How to read the ECG in athletes:
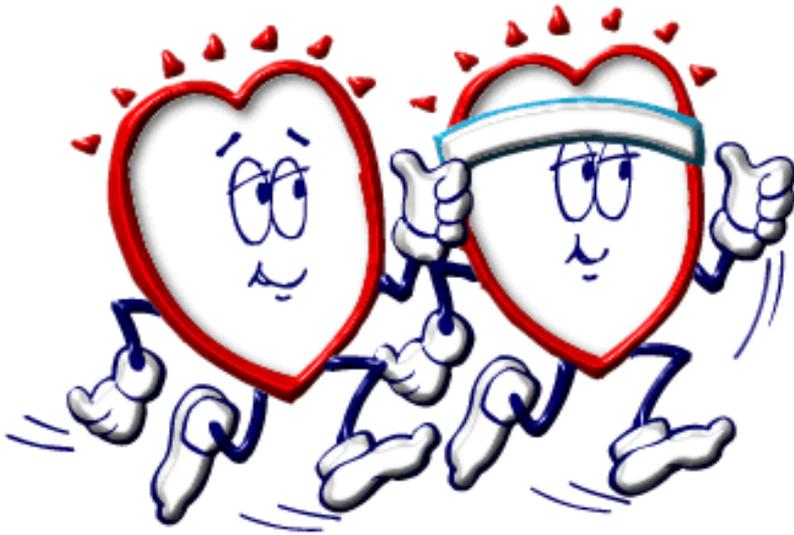
distinguishing normal form abnormal

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Cardiac adaptations to Rowing

Vagotonia
Sinus bradycardia
Sinus arrhythmia
First and second degree AVB
Early repolarization

Increased chamber size
Left ventricular enlargement and hypertrophy
Right ventricular enlargement
Left and right atrial enlargement
Right atrial enlargement

Recommendations for interpretation of 12-lead electroca...

Domenico Corr...
Mark Link[5], Cris...
Ihor Gussac[8], A...
François Carrè[1...
Jan Hoogsteen[1...
Luc Vanhees[3], J...
Barry J. Maron[2...
Gaetano Thiene...
Sections of Spo...
Prevention and ...
Disease of the E...

Original articles

Electrocardiographic interpretation in athletes: the 'Seattle Criteria'

Jonathan A Drezner,[1] Michael John Ackerman,[2] Jeffrey Anderson,[3] Euan Ashley,[4] Chad A Asplund,[5] Aaron L Baggish,[6] Mats Börjesson,[7] Bryan C Cannon,[8] Domenico Corrado,[9] John P DiFiori,[10] Peter Fischbach,[11] Victor Froelicher,[4] Kimberly G Harmon,[1] Hein Heidbuchel,[12] Joseph Marek,[13] David S Owens,[14] Stephen Paul,[15] Antonio Pelliccia,[16] Jordan M Prutkin,[14] Jack C Salerno,[17] Christian M Schmied,[18] Sanjay Sharma,[19] Ricardo Stein,[20] Victoria L Vetter,[21] Mathew G Wilson[22]

This document was developed in collaboration between the American Medical Society for Sports Medicine (AMSSM), the Section on Sports Cardiology of the European Association for Cardiovascular Prevention and Rehabilitation (EACPR), a registered branch of the European Society of Cardiology (ESC), the FIFA Medical Assessment and Research Center (F-MARC), and the Pediatric & Congenital Electrophysiology Society (PACES).

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                    ECG abnormalities
                      in the athlete

        (Group 1)                      (Group 2)
    common (up to 80%)              Uncommon (< 5%)

