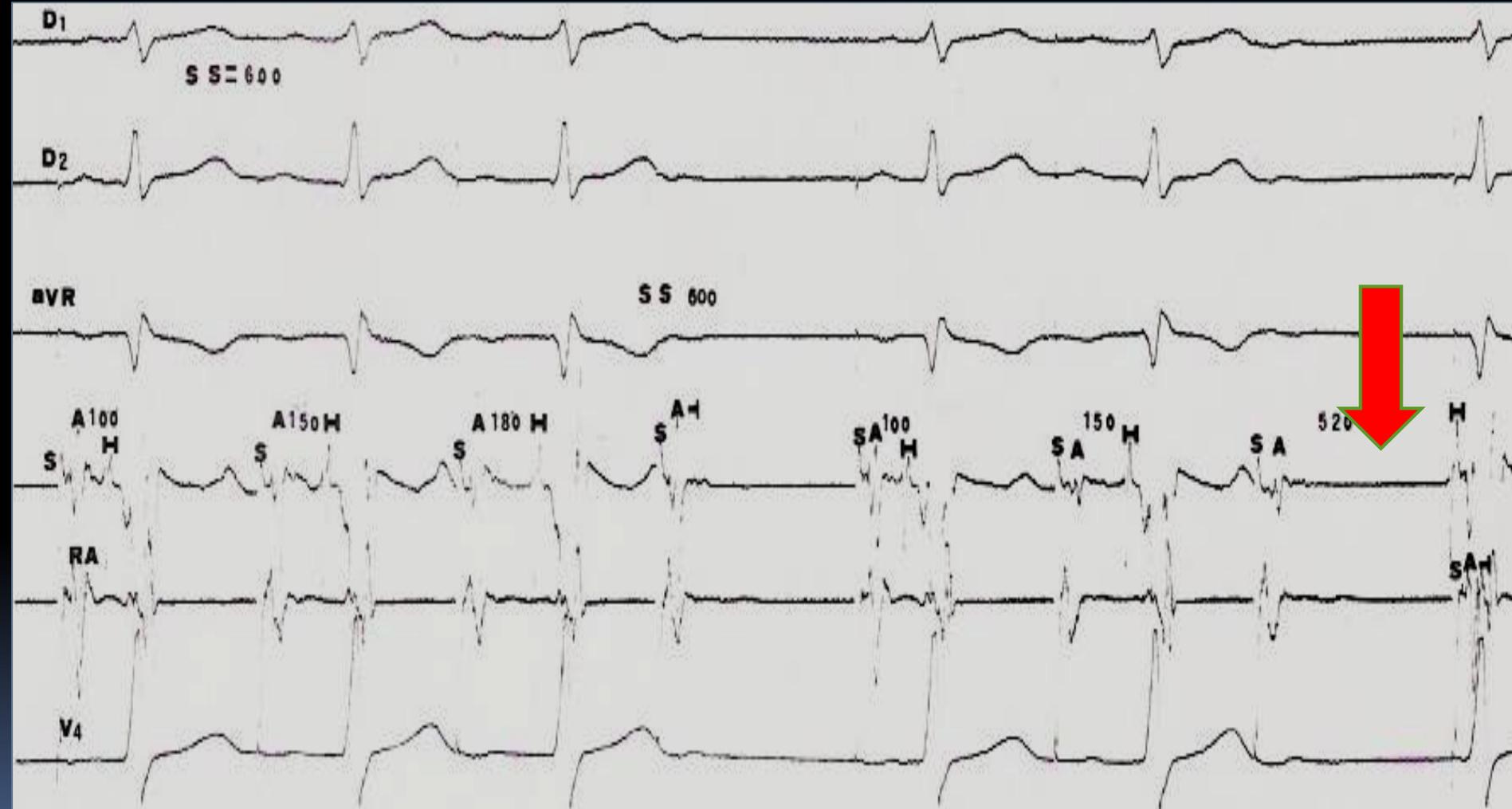
Ch.mo Prof. A. VENERANDO
ISTITUTO DI MEDICINA DELLO SPORT

**Risposta
di
Dalla Volta**

(Studio EFG)

Electrophysiologic study

Supra-hissian high-degree AV block



----- Nato/a il: Pressione: 0/0

diagnosi:

analisi del ritmo
ritmo sinusale normale

commento:

analisi della morfologia
blocco di branca destra incompleto

CONFEZIONE

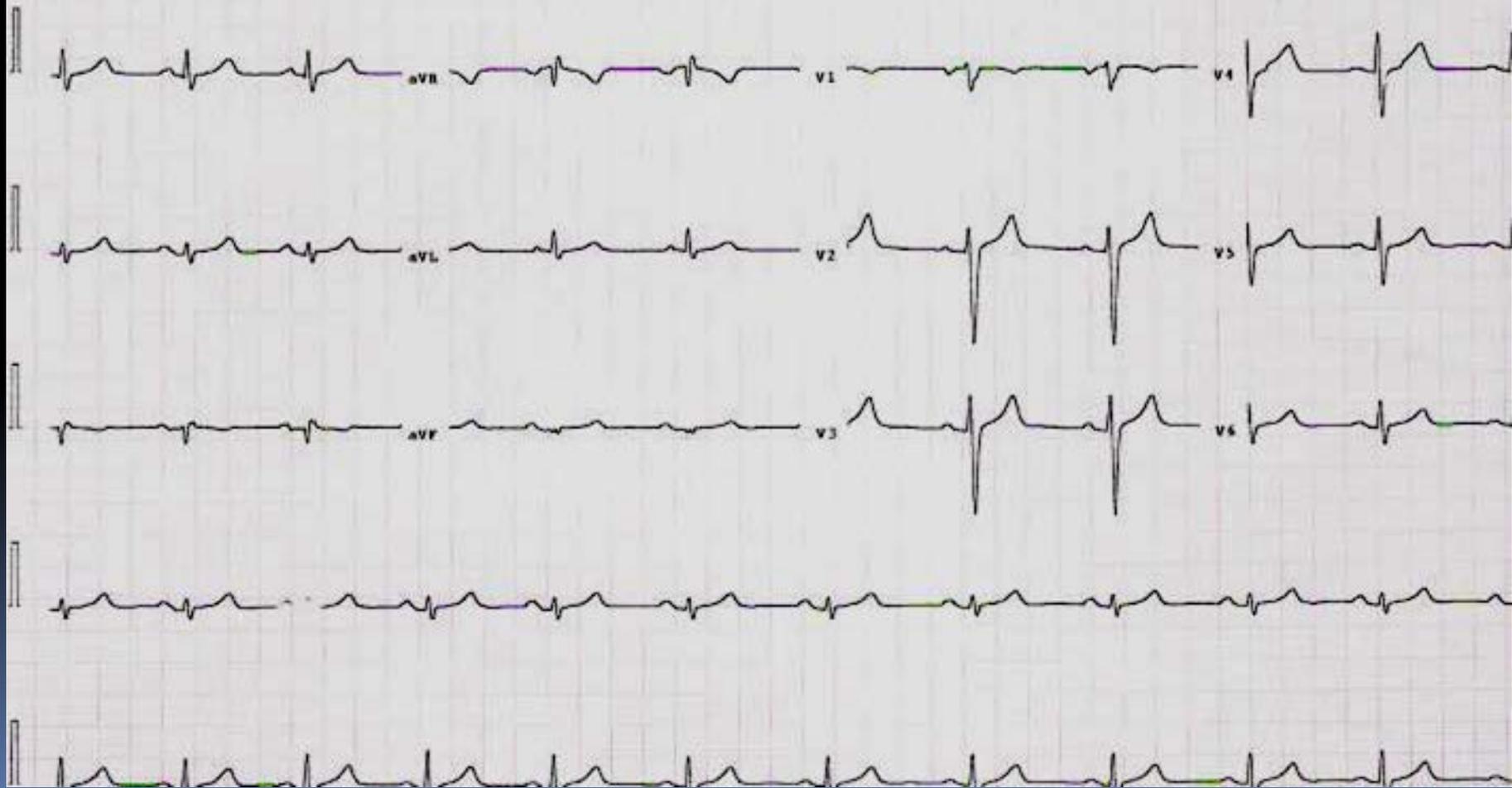
VALORI GLOBALI

PR: 152 ms
PRP: 372 ms
PRP: 403 ms

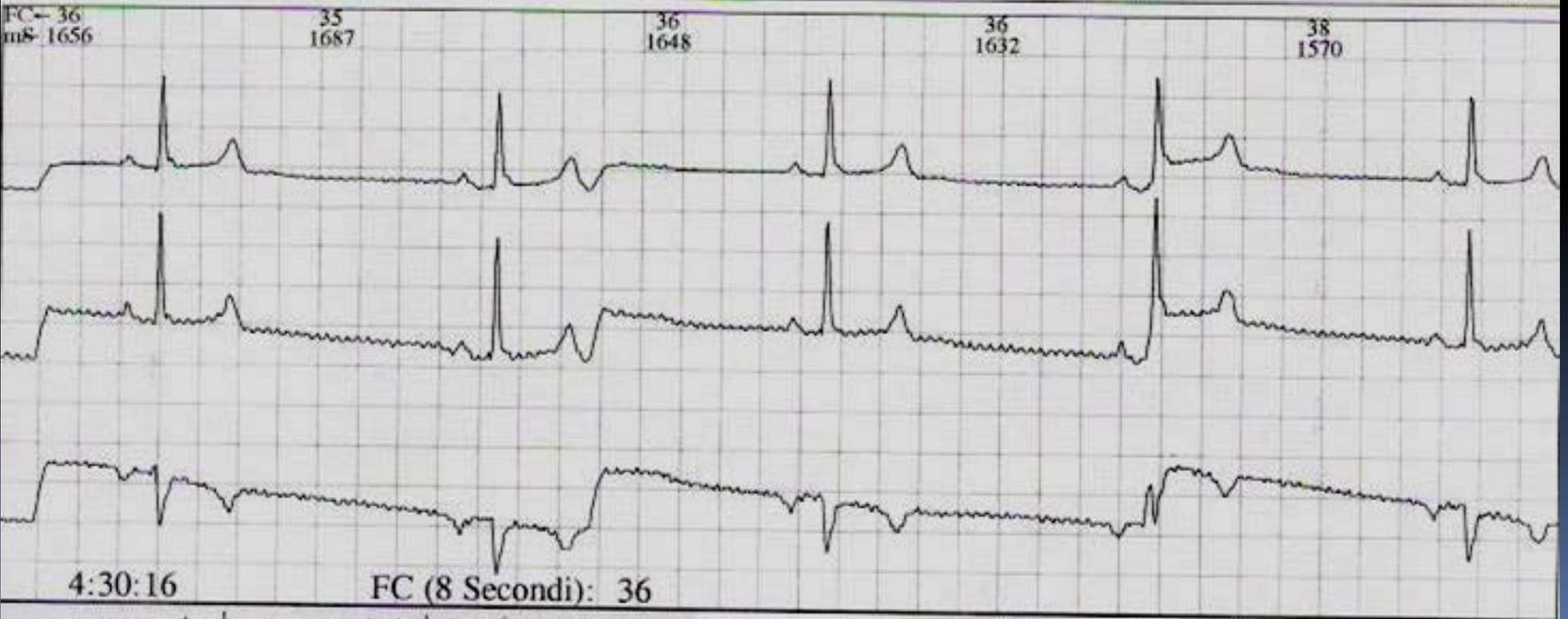
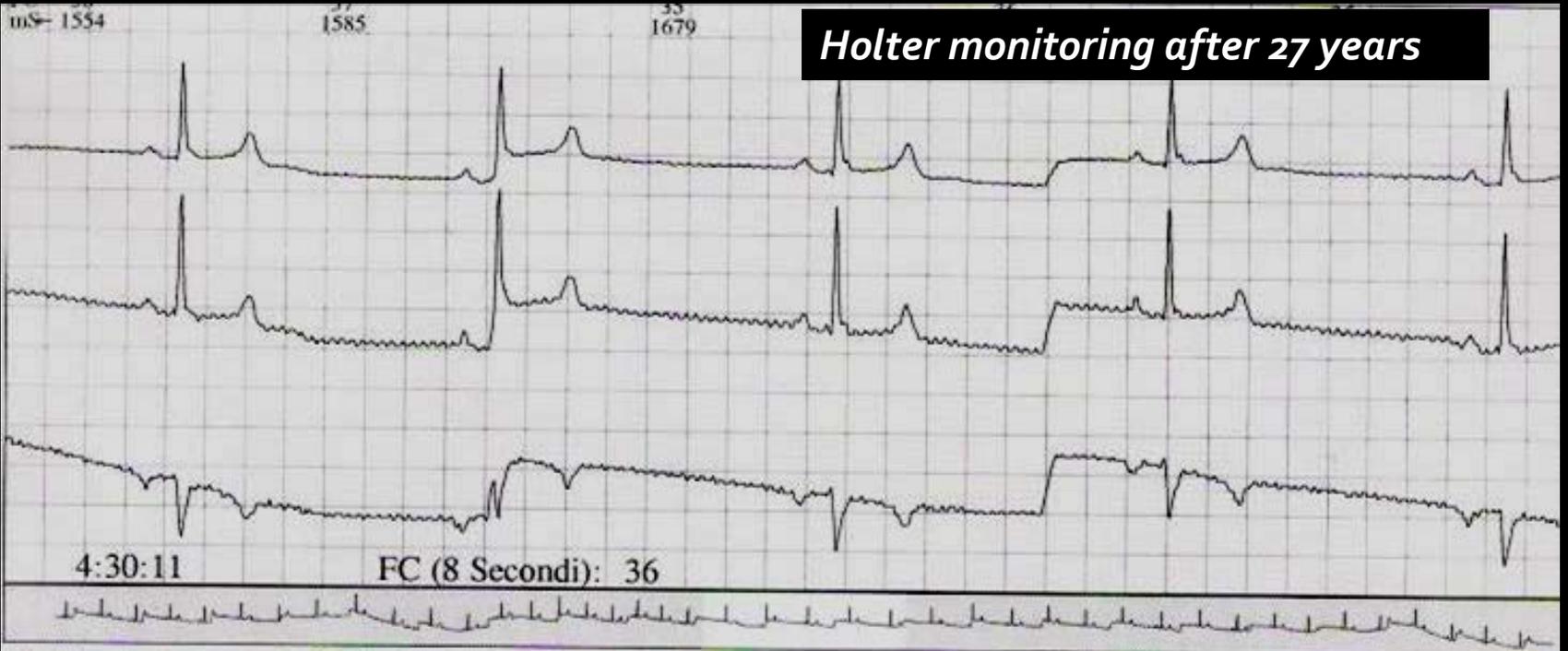
FC: 72 bpm
P: 116 ms
QRS: 102 ms

Asse P: 46
Asse QRS: 6
Asse T: 29

12-lead ECG after 27 years



Holter monitoring after 27 years

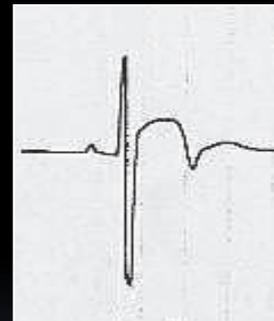


EARLY REPOLARIZATION

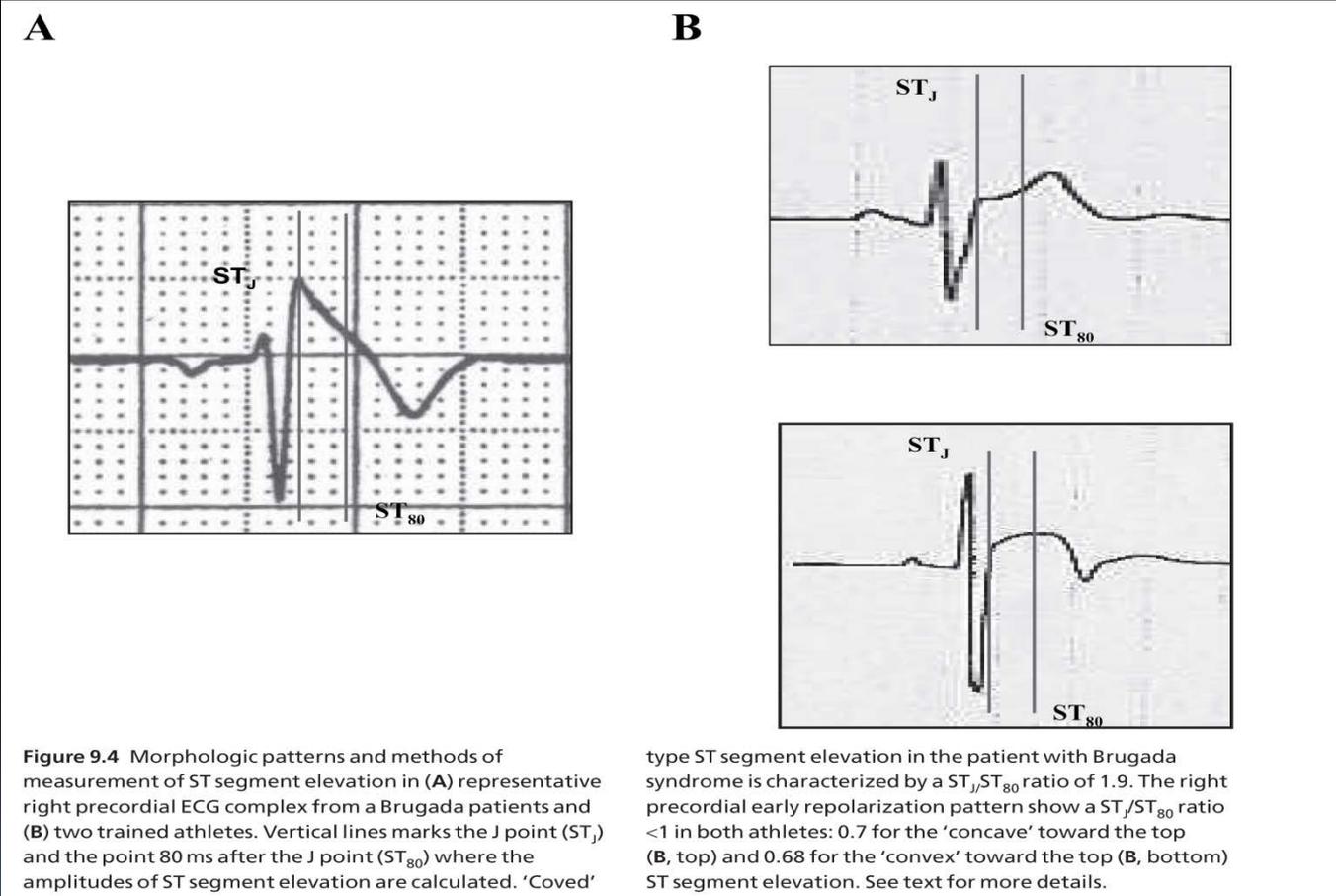
- Early repolarization has traditionally been regarded as an **idiopathic and benign** ECG phenomenon.
- The prevalence of this ECG pattern ranges between 1 and 2 % and is more commonly observed **in young individuals and athletes with a male preponderance**. Early repolarization has been noted to be present in 50% to 80% of resting athletes' ECGs, and is the rule rather than the exception in highly trained endurance athletes with sinus bradycardia.
- The most notable ECG feature is ST-segment elevation, that may vary on morphology, location and degree.

EARLY REPOLARIZATION

- The most common pattern (Type 1) is characterized by an elevated J point, often associated with notching or slurring of the terminal QRS complex ("J wave"), an upward concavity of the elevated ST-segment and a positive ("peaked and tall") terminal T-wave
- An other pattern of ER (type 2) consists of an elevated ST-segment which is convex on the top ("domed") and followed by a negative or small/indistinct T-wave; this type is most often observed in black people (African's ECG).
- ER is usually localized in precordial leads, with the greatest ST-segment elevation in mid-to-lateral leads (V₃-V₄). Maximal ST-segment displacement may also occur more laterally (leads V₅, V₆, L₁ and aVL), inferiorly (L₂, L₃ and aVF) or anteriorly (leads V₂-V₃).



The finding of right precordial ST-segment elevation in a healthy young trained athlete, particularly a type 2 pattern with a domed upward ST-segment followed by a negative T-wave, may raise clinical suspicion of a potentially lethal **Brugada syndrome** and the need for a differential diagnosis.



The ECG pattern of the early repolarization shows distinctive features allowing accurate distinction from the ECG abnormalities of patients with Brugada syndrome. Such ECG criteria include the J-ST-T waveforms, the amplitude of maximum ST segment displacement at J point (ST_j) and after 80 ms (ST_{80}), and ST_j/ST_{80} ratio.

EARLY REPOLARIZATION

- The magnitude of ST-segment elevation is characteristically modulated by **autonomic influences**, **heart rate changes** and **drugs**; this explains the dynamic nature of the ECG abnormalities and a waxing and waning of the ST-T segment over time.
- **Slowing of heart rate exaggerates ST-segment elevation**, whereas **sinus tachycardia** occurring during exercise or after isoproterenol reduces and **often eliminate ER changes**.
- Accordingly, the mechanisms underlying ER in trained athletes are characteristically related to the **hypervagotonia** which result from athletic training, so that ER is a reversible phenomenon which disappears with deconditioning.

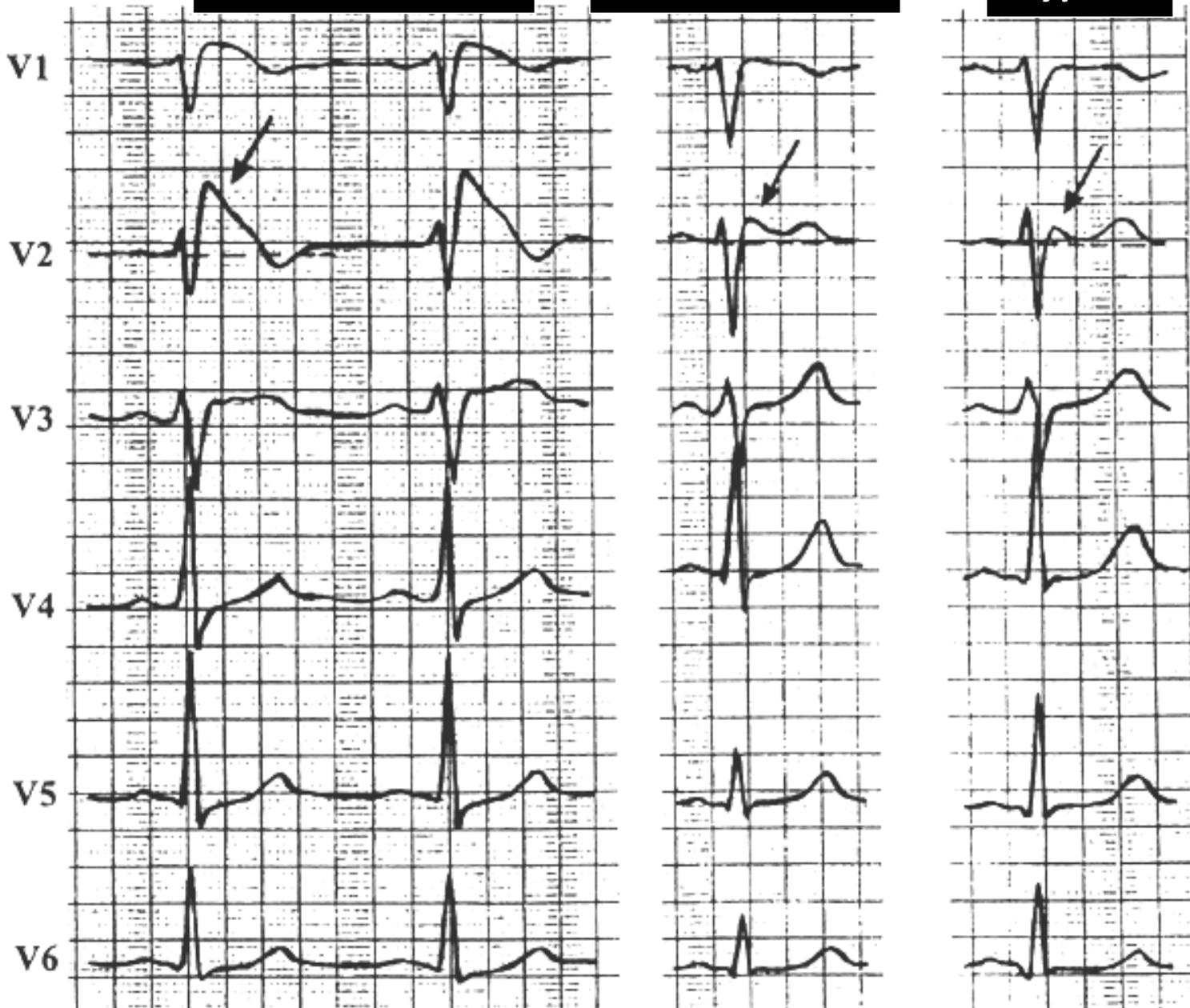
5/2/99

13/2/99

Type 1

Type 2

Type 3



Early rep. in Athletes

- **Up-sloping ST**
- **High positive T waves**
- **QRS high voltages**
- **Normal QRS duration**
- **Increase** after β -blockage, bradycardia, etc.
- **Decrease** after β -stimul., tachycardia, exercise, etc.

Brugada

- **Down-sloping ST**
- **low negative T waves**
- **QRS low voltages**
- **Prolonged QRS**
- **Increase** after β -blockage, bradycardia, etc.
- **Decrease** after β -stimul., tachycardia, exercise, etc.

Physiologic vs pathologic LVH

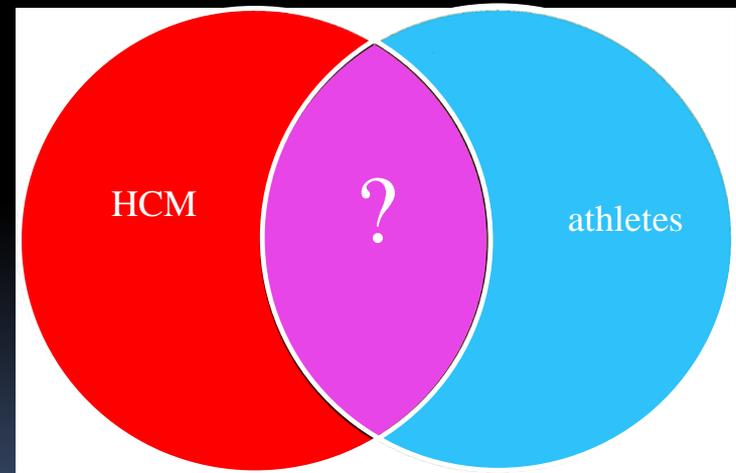
- In order to differentiate physiologic from pathologic LV hypertrophy, it is important to know whether differences exist between:
 - the ECG pattern associated with physiologic remodelling in the context of "athlete's heart" and
 - the ECG abnormalities occurring in structural heart diseases manifesting with an increased LV wall thickened, including hypertrophic cardiomyopathy (HCM), aortic valve diseases, or hypertensive heart disease.

OBJECTIVES

AHA Scientific Sessions 2007

□ The purpose of the study is to assess whether ECG abnormalities of pathologic hypertrophy (HCM) overlaps with those of physiologic hypertrophy (athlete's heart).

□ The ECG analysis was focused on what proportion of both groups presented with the ECG pattern of **isolated voltage criteria for LVH**.