  -Sinus bradycardia              - T-wave inversion
  -First degree AV block          - ST-segment depression
  -Notched QRS in V1 or           - Pathological Q waves
    incomplete RBBB               - Left atrial enlargement
  -Early repolarization           - Left axis deviation/left anterior
  -Isolated QRS voltage criteria    hemiblock
    for left ventricular hypertrophy - Right axis deviation/left posterior
                                      hemiblock
                                  - Right ventricular hypertrophy
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                                  - Long or short QT interval
                                  - Brugada-like early repolarization
                                  - Ventricular arrhythmias
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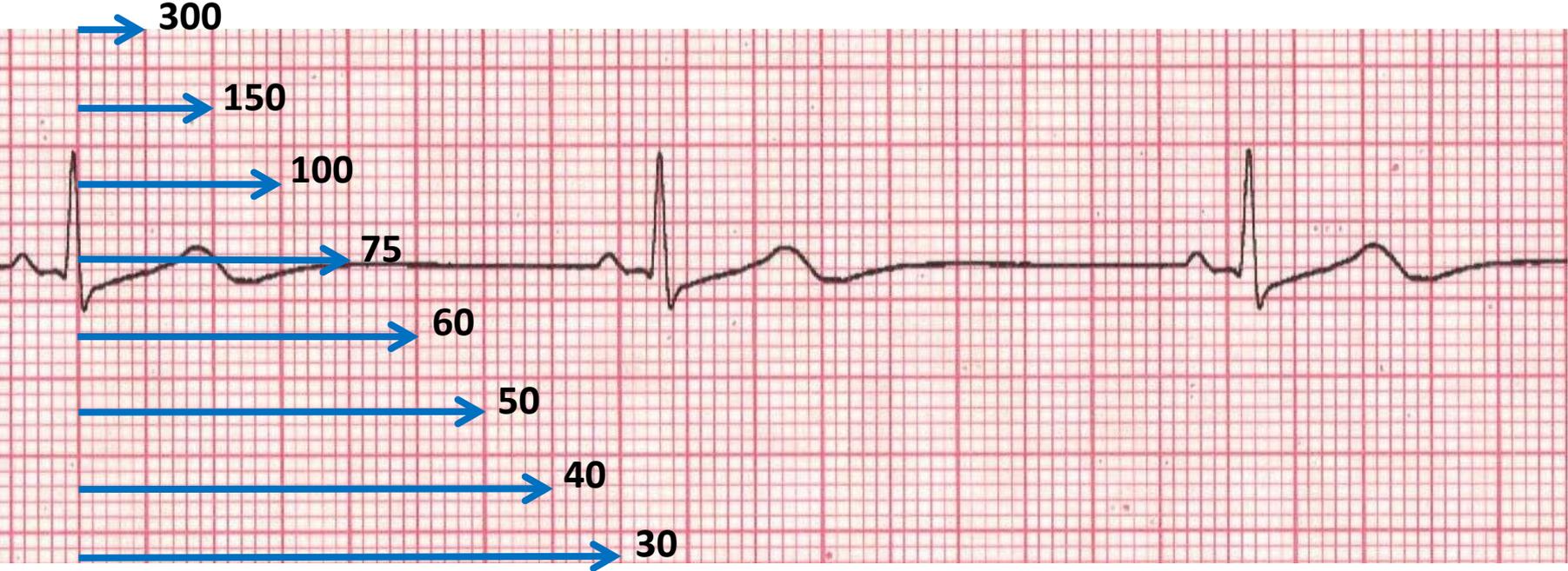
Sinus Bradycardia
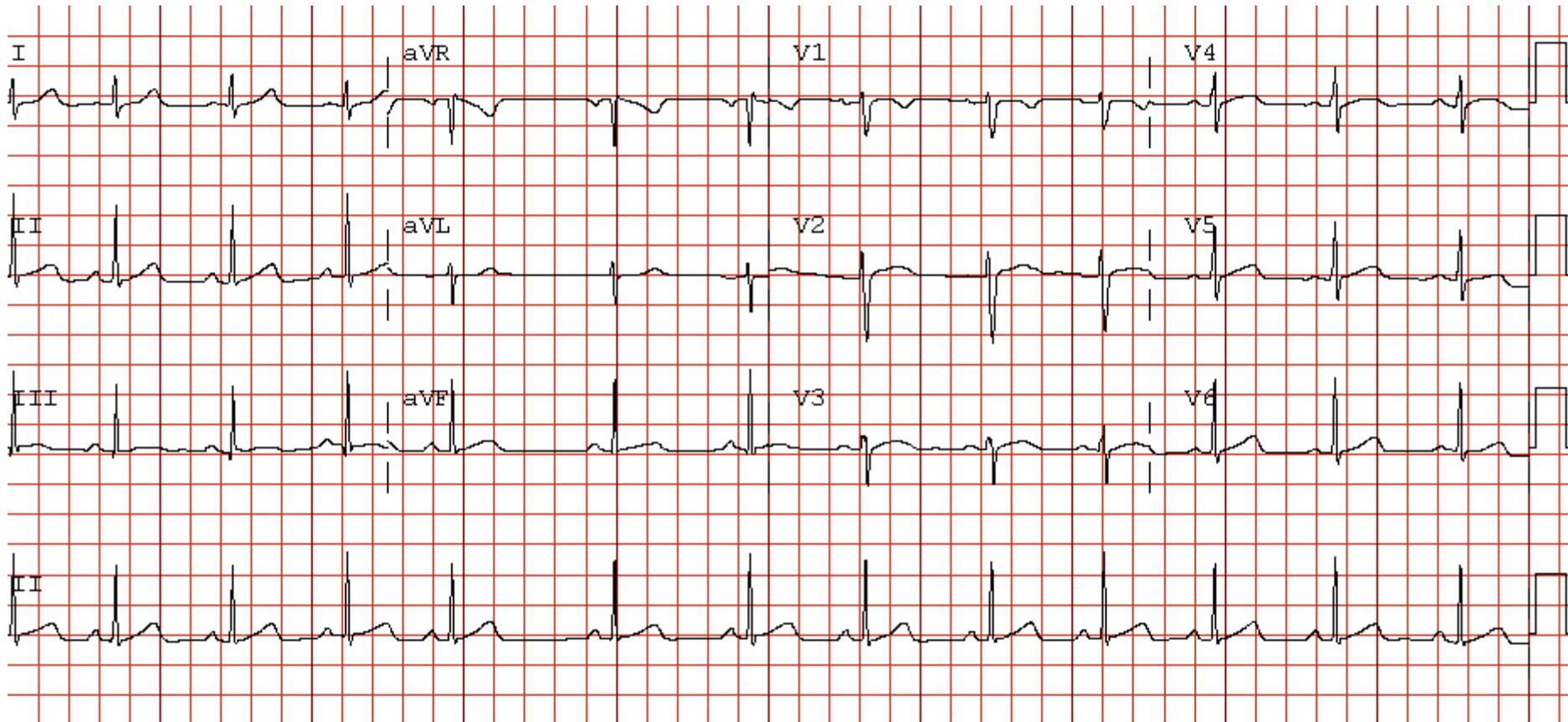# &
Sinus Arrhythmia
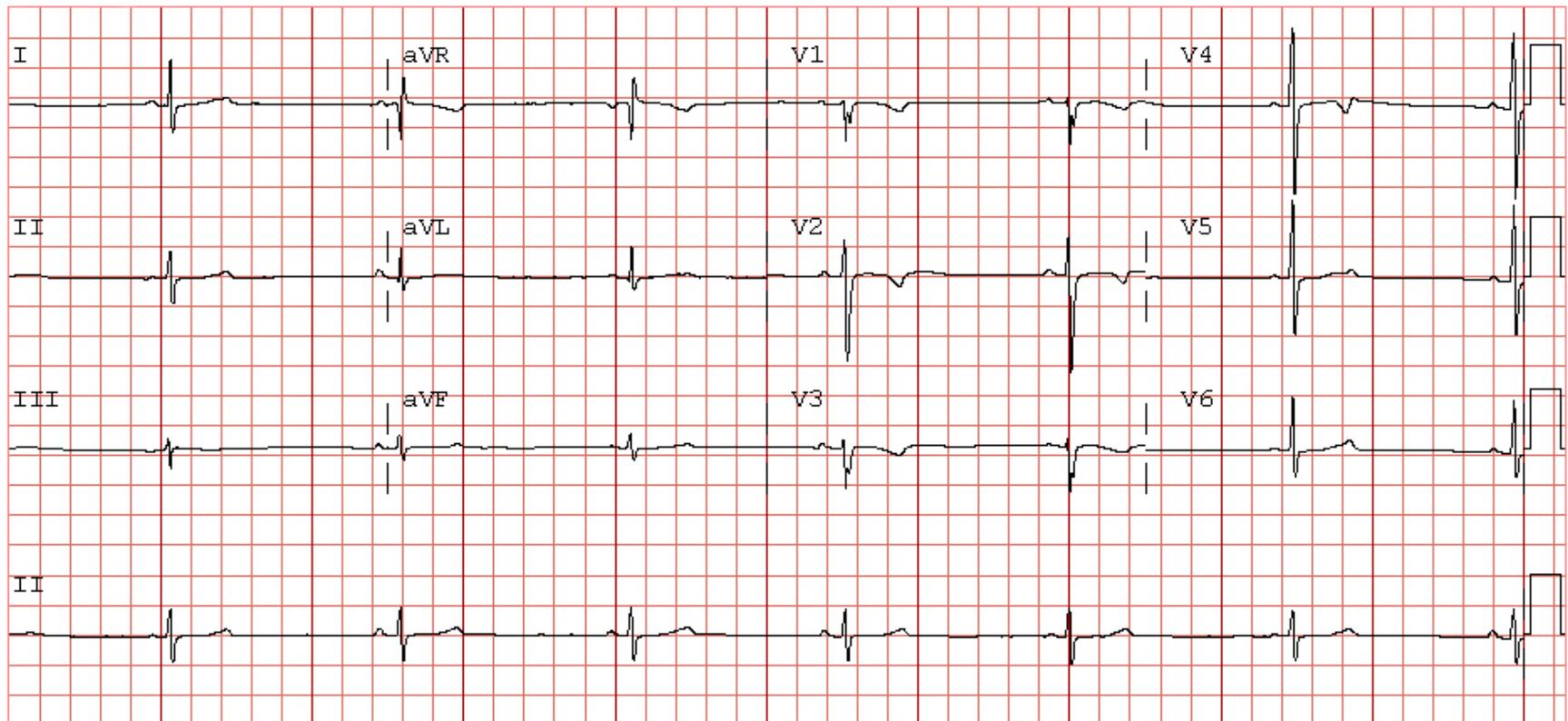
Sinus Bradycardia

ECG demonstrates sinus bradycardia with a heart rate of 40 bpm and P-wave axis of 17 degrees (arrows). The 3 required features of sinus bradycardia include: 1) P-wave before every QRS complex, 2) QRS after every P-wave , and 3) normal P-wave axis (frontal plane 0-90 degrees).

Simple method to calculate heart rate

Sinus Arrhythmia

ECG demonstrates sinus arrhythmia. Note the irregular heart rate that varies with respiration. The P-waves are upright in leads I and aVF (frontal plane) suggesting a sinus origin.
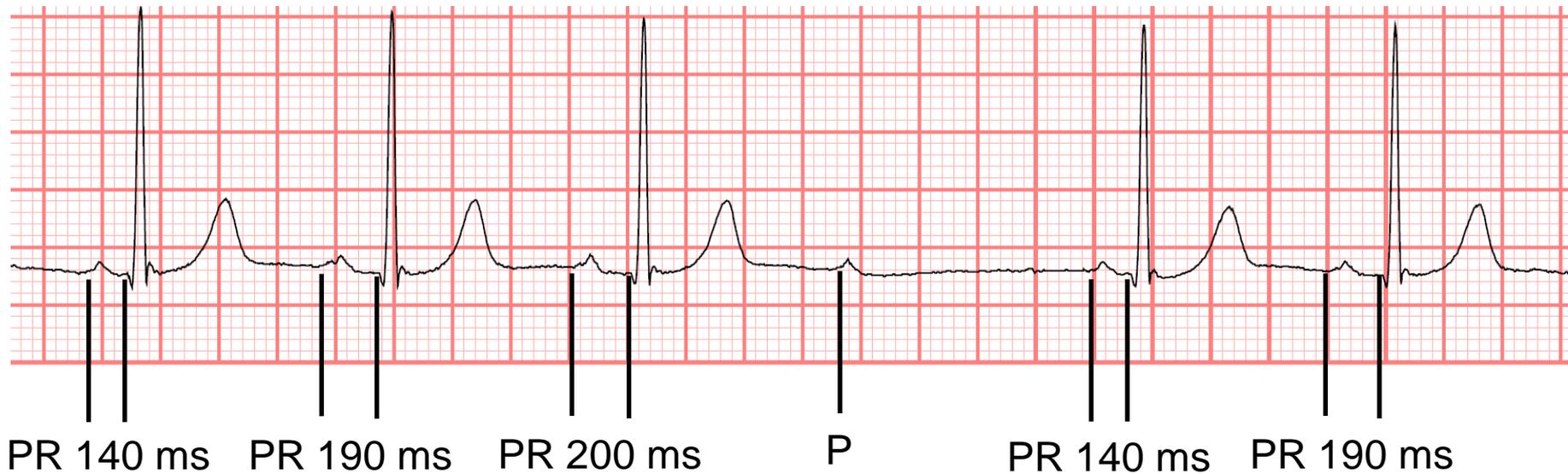
1st Degree AV Block
&
2nd Degree AV Block, MobitzType I

1° AV block (PR interval > 200 ms) /2

The PR interval is measured from the beginning of the P-wave to the beginning of the R-wave. In this ECG tracing, the PR interval is constant from beat to beat and measures 300 ms.

Mobitz Type I (Wenckebach) 2° AVB /2

PR 140 ms   PR 190 ms   PR 200 ms   P   PR 140 ms   PR 190 ms

2nd degree AV block, Mobitz Type I (Wenckebach) is demonstrated by progressively longer PR intervals until a dropped QRS. The PR interval on the first three beats are 140 ms, 190 ms, and 200 ms, respectively. The fourth P wave has no QRS complex following it. The PR interval after the fifth P wave is 140 ms again.
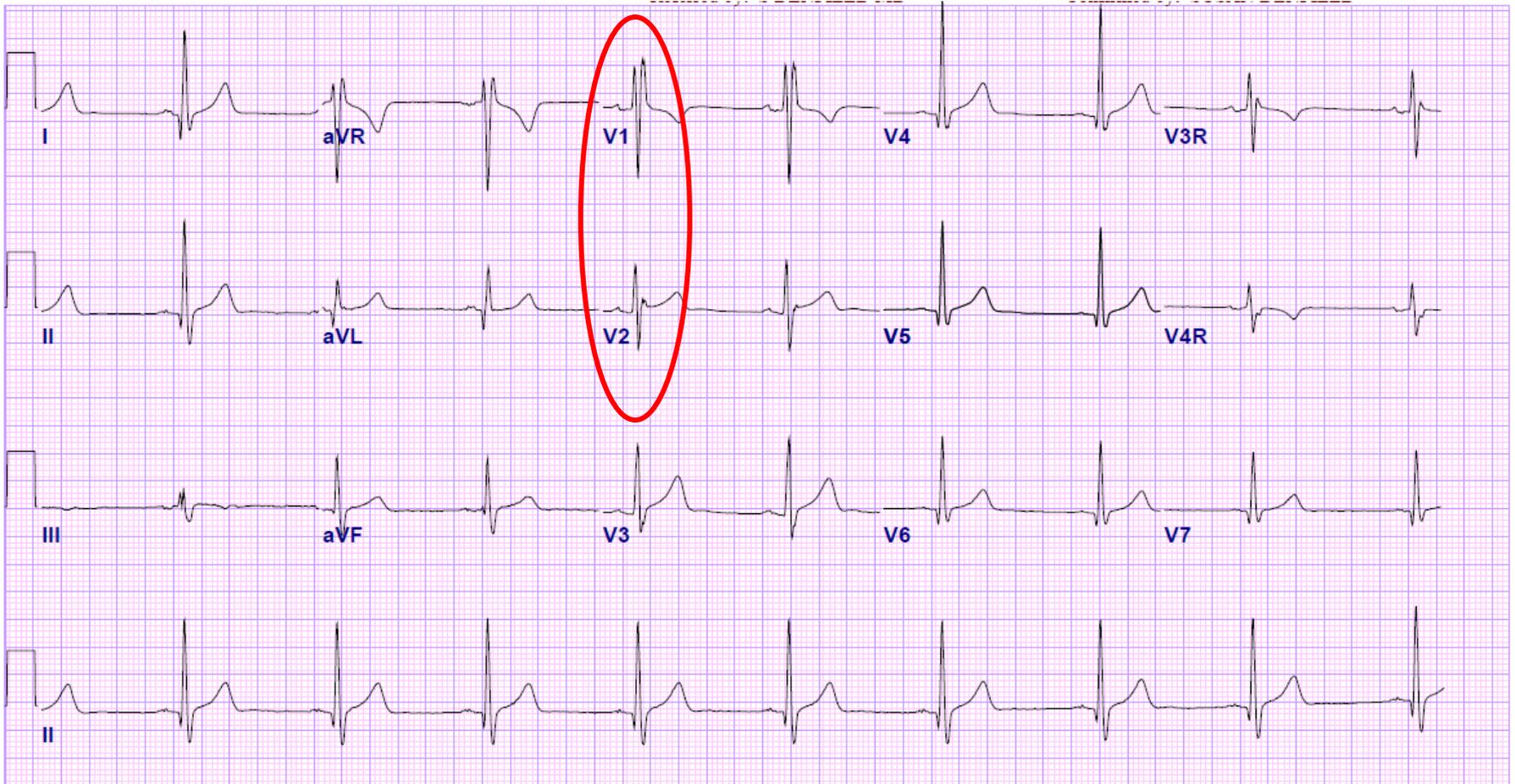
Mobitz Type I 2nd Degree AV Block
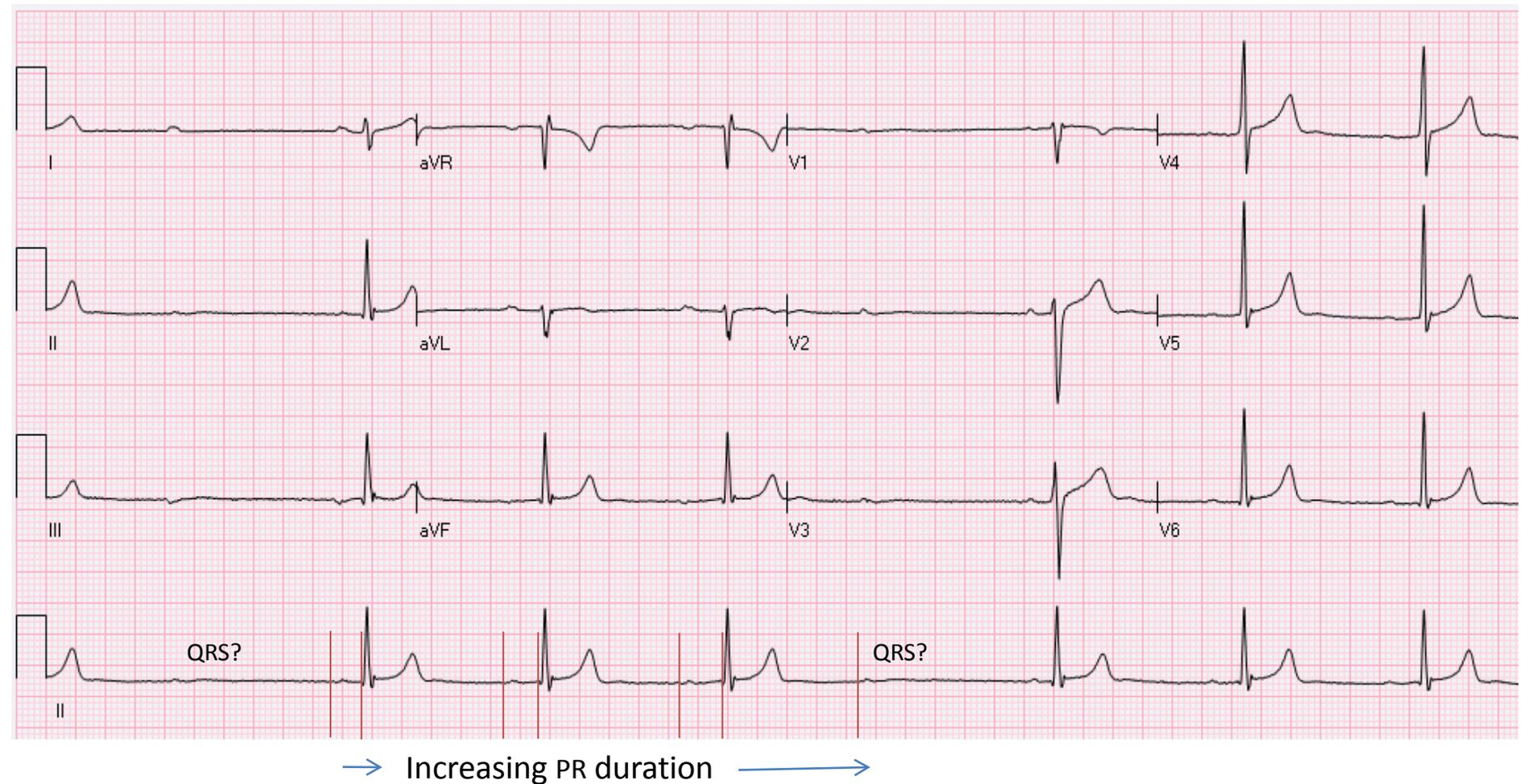
A 19-year old asymptomatic rower demonstrating sinus bradycardia with 2nd degree AV Block, Mobitz type I.
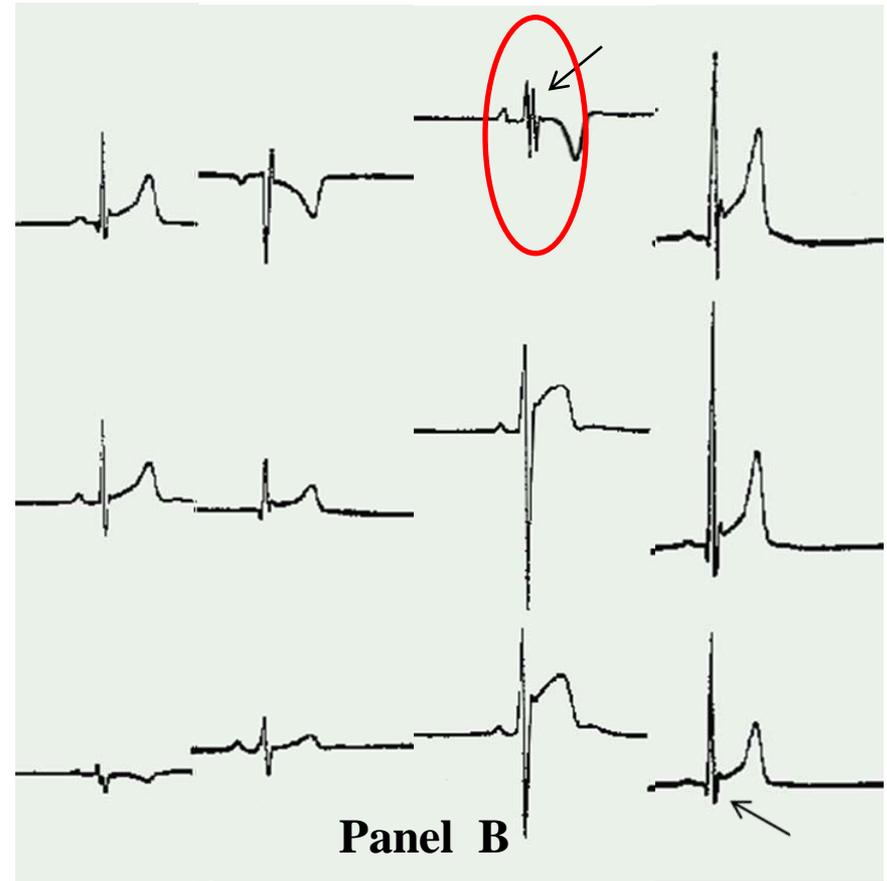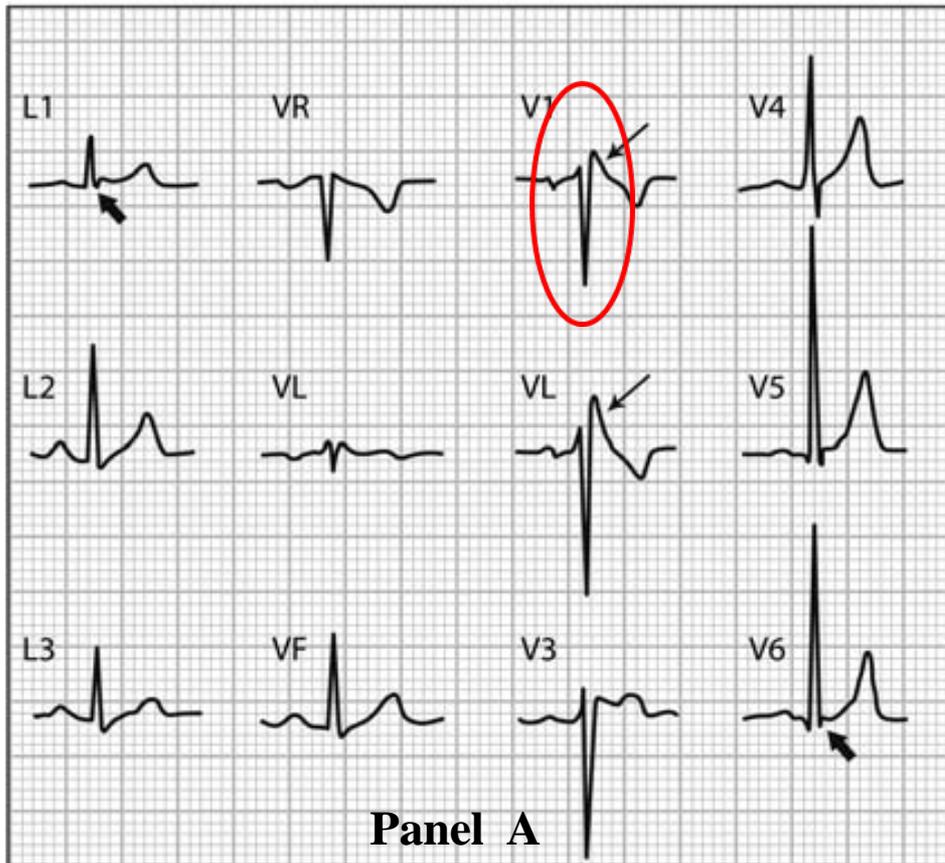
Incomplete Right Bundle Branch Block