HCM
(n. 260)

- Age: 42 years (3 – 82 ys)
- Sex: 184 male (71%)
- DNA analysis: 48 pts (18%)
- Max LV WT: 22 mm \pm 6
(range 16 – 46 mm)
- LV EDD: 43 mm \pm 10
- Obstruction: 22% (57 pts)

ATHLETES
(n. 1005)

- Age: 23 years (9 - 55 ys)
- Sex: 745 male (74%)
- Race: 1003 Europeans, 2 Africans
- Sport discipline: 38
- Training program: median of 7 years (2-30 ys)
- Max LV WT: 10.7 \pm 4
- LV EDD: 54 mm \pm 6

STUDY POPULATIONS & DEMOGRAPHICS

RESULTS: ECG PATTERNS IN HCM PTS AND IN ELITE ATHLETES

AHA Scientific Sessions 2007

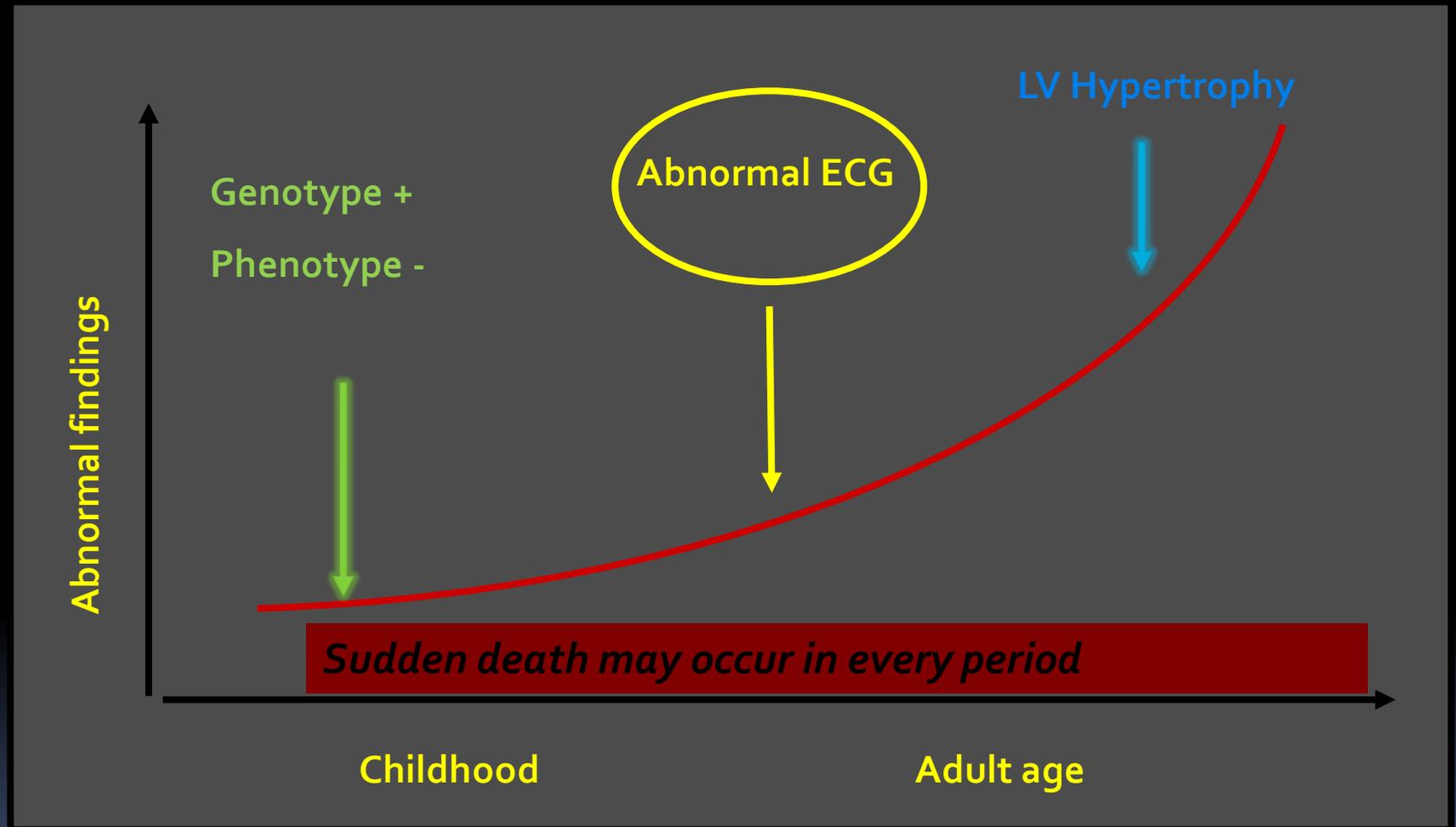
ECG pattern	Athletes n=1005 (%)	HCM n=260 (%)	p value
Isolated increase of QRS voltages	403 (40)	5 (1.9)	<0.0001
Nonvoltage criteria of LVH	13 (1.3)	155 (59.6)	<0.0001
ST/T repolarization abnormalities	27 (2.7)	209 (80)	<0.0001
Pathologic Q waves	17 (1.7)	103 (39.6)	<0.0001

CONCLUSIONS

AHA Scientific Sessions 2007

- The vast majority of patients with HCM have an abnormal ECG, with left ventricular hypertrophy associated to repolarization changes and/or pathological Q waves, as well as left atrial enlargement and conduction disturbances.
- Instead, trained athletes usually show an isolated increase of QRS amplitude, right QRS axis deviation, normal atrial and ventricular activation patterns and normal ST-T repolarization.

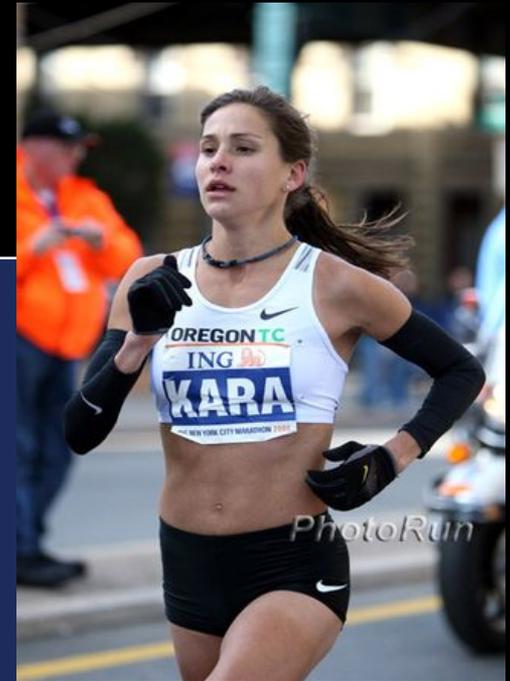
HCM diagnosis in Young Athletes



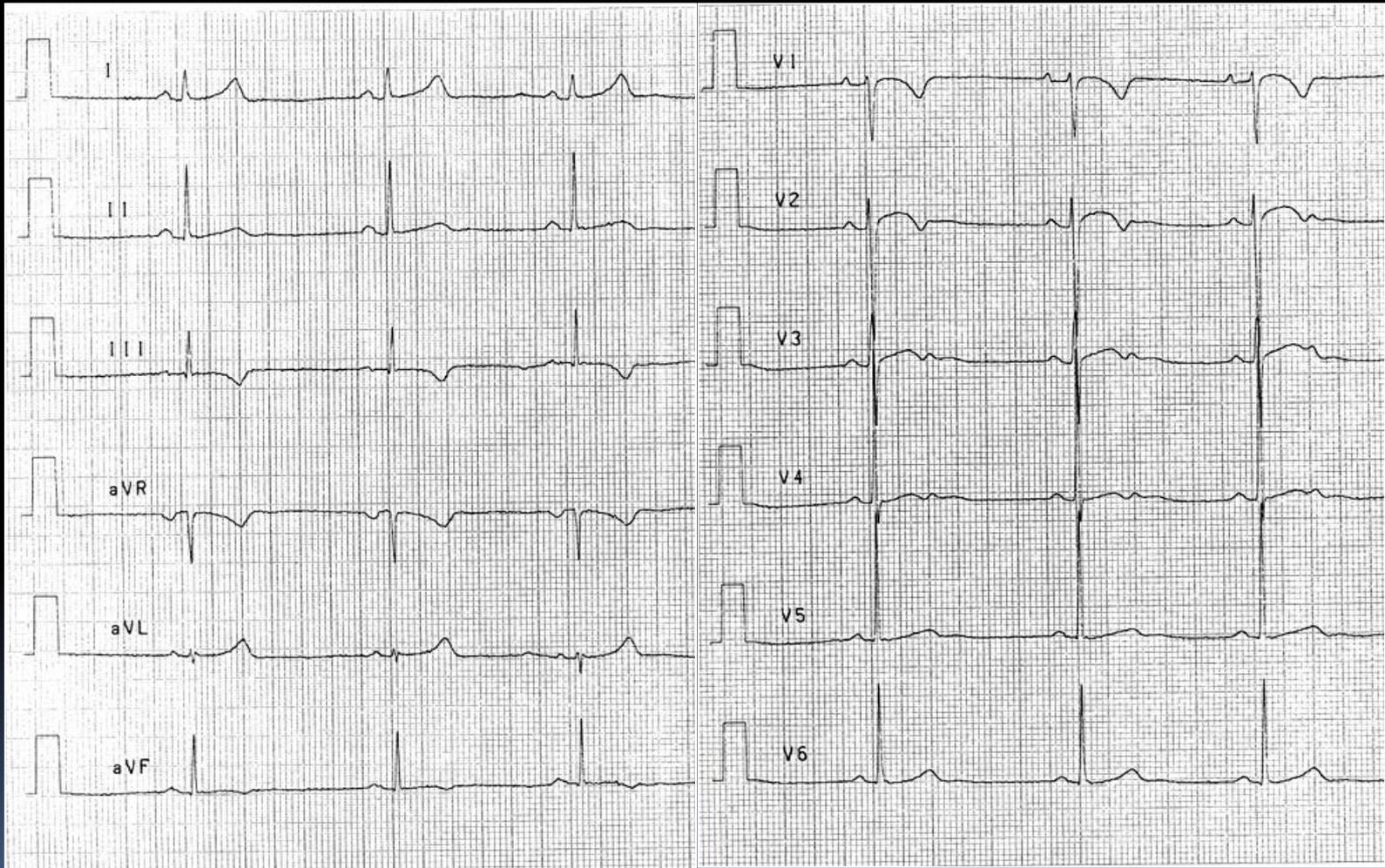
ECG is abnormal before the appearance of hypertrophy

Female 47 yrs, runner

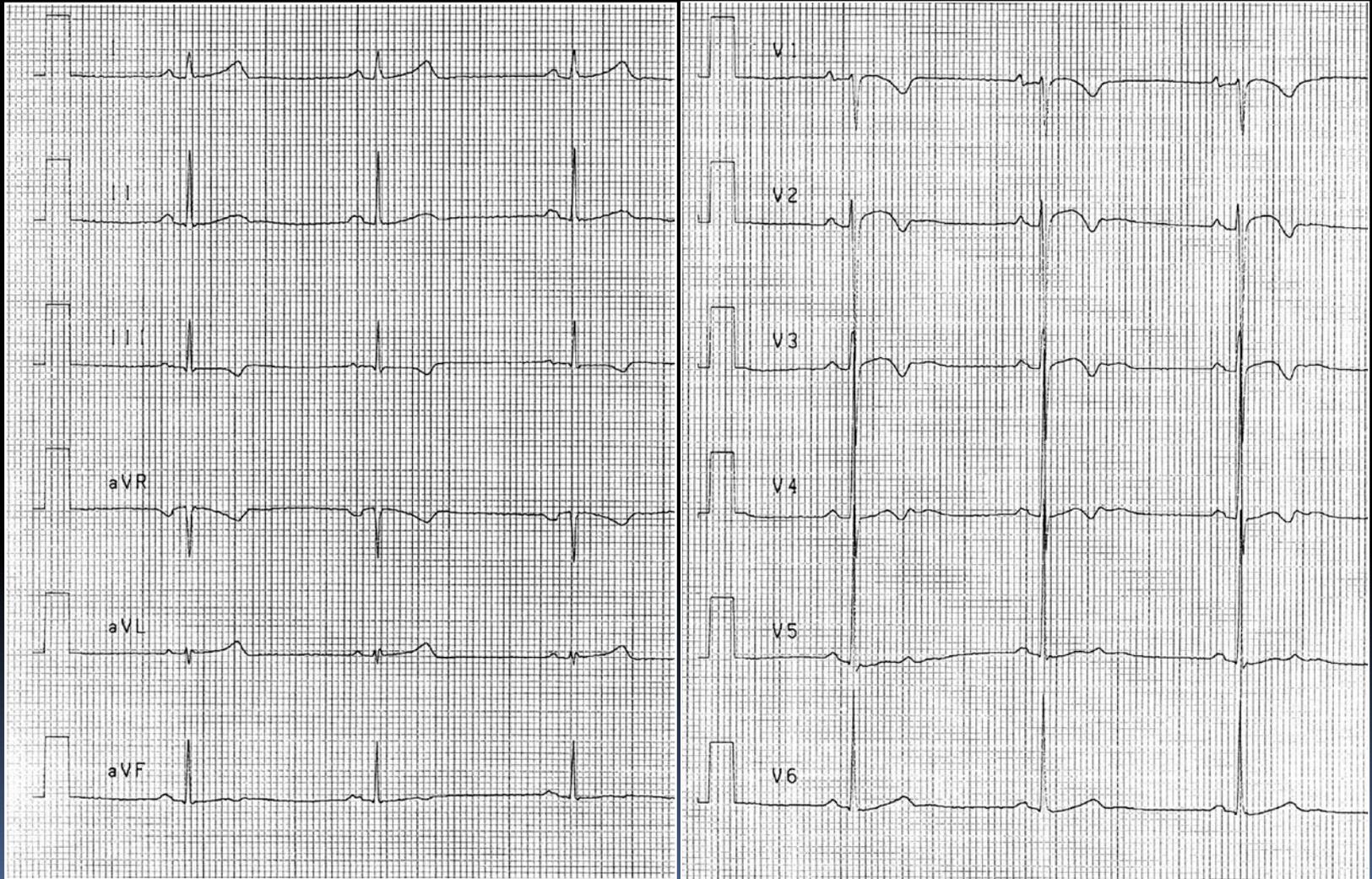
- Height 160 cm; weight 56 Kg;
- Asymptomatic
- Family history negative
- Request for echo because of negative T wave
- PE negative; BP: 115/75mmHg