Incomplete Right Bundle Branch Block

ECG demonstrates incomplete right bundle branch block (IRBBB) with rSR' pattern in V1 and QRS duration of < 120 ms. IRBBB is a common and normal finding in athletes and does not require additional evaluation.

# Incomplete RBBB versus Brugada-ECG Pattern

Panel A. Brugada-ECG pattern mimicking IRBBB. The "J-wave" (arrows) of Brugada-ECG is confined to right precordial leads (V1 and V2) without reciprocal "S-wave" (of comparable voltage and duration) in leads I and V6 (arrowheads).

Panel B. IRBBB in a trained athlete. The RV conduction interval is mildly prolonged (QRS duration = 115 ms) with a typical rSR' pattern in V1 (arrow). Note also the reciprocal "S-wave" in V6 (arrow).
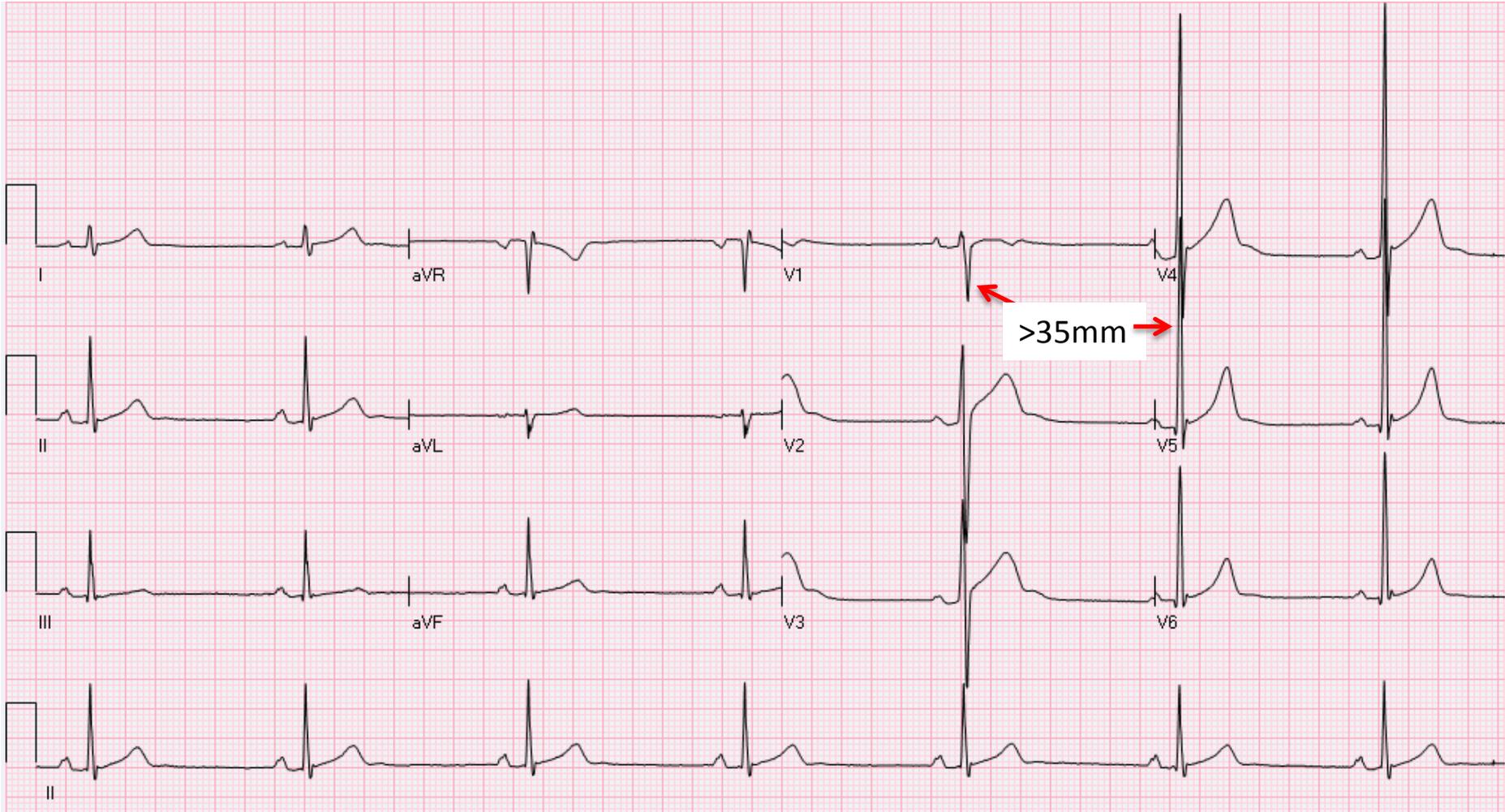
Increased R/S-wave Voltages (Criteria for Left Ventricular Hypertrophy)