12-lead ECG in 2008



12-lead ECG in 2010



PHILIPS 10/03/2011 17:03:58 TISO.8 MI 1.4
10011720110310 S5-1/AdultI

FR 50Hz
15cm
2D
52%
C 49
P Bassa
AGen



46 bpm

- LV Dd 47 mm
- AVS 12 mm
- Pfw 11 mm
- LA 45 mm
- EF 70%

PHILIPS 10/03/2011 17:10:30 TISO.8 MI 1.4
10011720110310 S5-1/AdultI

FR 47Hz
17cm
2D
62%
C 49
P Bassa
AGen

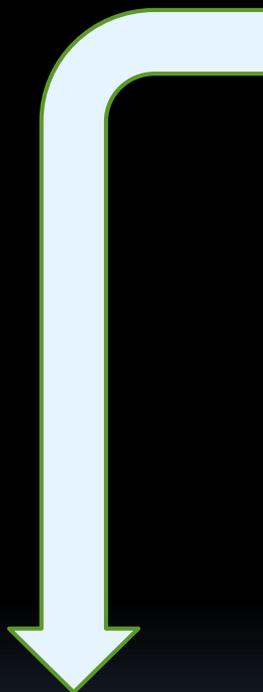


46 bpm

10/03/2011 17:06:02 TISO.8 MI 1.4
S5-1/AdultI



48 bpm

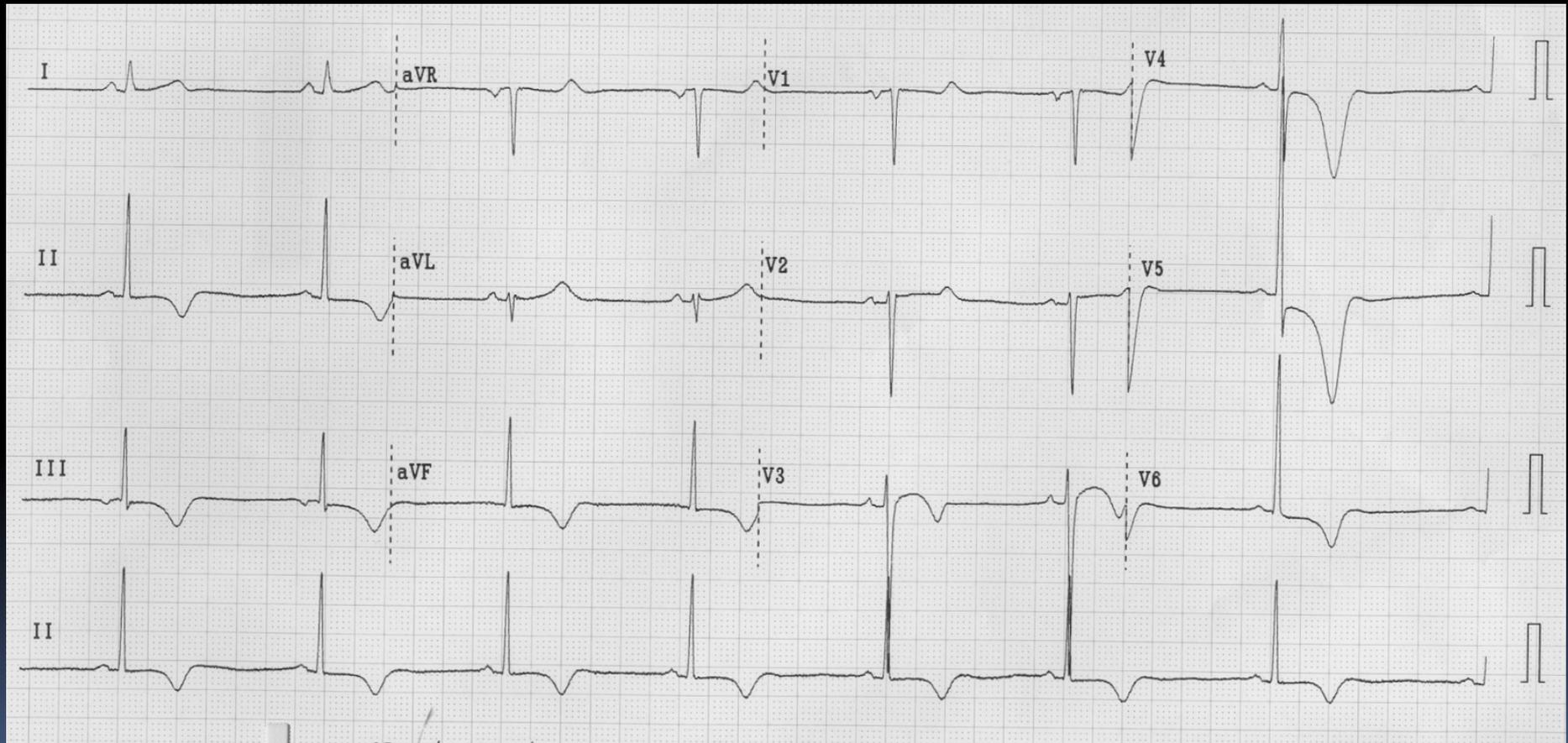


Athlete's heart



HCM

12-lead ECG in 2011

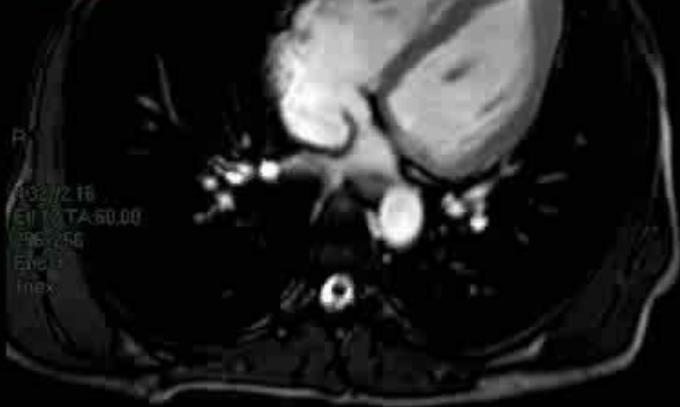


DE VILLA LAJIRA
46S 4M F 444612
11/11/1964
St.61
Pos: 24.00 mm
Dof: Obi

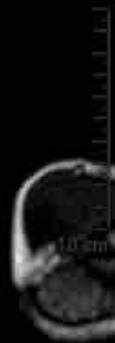
Con perdita

Policlinico Casilino
2400002 - AMBULATORIO
06/04/2011 09.10.20
LF: 8.00 mm
234% Pixel

Ec1
Fr 1
Patient Pos: HFS
Study Desc: RM Cardiaco
Series Desc: B-TFE_BH
< 1201 - 61 >



R
132/2 18
EI TA: 60.00
256/256
Enc 3
Inex



PS

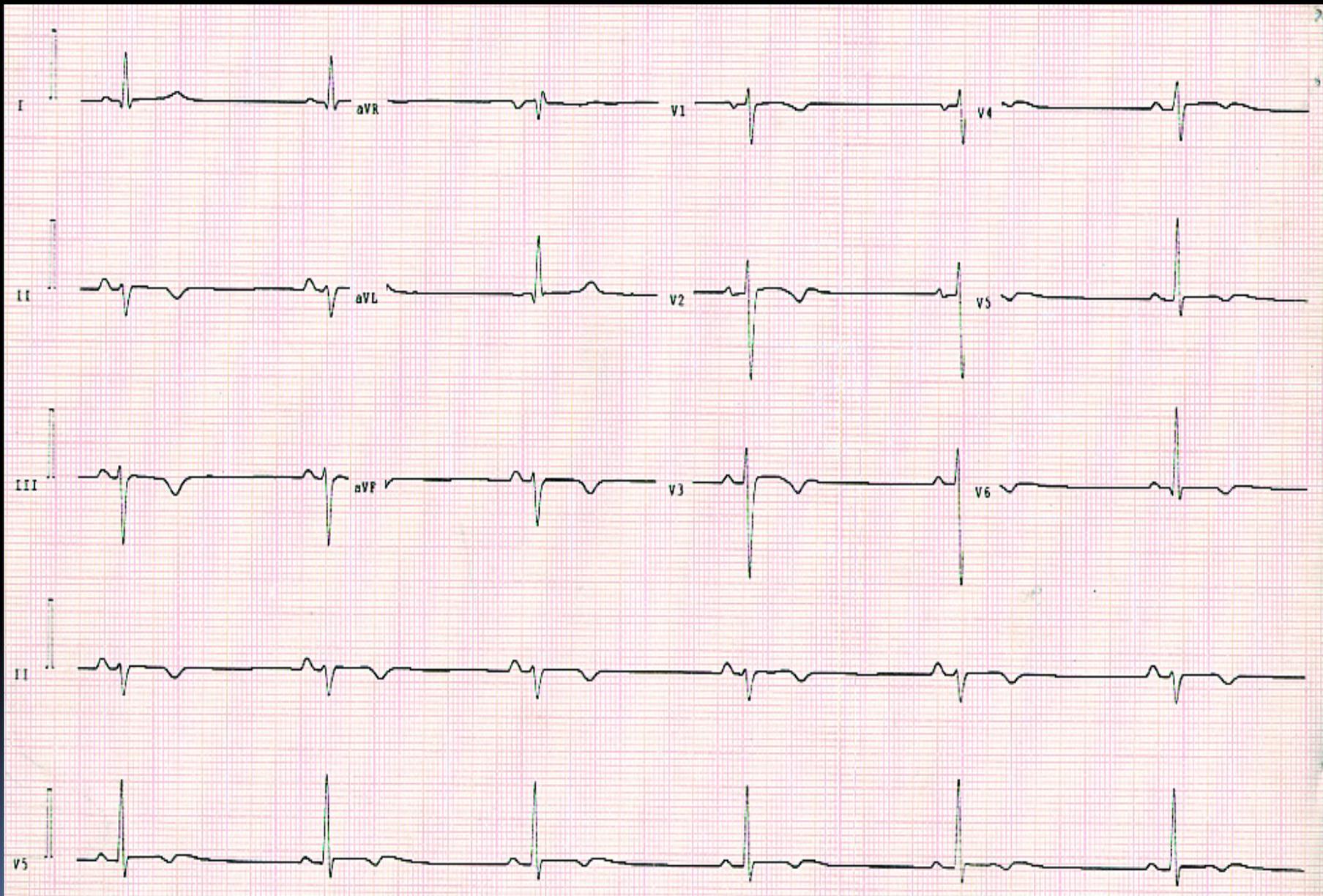
C: 357
W: 622



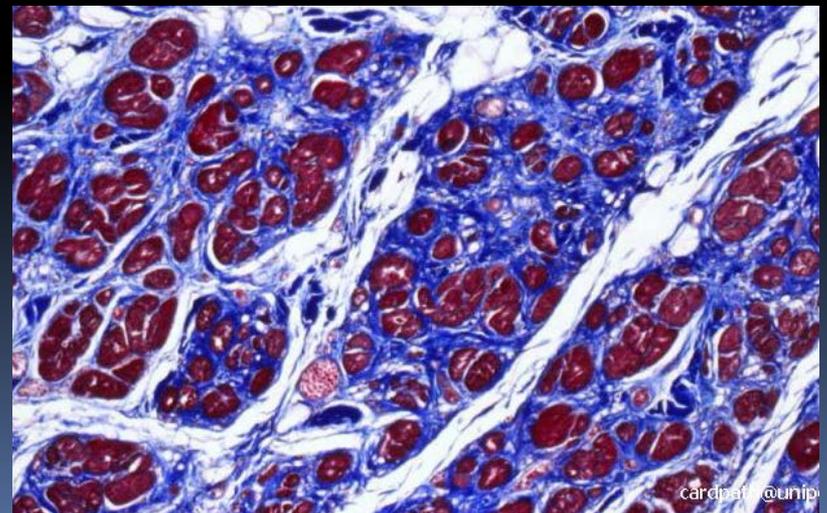
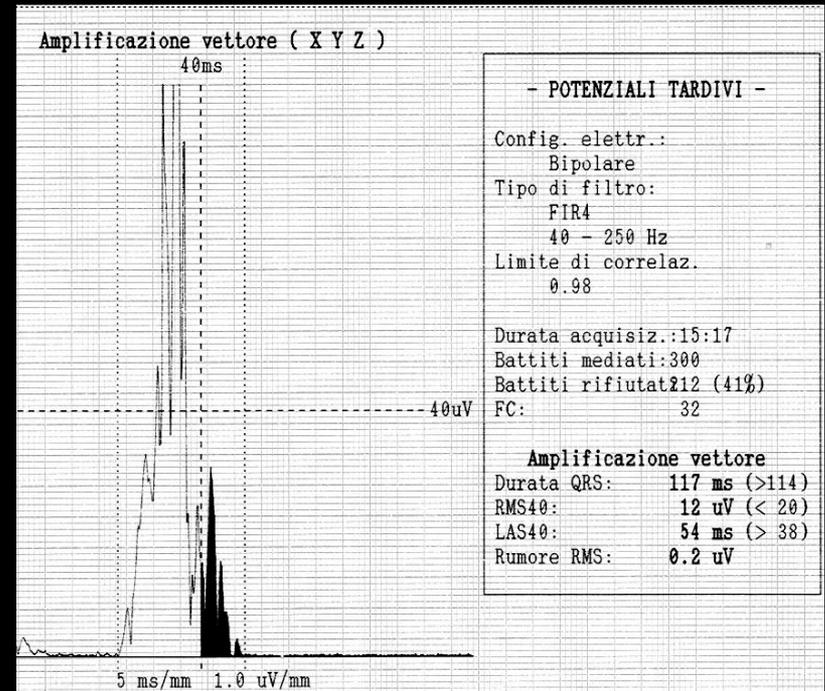
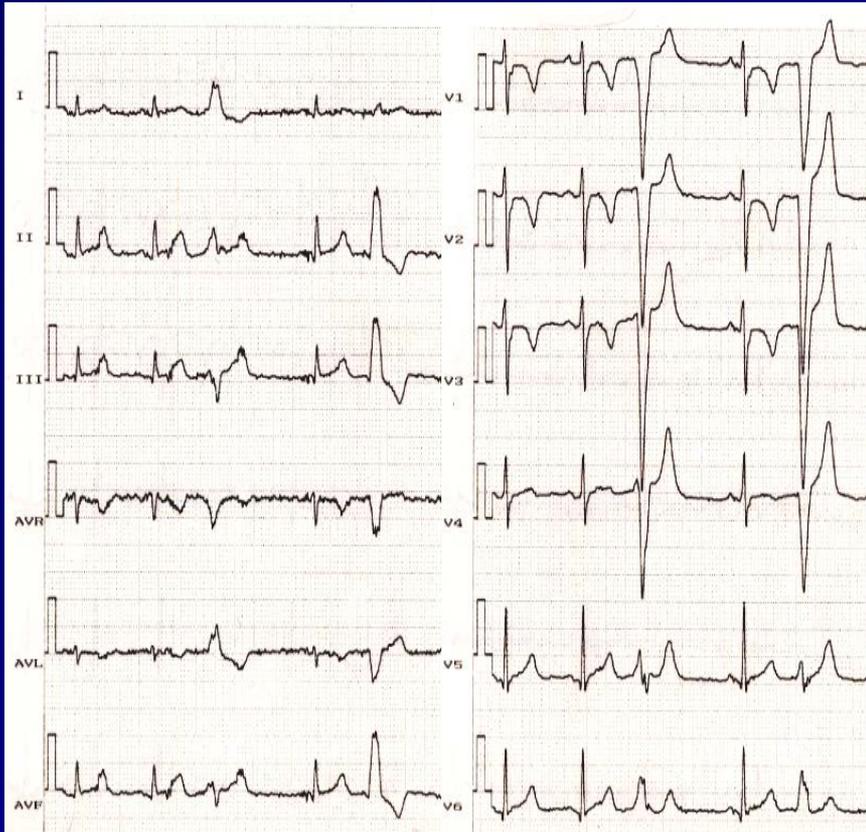
Prevalence of right precordial T-wave inversion at preparticipation ECG screening: a prospective study on 3086 young competitive athletes