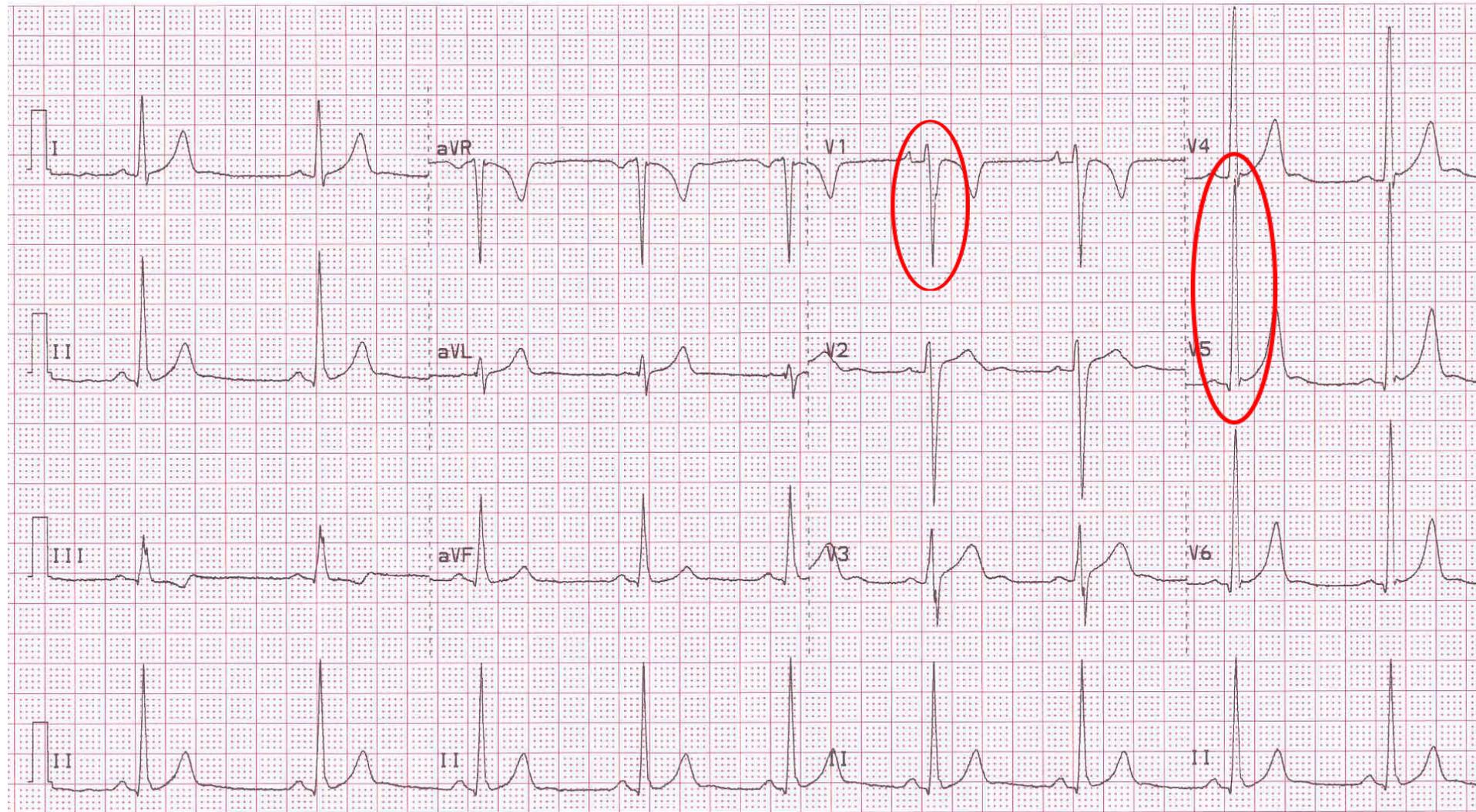# Isolated Increased QRS Voltage
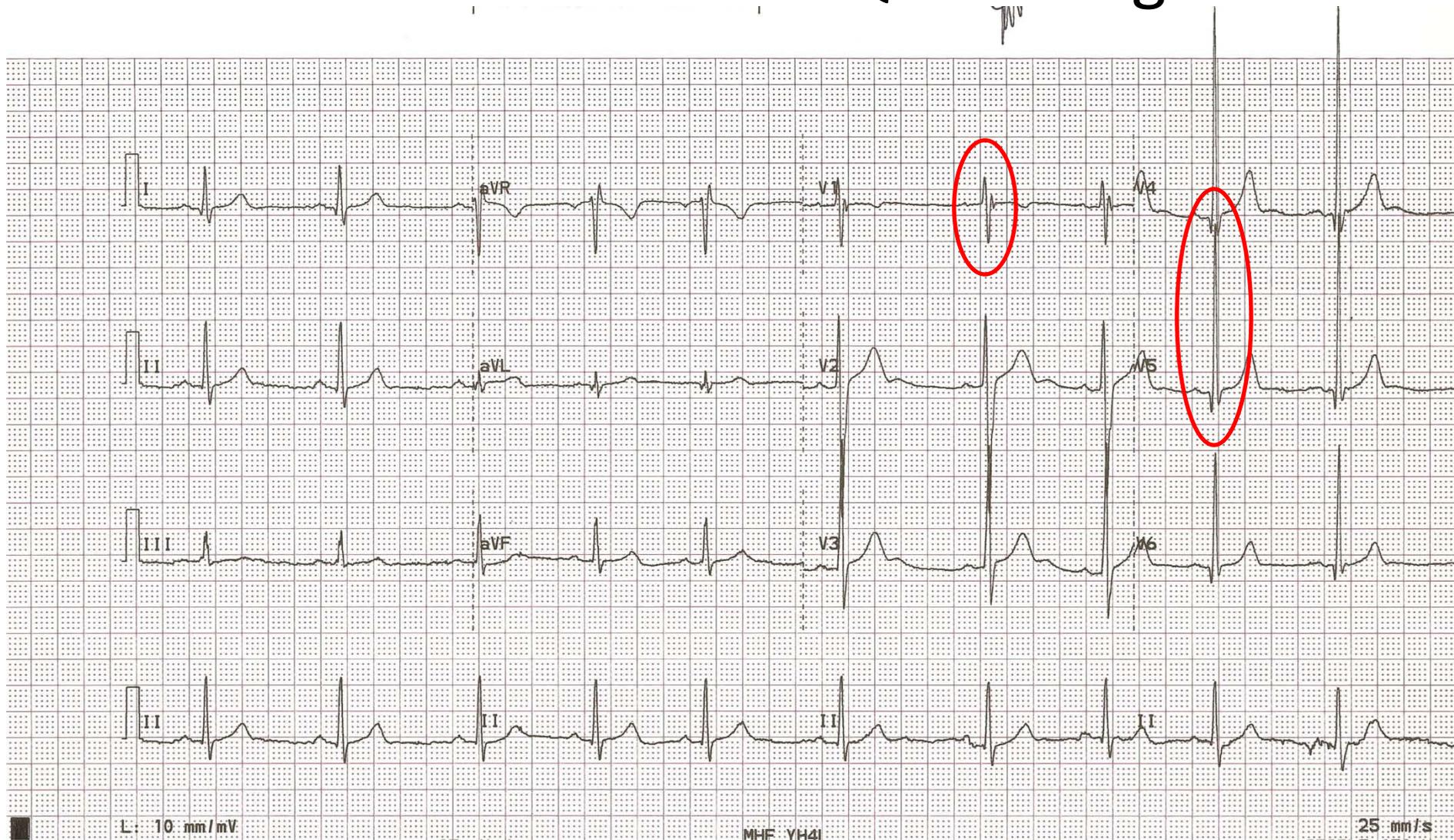
ECG from a male rower demonstrating isolated increased QRS voltage (S-V1 + R-V5 = 40 mm) without other abnormalities.