- Study population: 3086 consecutive athletes
- Gender: 2138 M, 948 F
- Age: mean 15.4±9 yrs; range 7-35 yrs
- T-wave inversion beyond V₁(overall): 127 athl. (4.1%)
 - 70 (2.3%) in leads V₁ and V₂
 - 57 (1.8%) in leads V₁ to V₃ or beyond
- T-wave inversion (ath.≥14 years): 1.4%
- T-wave inversion (ath.<14 years): 9.3% } (p<0.001)
- ARVC/D diagnosis (Echo/cardiac MR): 3 of 127 (2.3%)
- ARVC/D prevalence in this population: 0.1%

Top-level canoeist, died at 24 yrs for ARVC



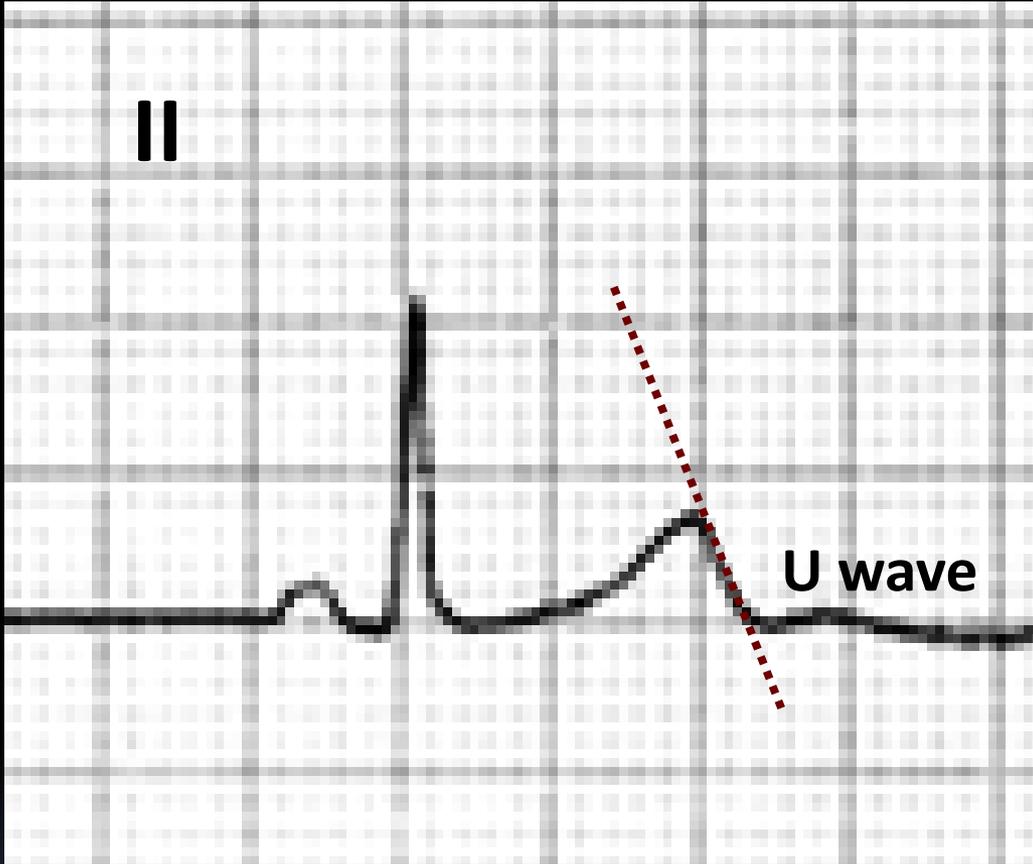
Elite rower with ARVC and complex VAs at 24-hour Holter



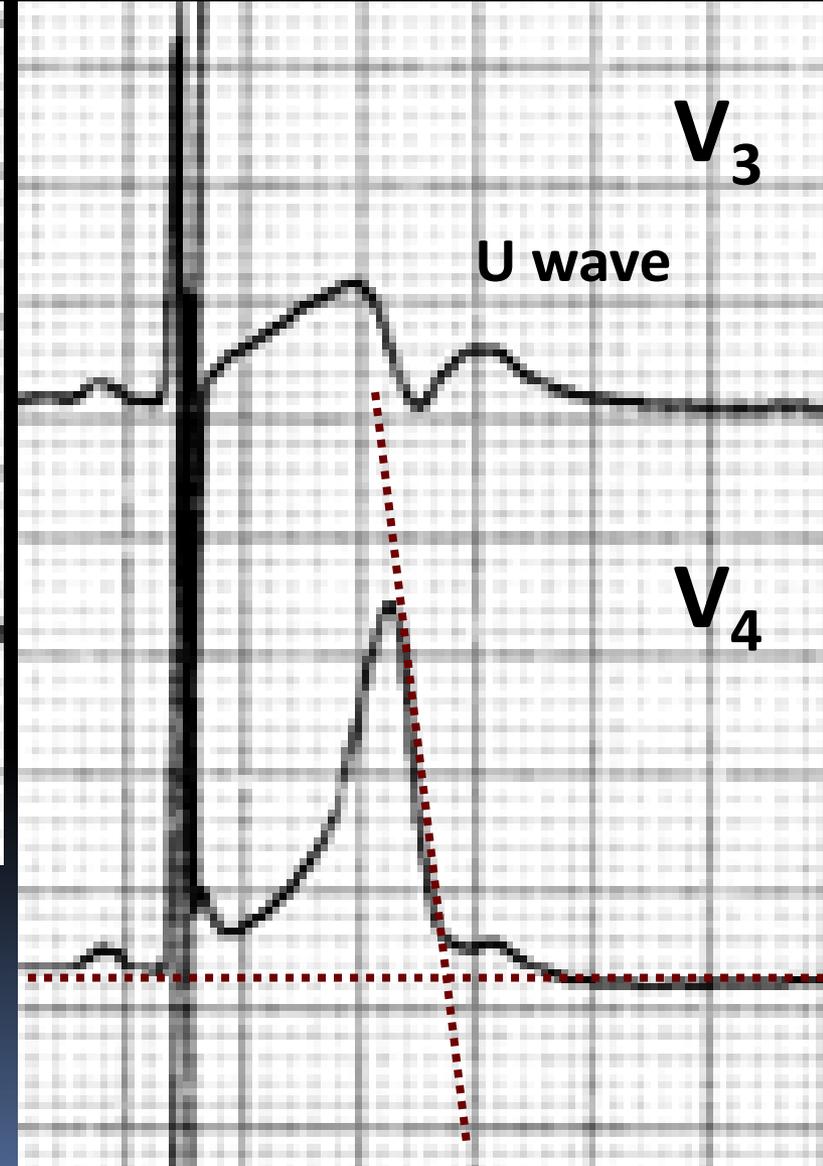
Long QT syndrome: Problems in Athletes

Difficulty to **precisely** measure QT interval in athletes, because of the frequent presence of bradycardia and U wave