Isolated Increased QRS Voltage

ECG from a 19 year old asymptomatic rower demonstrating sinus bradycardia (42 bpm) and voltage criteria for LVH. Increased QRS amplitude without other ECG abnormalities is a common finding in trained athletes and does not require additional testing.
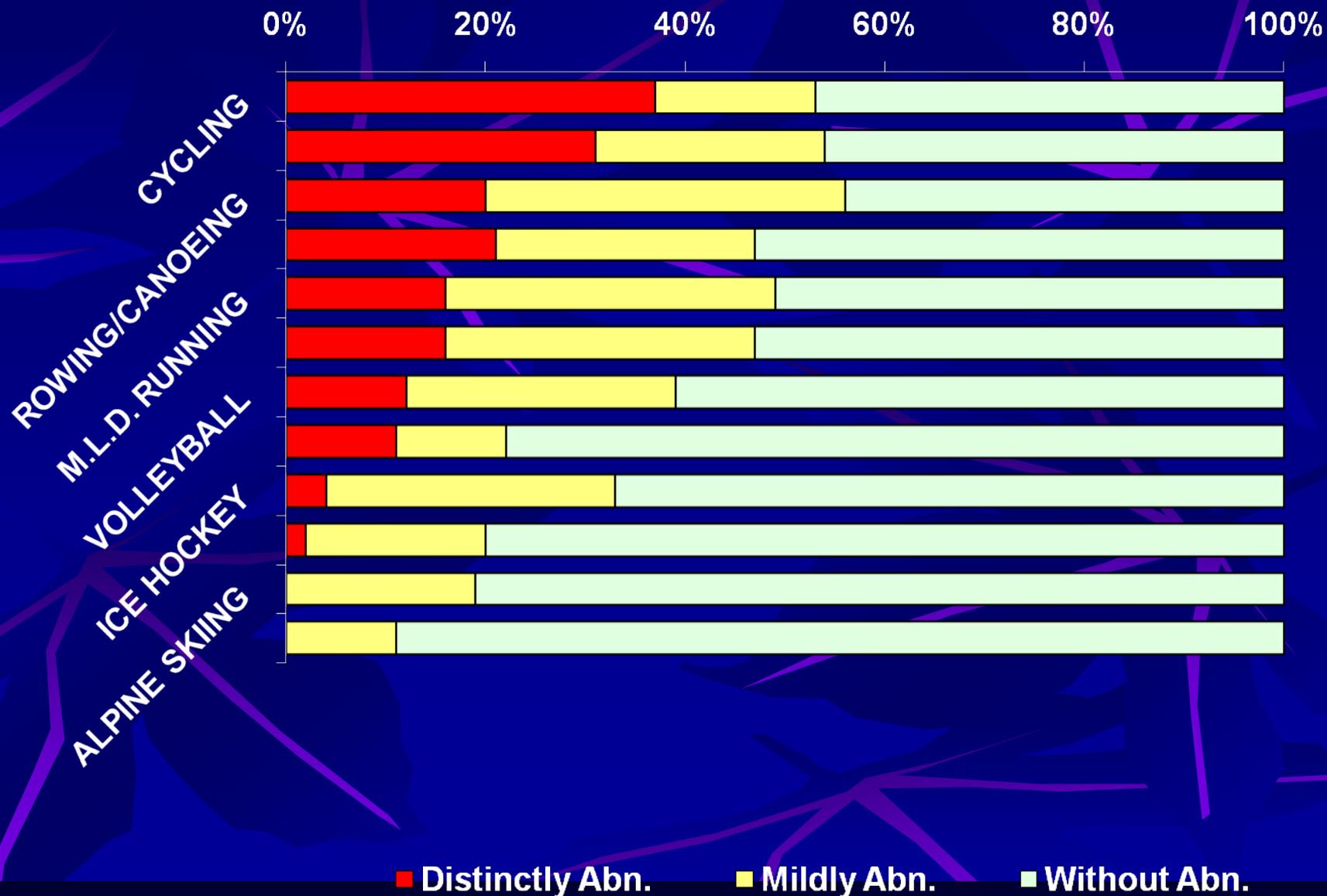
Isolated Increased QRS Voltage

ECG demonstrating isolated increased QRS voltage (S-V1 17 + R-V5 33 = 50 mm) in an 18 year old asymptomatic male. Note the absence of ST depression, T-wave inversion, pathologic Q waves, left atrial enlargement, or left axis deviation.
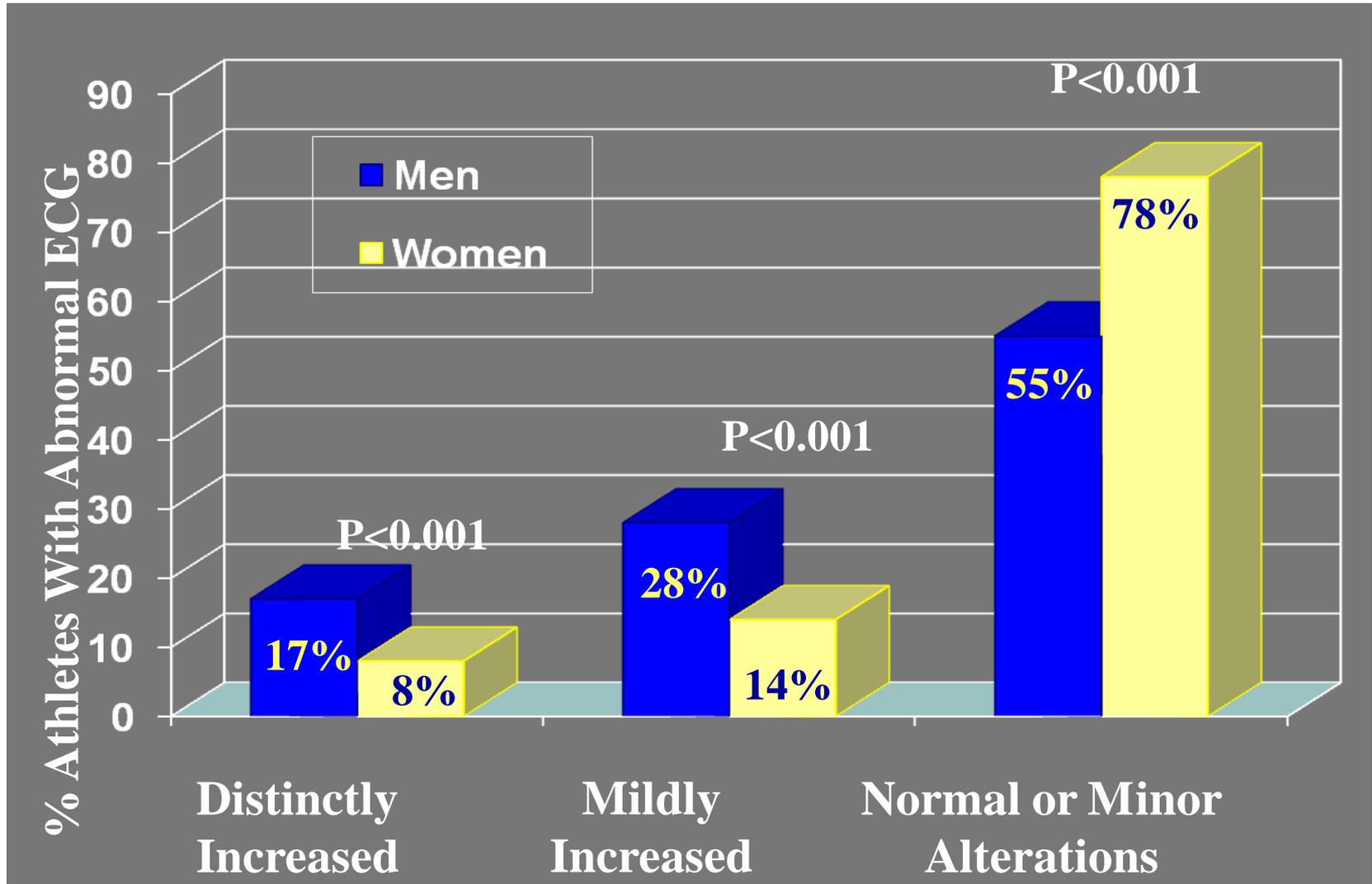
Increased QRS voltages in athletes

- A pure increase of QRS voltages accounts for about 60% of all the ECG abnormalities in athletes and occurs in the absence of underlying cardiac pathology.

- Major determinants for LVH pattern are: the extent of morphologic LV enlargement/hypertrophy, the endurance type of sport (cycling, cross-country skiing, rowing, canoeing) and the male gender.

Prevalence of criteria for LVH in relation to sport

Legend: Distinctly Abn. (red), Mildly Abn. (yellow), Without Abn. (light green)

Y-axis categories: CYCLING, ROWING/CANOEING, M.L.D. RUNNING, VOLLEYBALL, ICE HOCKEY, ALPINE SKIING

X-axis: 0%, 20%, 40%, 60%, 80%, 100%

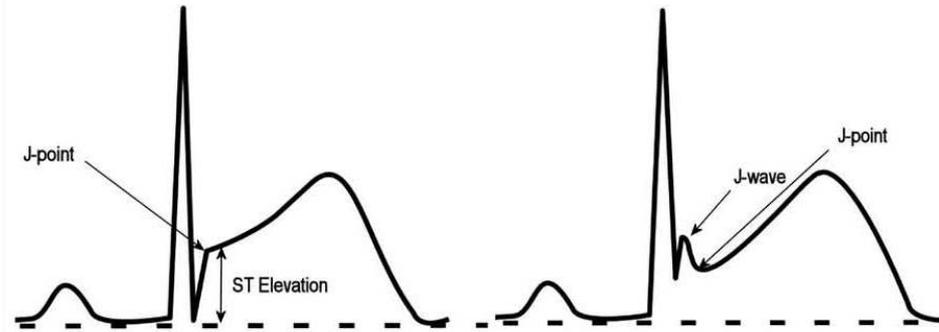Prevalence of criteria for LVH according to gender

Increased QRS voltages (LVH) in athletes

Adult Caucasian Athletes   **23 %** [1]

Adult Afro-Caribbean Athletes   **23 %** [2]

Young Caucasian Athletes   **45 %** [3]

Adolescent African Athletes   **89%** [4]

1,3) Pelliccia et al. 2000, 2011; 2,4) Sharma 1999, 2011

Early Repolarization

Classic Definition of Early Repolarization:ST Elevation

J-point

ST Elevation

Classic Early Repolarization Without a J-wave

J-point
J-wave

Classic Early Repolarization With a J-wave

New Definitions of Early Repolarization

New J-point

Terminal QRS Slurring
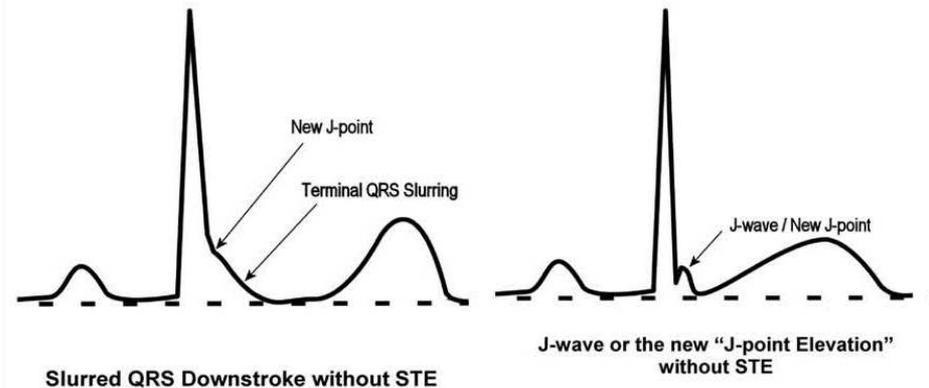
Slurred QRS Downstroke without STE

J-wave / New J-point

J-wave or the new "J-point Elevation" without STE

Figure Examples of the classic and new definitions of early repolarization and J point.
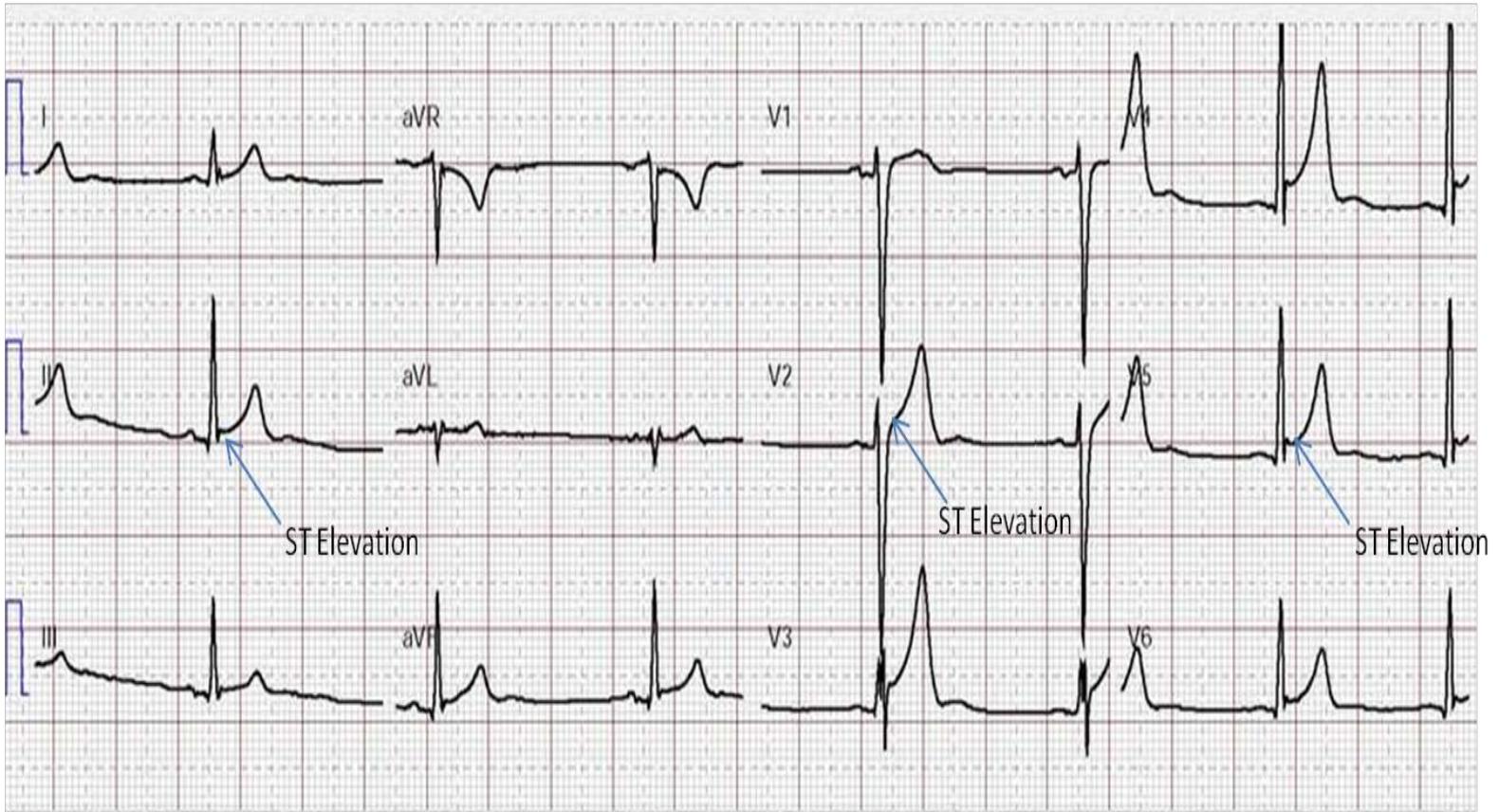
Classic and new definitions of early repolarization. (STE= ST elevation)