35 yrs, elite marathon runner

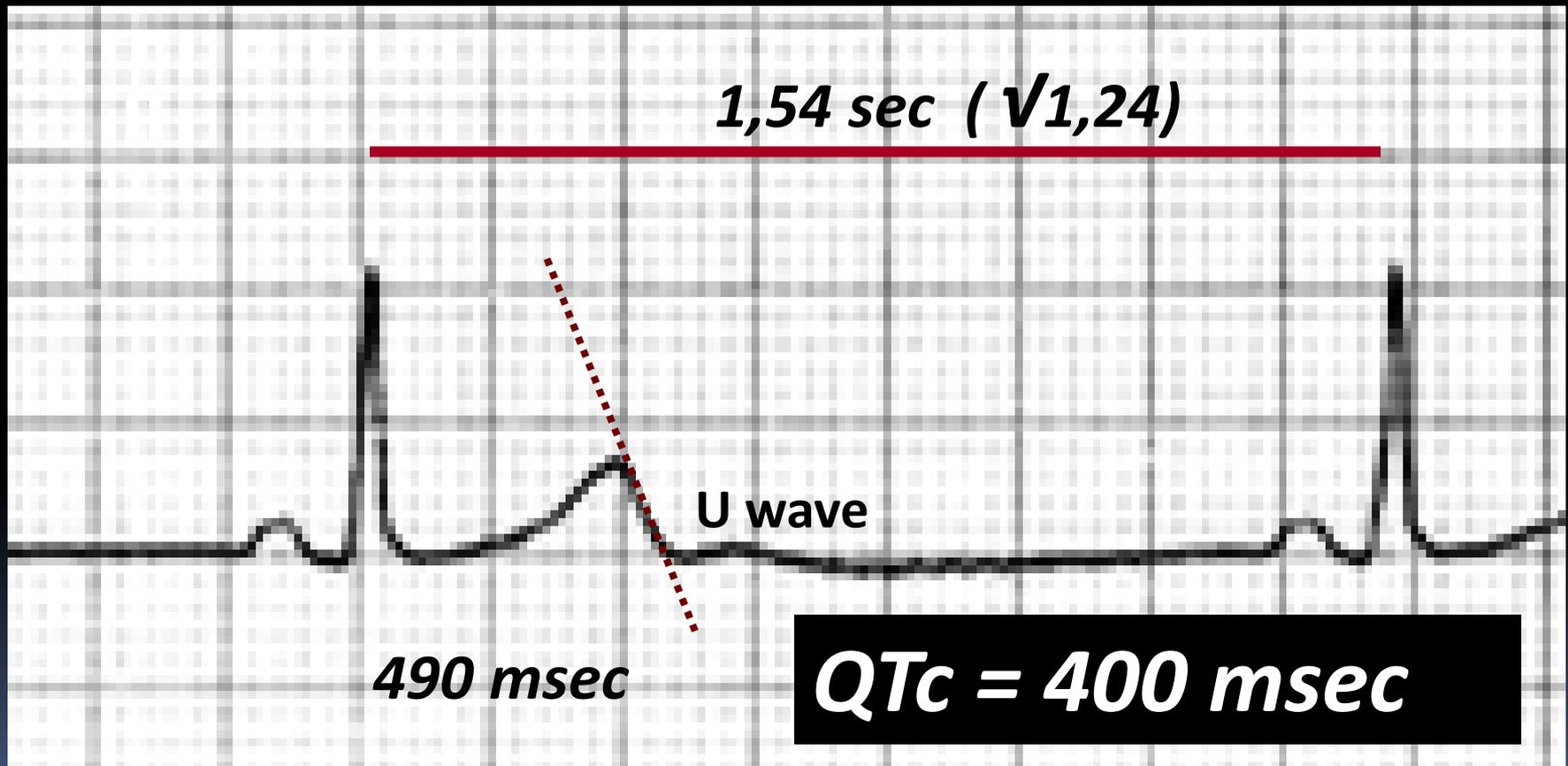


Sinus bradycardia 36 bpm



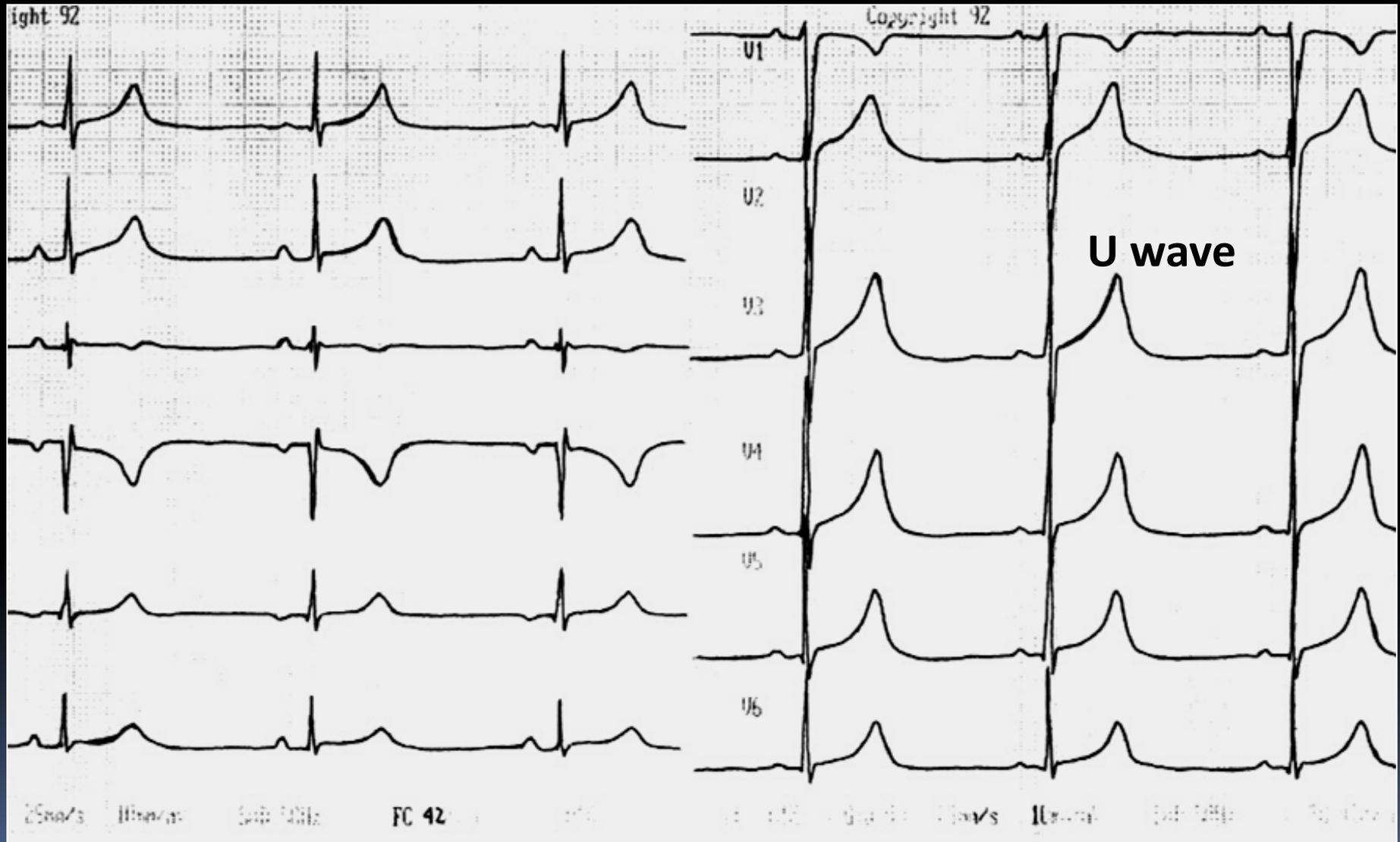
Bazett Formula

$$QTc \text{ (msec)} = QT / \sqrt{R-R} \text{ (sec)}$$



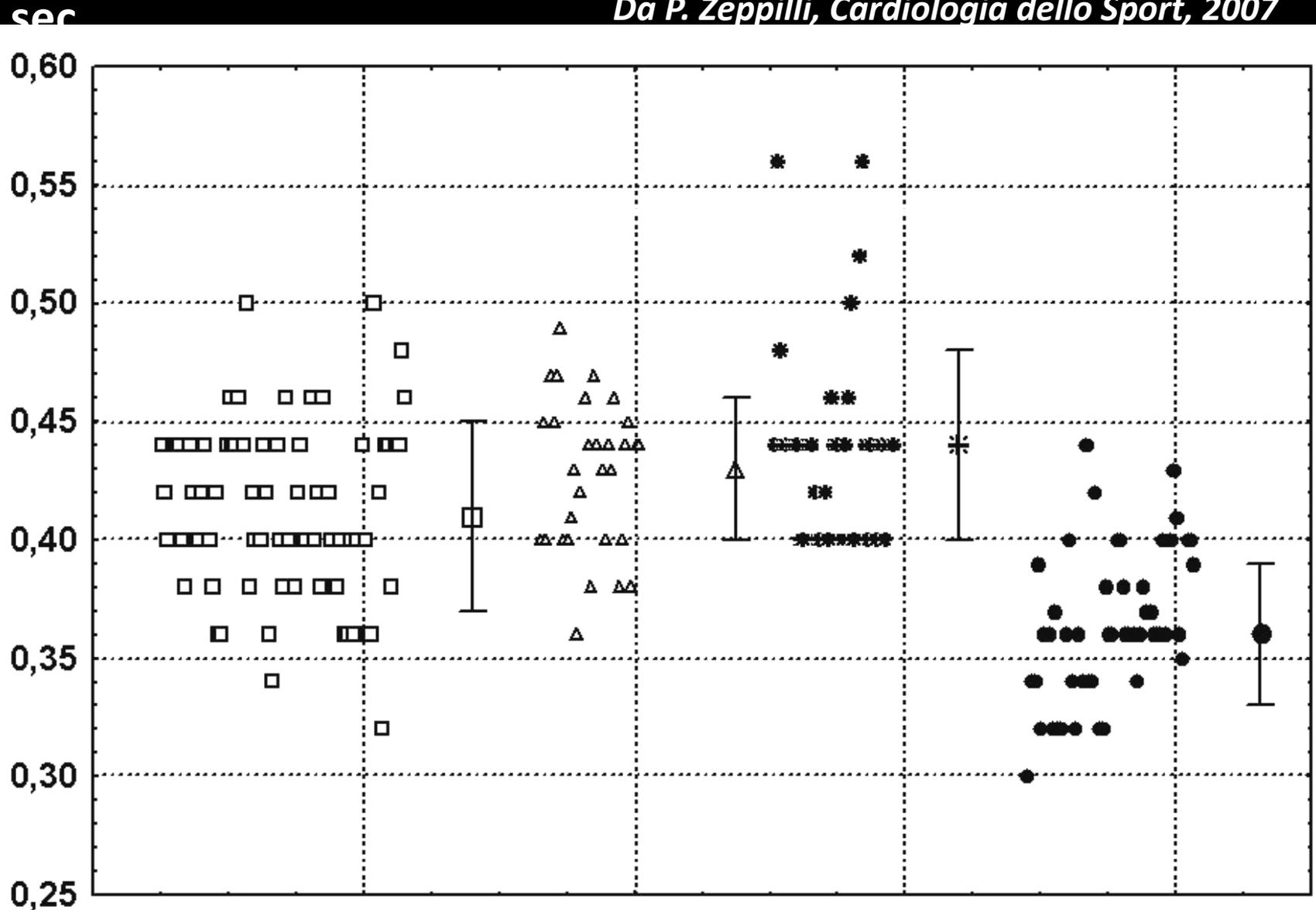
29 yrs., elite cyclist

HR 42 bpm, QT 0,53 sec, QTc 0,435



QT interval in athletes and sedentary subjects

Da P. Zeppilli, Cardiologia dello Sport, 2007



Marathon run.

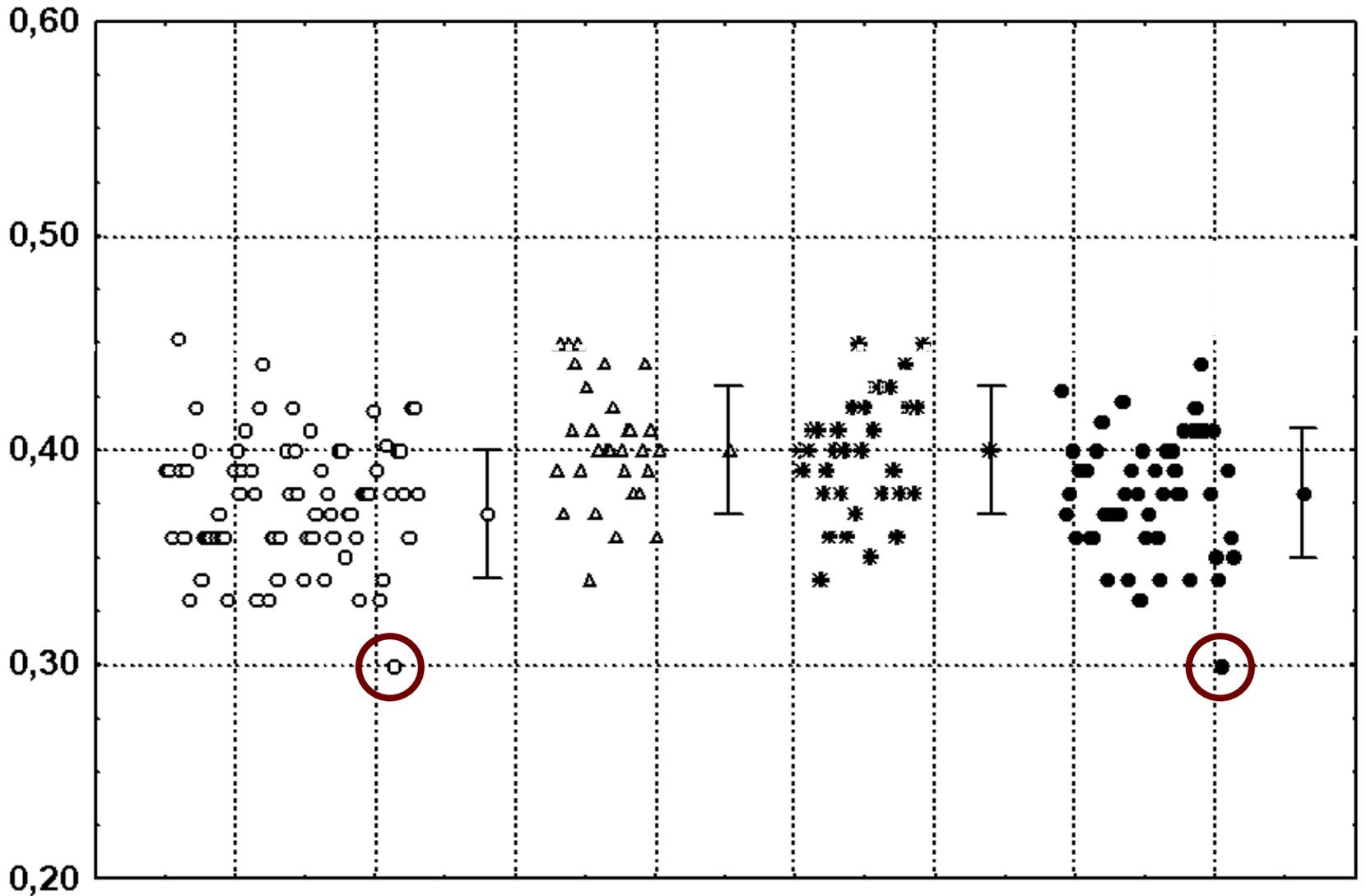
Cyclists

Soccer pl.

Sedentary

QT c in athletes and sedentary subjects

sec.



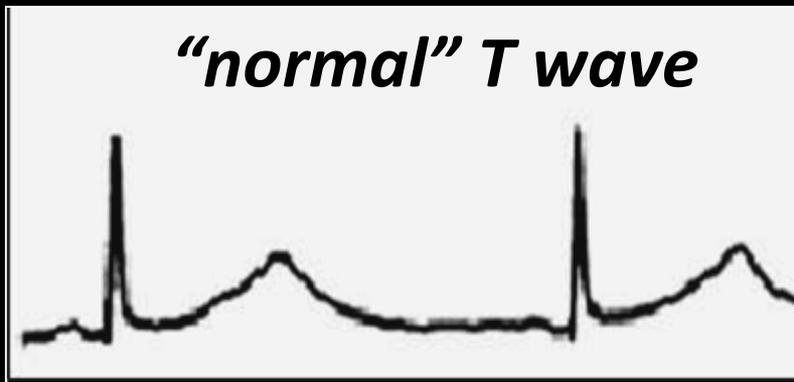
Marathon run.

Cyclists

Soccer pl.

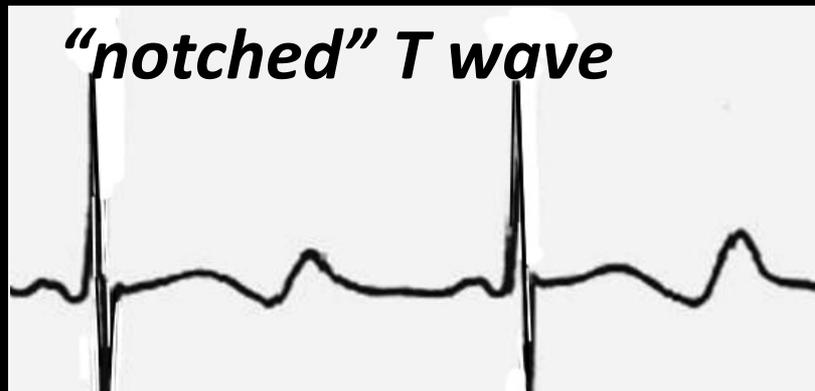
Sedentary

LQT1
(42-55%)



- *KCNQ1 gene*
- *K⁺ channel, slow current*
- *Loss of function*
- *Syncope or SD during exercise (swimming)*

LQT2
(35-45%)



- *KCNH2 gene (Herg)*
- *K⁺ channel, fast current*
- *Loss of function*
- *Syncope or SD during emotional stress (alarm-clock)*

LQT3
(8-10%)



- *SCN5A gene*
- *Na⁺ channel,*
- *Gain of function*
- *SD during sleeping*

Practical guidelines for suspected long QT in Athletes

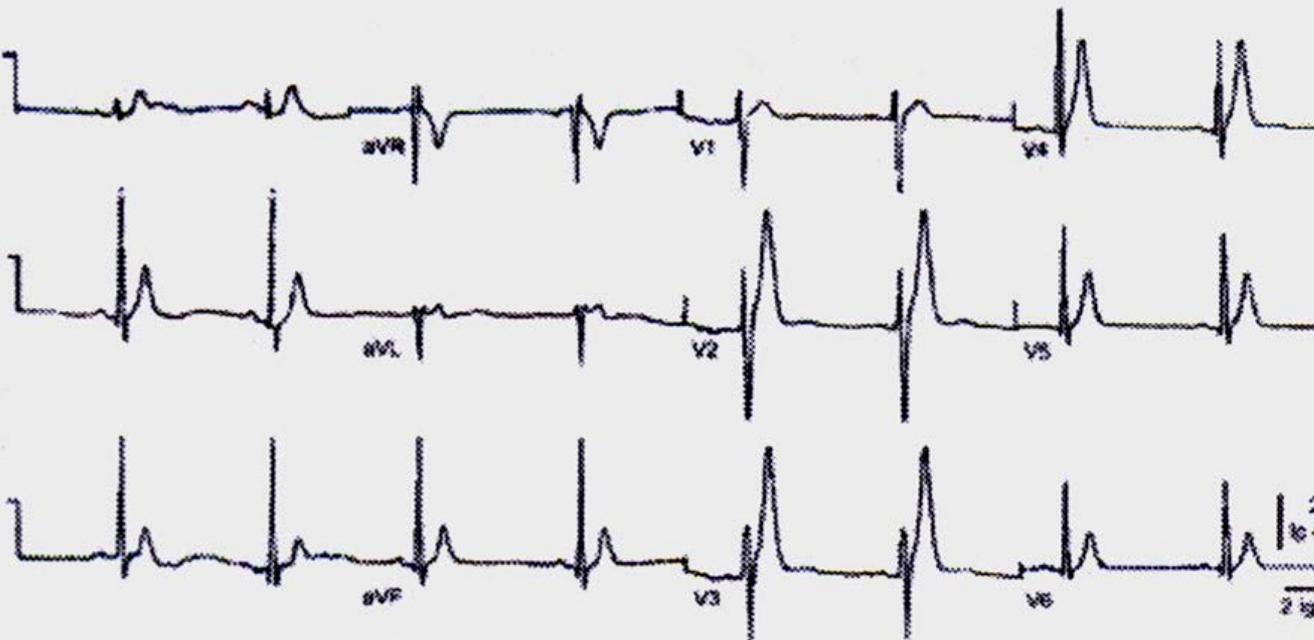
- ***To exclude acquired long QT***
(drugs, hypokalemia, salt-losing tubulopathies, such as Bartter-Gitelman syndrome etc)
- ***Family history*** *(syncope, sudden death)*
- ***Stress-test ECG, Holter (12 leads):***
paradox increase of QTc interval with exercise
- ***Re-evaluation after detraining***
verify a reduction of QTc
- ***Genetics: is it really helpful?***

Short QT Syndrome

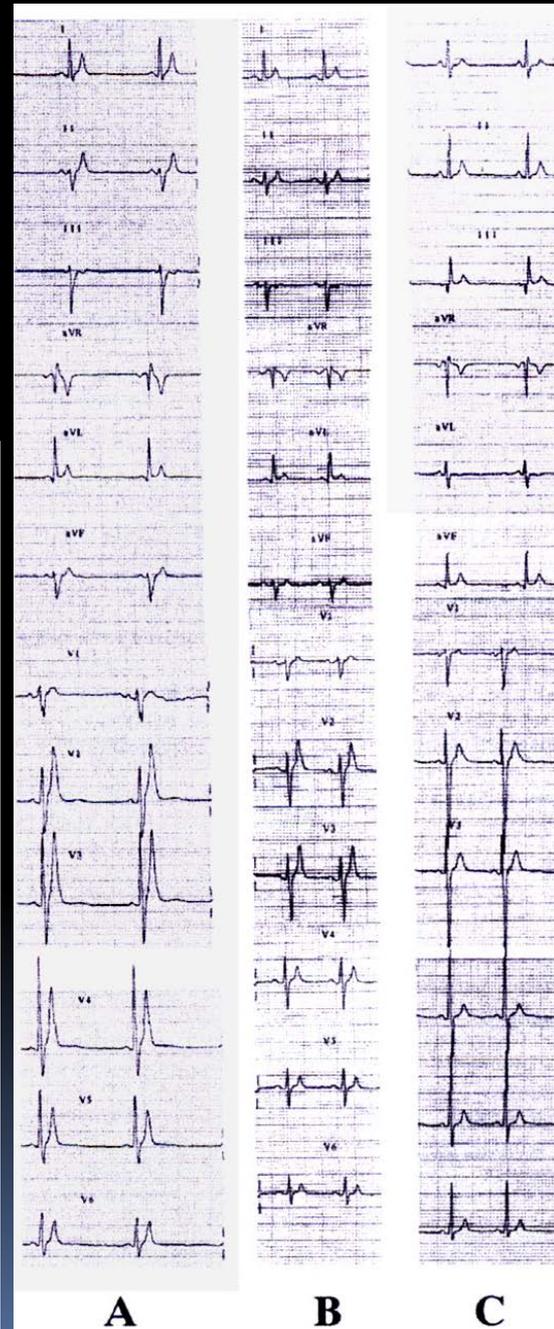
A Familial Cause of Sudden Death

Fiorenzo Gaita, MD; Carla Giustetto, MD; Francesca Bianchi, MD; Christian Wolpert, MD;
Rainer Schimpf, MD; Riccardo Riccardi, MD; Stefano Grossi, MD;
Elena Richiardi, MD; Martin Borggrefe, MD

Background—A prolonged QT interval is associated with a risk for life-threatening events. However, little is known about prognostic implications of the reverse—a short QT interval. Several members of 2 different families were referred for syncope, palpitations, and resuscitated cardiac arrest in the presence of a positive family history for sudden cardiac death. Autopsy did not reveal any structural heart disease. All patients had a constantly and uniformly short QT interval at ECG.



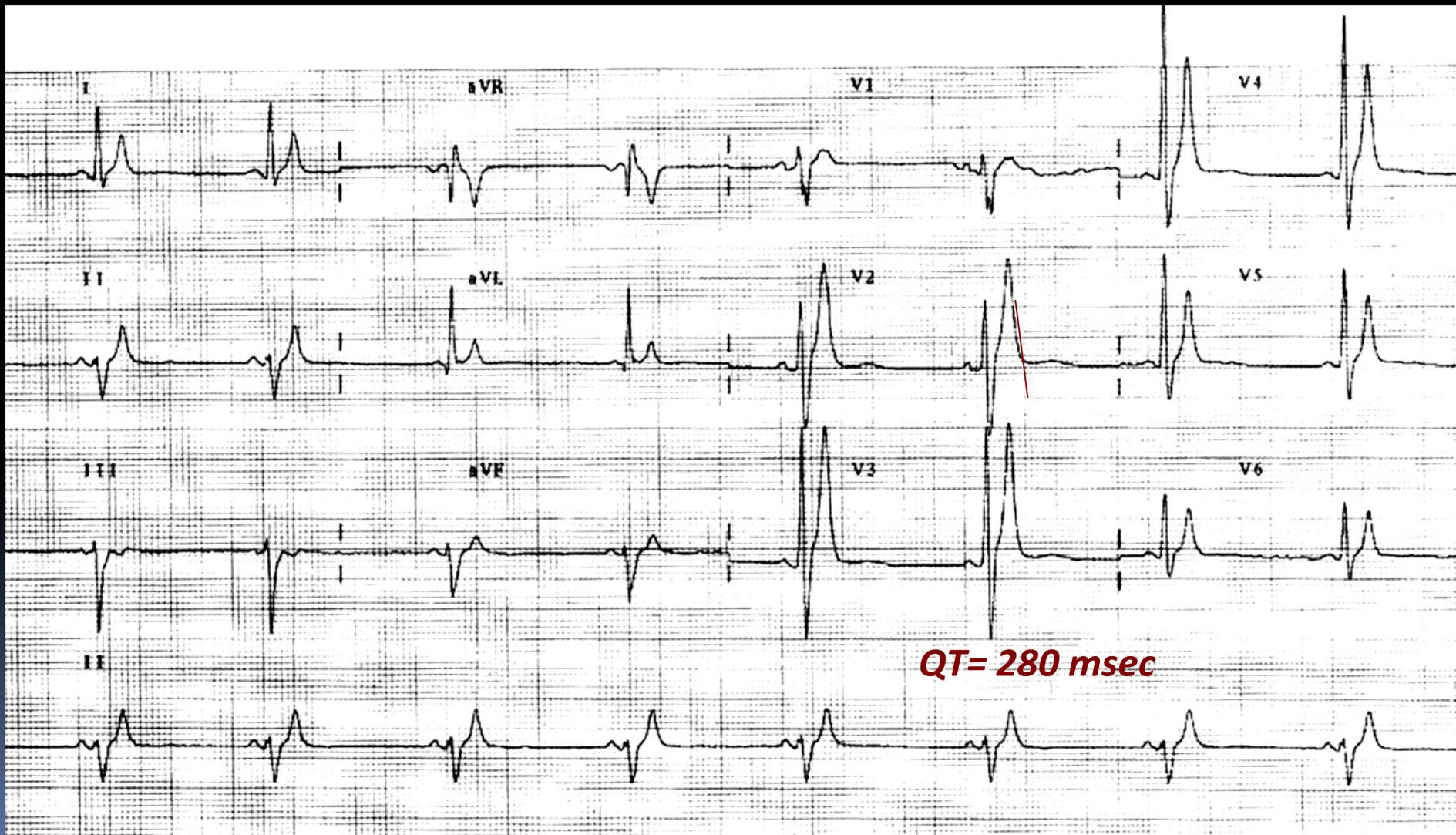
$QTc < 300$ msec

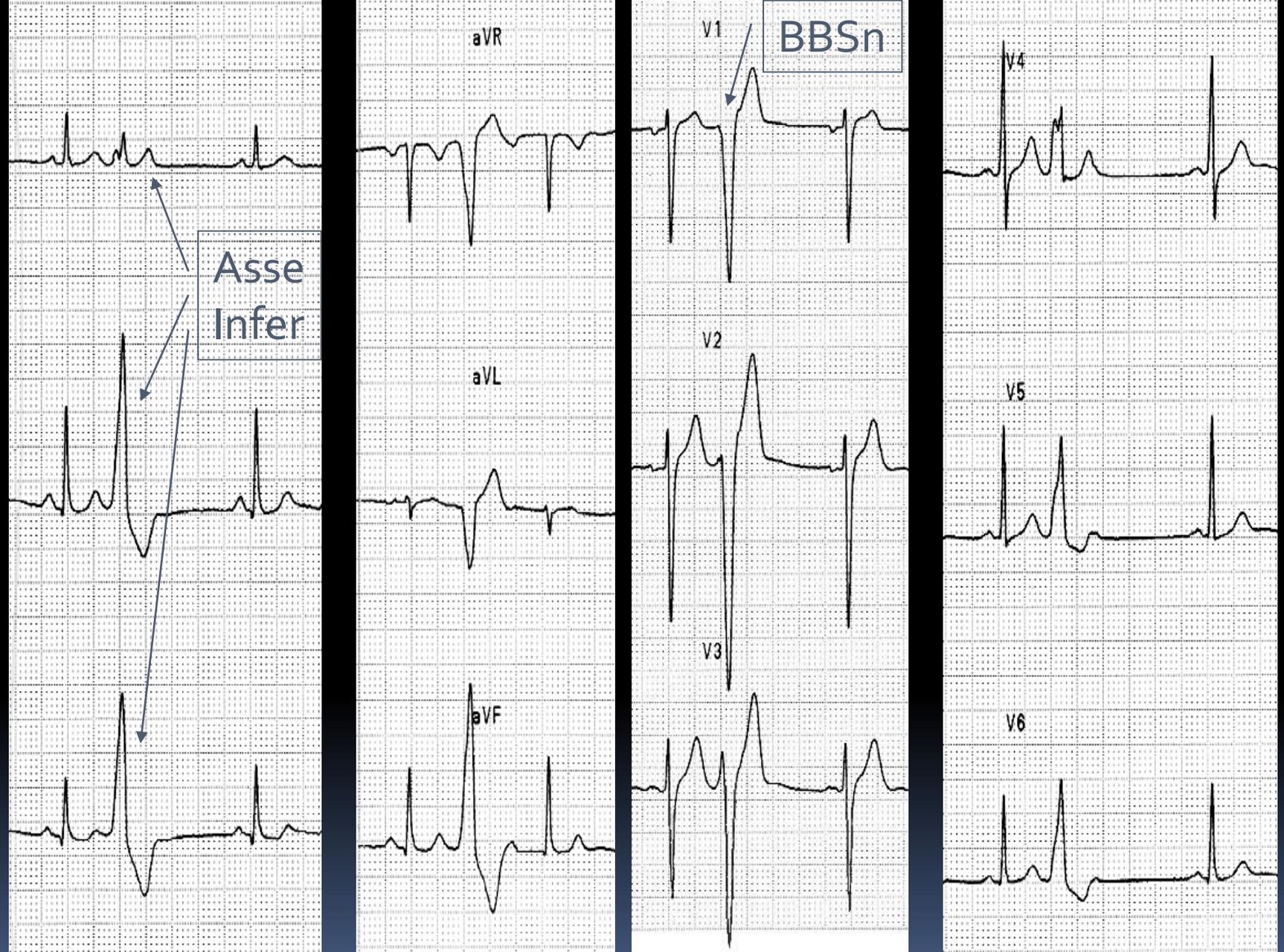


Short QT Syndrome

A Familial Cause of Sudden Death

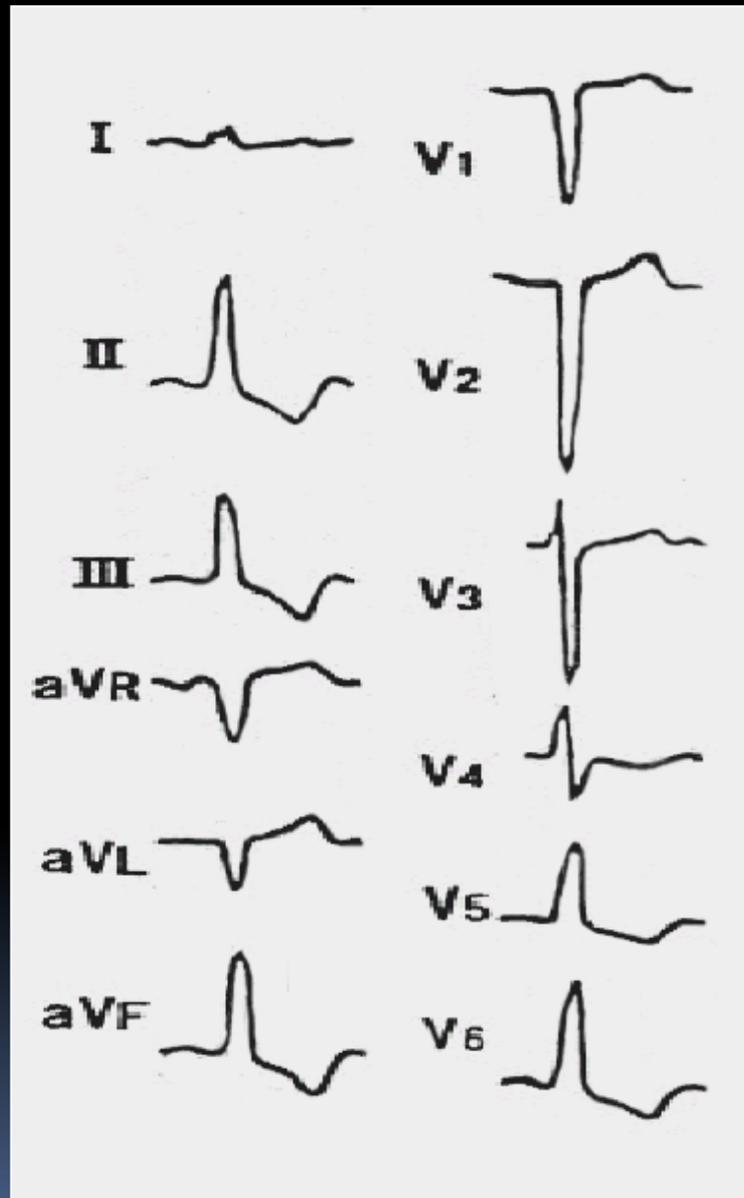
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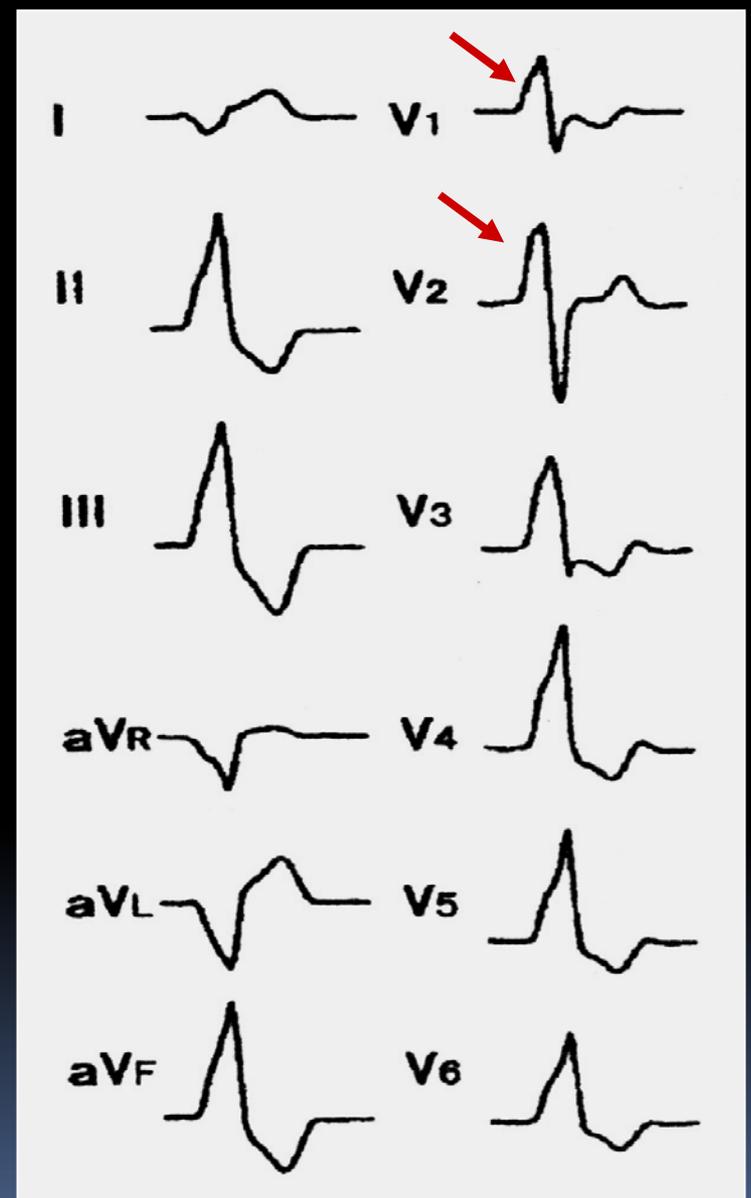


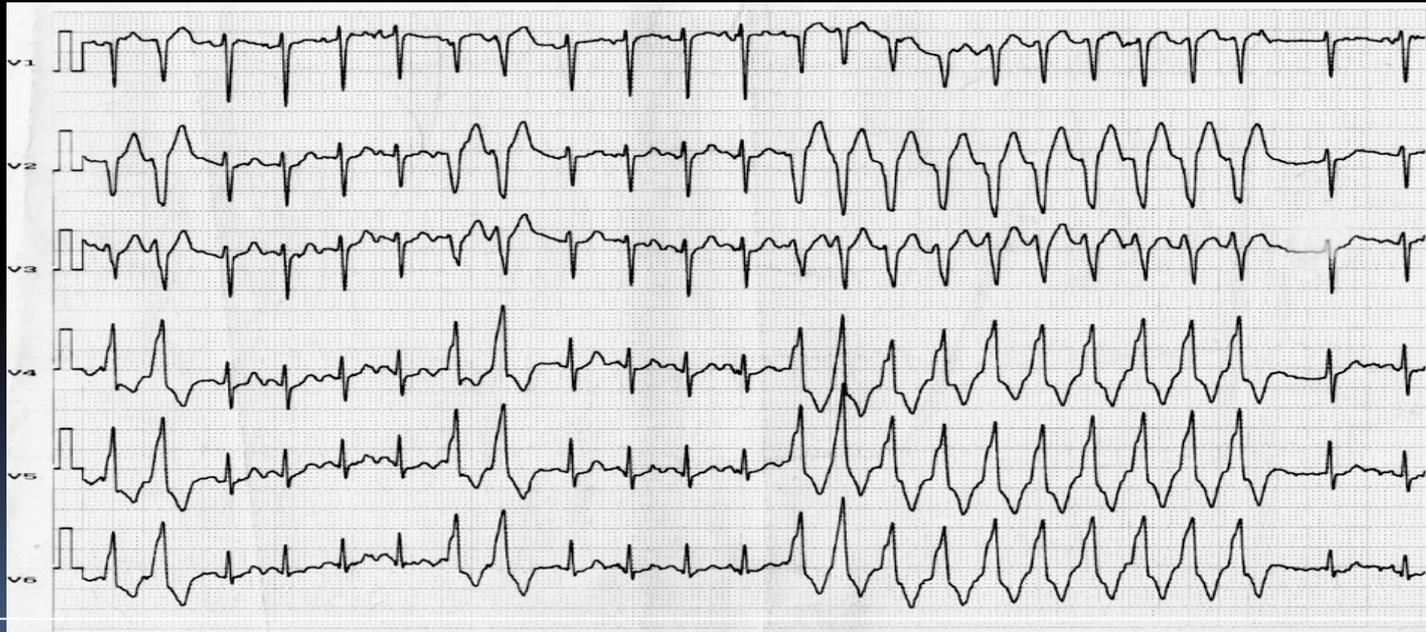
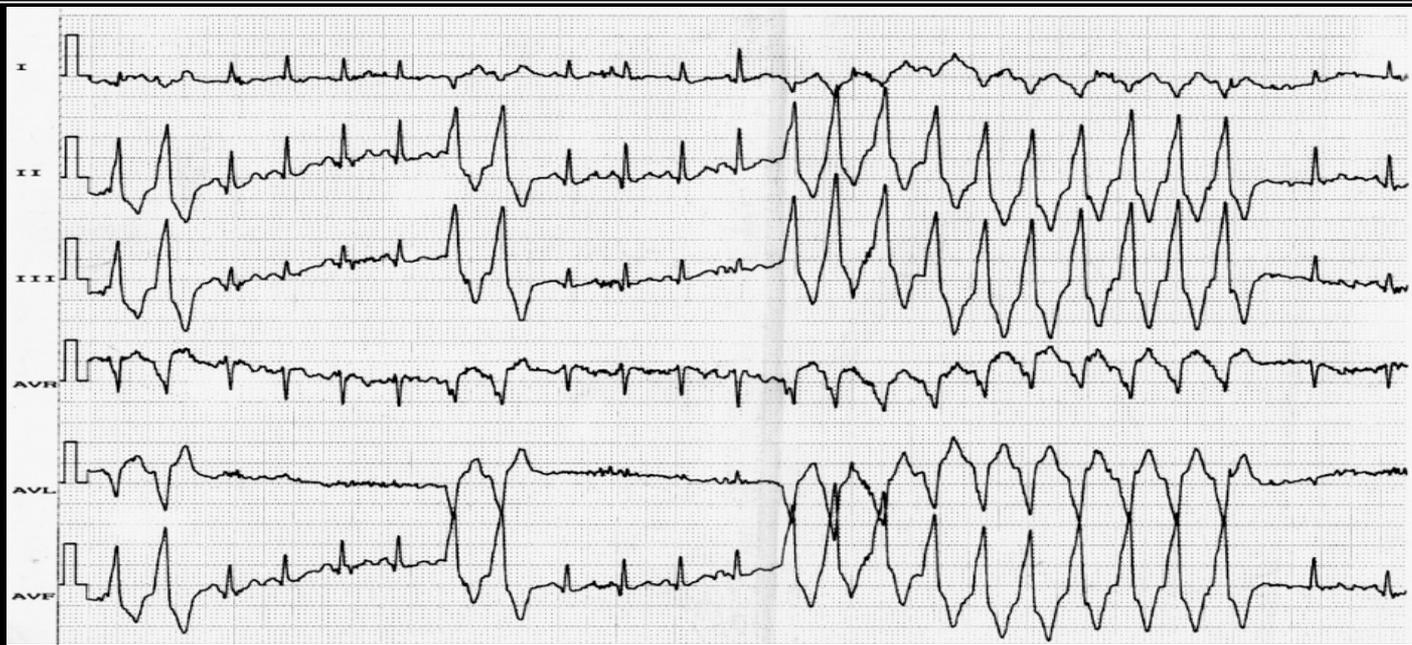
Tipica aritmia ventricolare idiopatica/benigna ad origine dal tratto d'efflusso del ventricolo destro (BBSn + Asse Inferiore)

BPV da efflusso destro



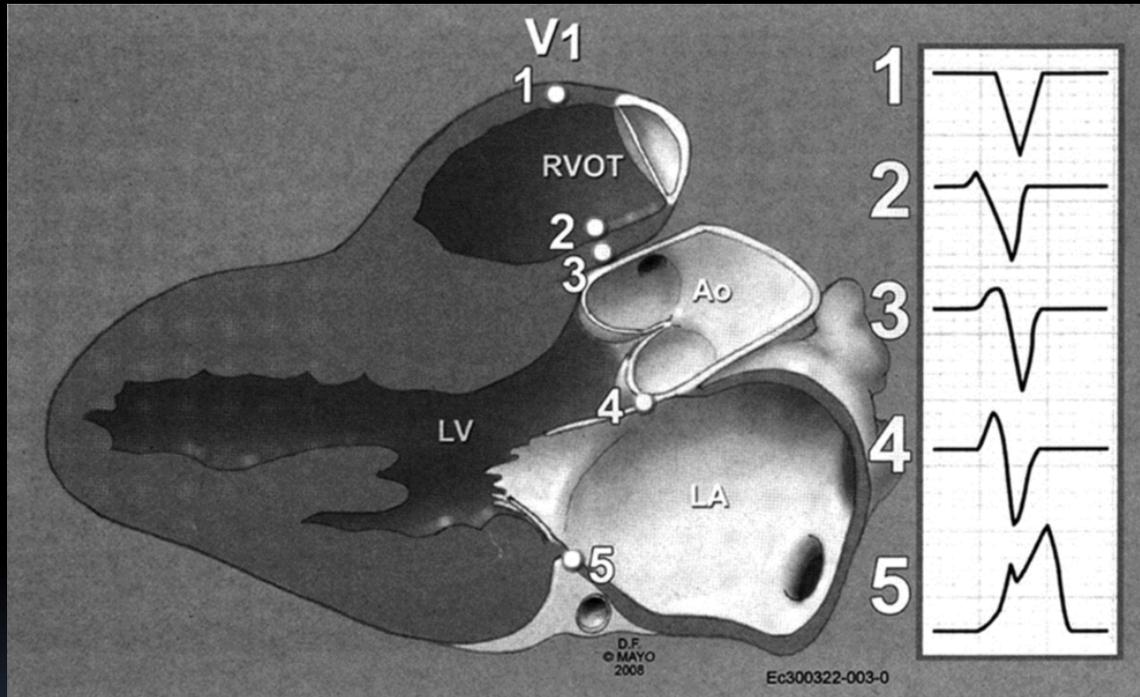
BPV da efflusso sinistro





From: Ventricular Arrhythmias in the Absence of Structural Heart Disease

J Am Coll Cardiol. 2012;59(20):1733-1744. doi:10.1016/j.jacc.2012.01.036



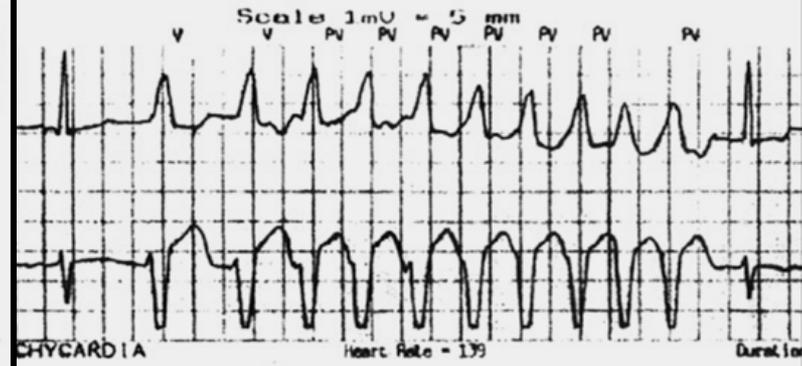
The anatomy of the outflow tract region is such that areas on the right and left sides of the heart can be in close proximity to each other. This can give similar ECG patterns in several leads. However, note that in V₁, there is a gradual increase in the amplitude of the r-wave as the site of origin of the ventricular ectopy moves leftward.

Ventricular Tachyarrhythmias in Athletes

Long-Term Clinical Significance of Frequent and Complex Ventricular Tachyarrhythmias in Trained Athletes

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Antonio Spataro, MD,* Stefano Caselli, MD,* Massimo Santini, MD,† Barry J. Maron, MD, FACC‡
Rome, Italy, and Minneapolis, Minnesota

- OBJECTIVES** The aim of this study was to clarify the clinical relevance of ventricular tachyarrhythmias assessed by 24-h ambulatory electrocardiograms (ECG) in a large, unique, and prospectively evaluated athletic population.
- BACKGROUND** For athletes with ventricular tachyarrhythmias, the risk of sudden cardiac death associated with participation in competitive sports is unresolved.
- METHODS** We assessed 355 competitive athletes with ventricular arrhythmias (VAs) on a 24-h ambulatory (Holter) ECG that was obtained because of either palpitations, the presence of ≥ 3 premature ventricular depolarizations (PVDs) on resting 12-lead ECG, or both.
- RESULTS** Athletes were segregated into three groups: Group A with $\geq 2,000$ PVDs/24 h ($n = 71$); Group B with ≥ 100 < 2,000 PVDs/24 h ($n = 153$); and Group C with only < 100 PVDs/24 h ($n = 131$). Cardiac abnormalities were detected in 26 of the 355 study subjects (7%) and were significantly more common in Group A (21/71, 30%) than in Group B (5/153, 3%) or Group C athletes (0/131, 0% $p < 0.001$). Only the 71 athletes in Group A were excluded from competition. During follow-up (mean, 8 years), 70 of 71 athletes in Group A and each of the 284 athletes in Groups B and C have survived without cardiovascular events. The remaining Group A athlete died suddenly of arrhythmogenic right ventricular cardiomyopathy while participating in a field hockey game against medical advice.
- CONCLUSIONS** Frequent and complex ventricular tachyarrhythmias are common in trained athletes and are usually unassociated with underlying cardiovascular abnormalities. Such VAs (when unassociated with cardiovascular abnormalities) do not convey adverse clinical significance, appear to be an expression of "athlete's heart syndrome," and probably do not per se justify a disqualification from competitive sports. (J Am Coll Cardiol 2002;40:446-52) © 2002 by the American College of Cardiology Foundation



Biffi et al.

"... ventricular tachyarrhythmias are common in trained athletes and are usually unassociated with underlying cardiovascular abnormalities ... do not convey adverse clinical significance, appear to be an expression of athlete's heart syndrome".

CONCLUSIONS REMARKS

ECG screening is a lifesaving strategy which meets the criteria for a good screening program:

- still asymptomatic athletes with at-risk cardiovascular diseases are accurately identified by 12-lead ECG;
- an effective management strategy exists based on restriction of life-threatening training/competition and subsequent clinical treatment
- early identification and management of asymptomatic athletes favourably modify the outcome of the underlying diseases leading to substantial reduction of SCD

THANK YOU