Patterns of ST-Segment elevation (1)

- Elevated J point, upward concavity of the ST-segment with peaked and tall terminal T-wave

- Usually, V_3 to V_5

- Present in 50-60% of elite Caucasian athletes
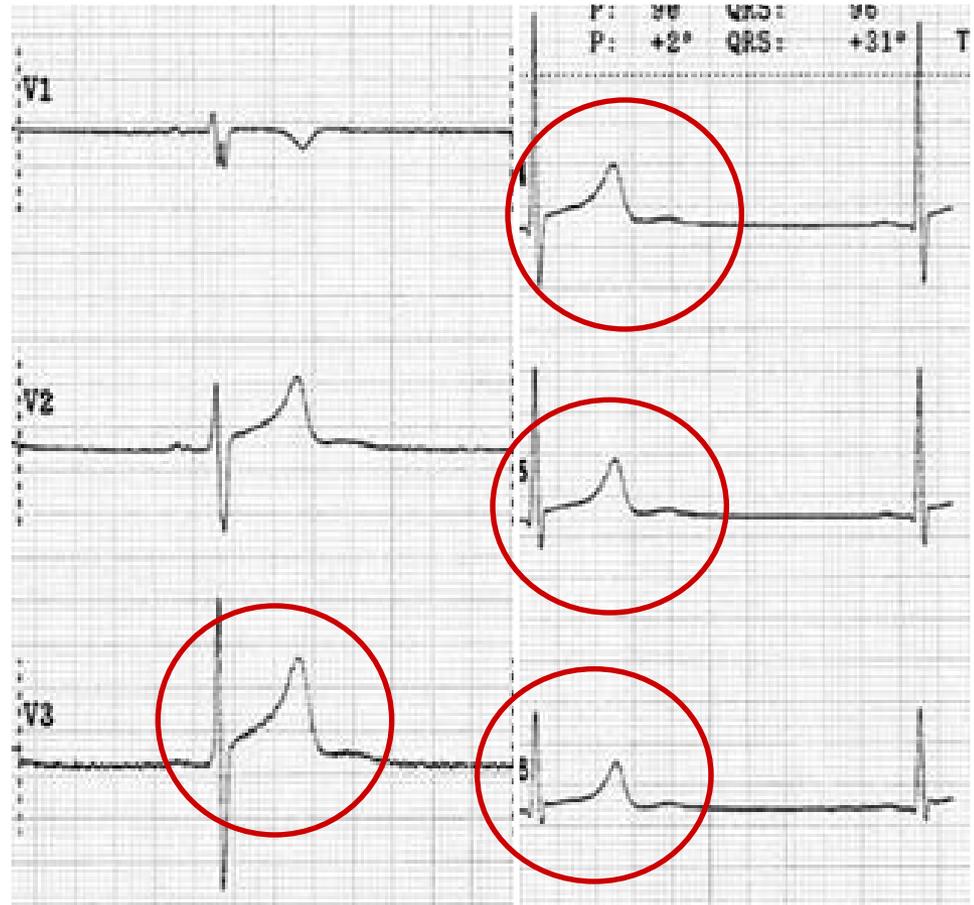
Early Repolarization

ECG shows early repolarization with ST elevation in all leads in an asymptomatic young athlete.

Early Repolarization in Black/African Athletes: Normal Variant

Patterns of ST-Segment elevation (2)

- Elevated ST-segment, convex on the top ("domed"), with small/indistinct, terminal negative T-wave

- Usually, located in leads V_2 to V_4

- Present in < 20% of elite Caucasian athletes

- More common in Afro-Caribbean (30-50%)

Normal Variant: Repolarization Changes in Black/African Athletes

A 21-year old black/African soccer player with sinus bradycardia, 1st degree AV Block, early repolarization in leads II, V2-V6 (arrows), and T-wave inversion in leads V1-V3 (circles). 'Domed' ST segment elevation followed by T wave inversion in the anterior precordial leads in black/African athletes is considered a normal finding.

Patterns of ST-Segment elevation (3)

- Mixed patterns, or a more unusual ST-segment and T wave morphology

- Less than 20% of elite athletes

Prevalence of Early Repolarization Pattern

General Population   1-2 %

Elite Caucasian Athletes   14 -26 %

Elite Afro-Caribbean Athletes   63 %

Adolescent Caucasian Athletes   44-56 %

Adolescent African Athletes   91 %

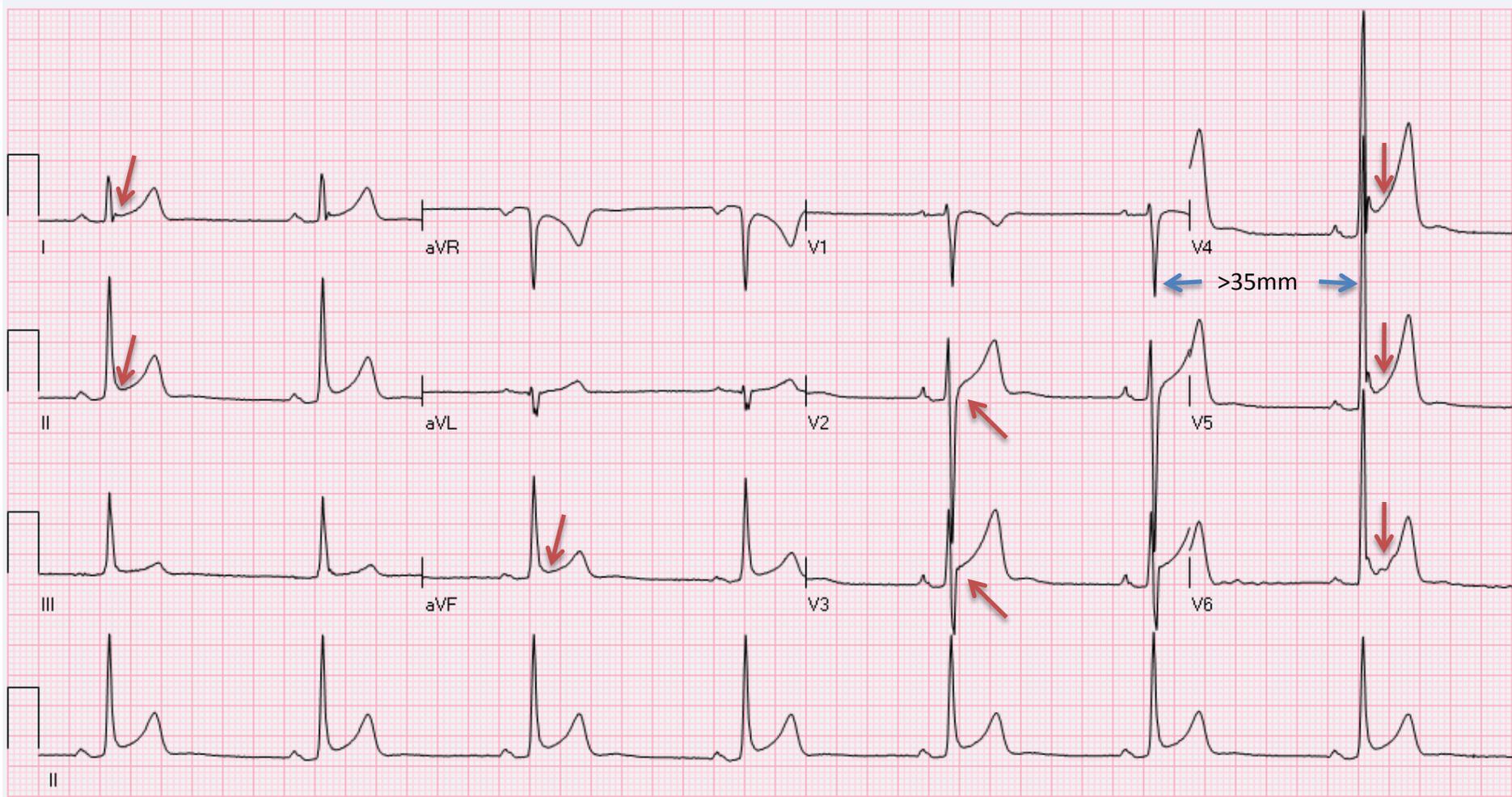*from: Pelliccia et al., Circulation 2000; Papadakis et al. Eur Heart J, 2011 and Di Paolo JACC 2012:*

Summary Examples:
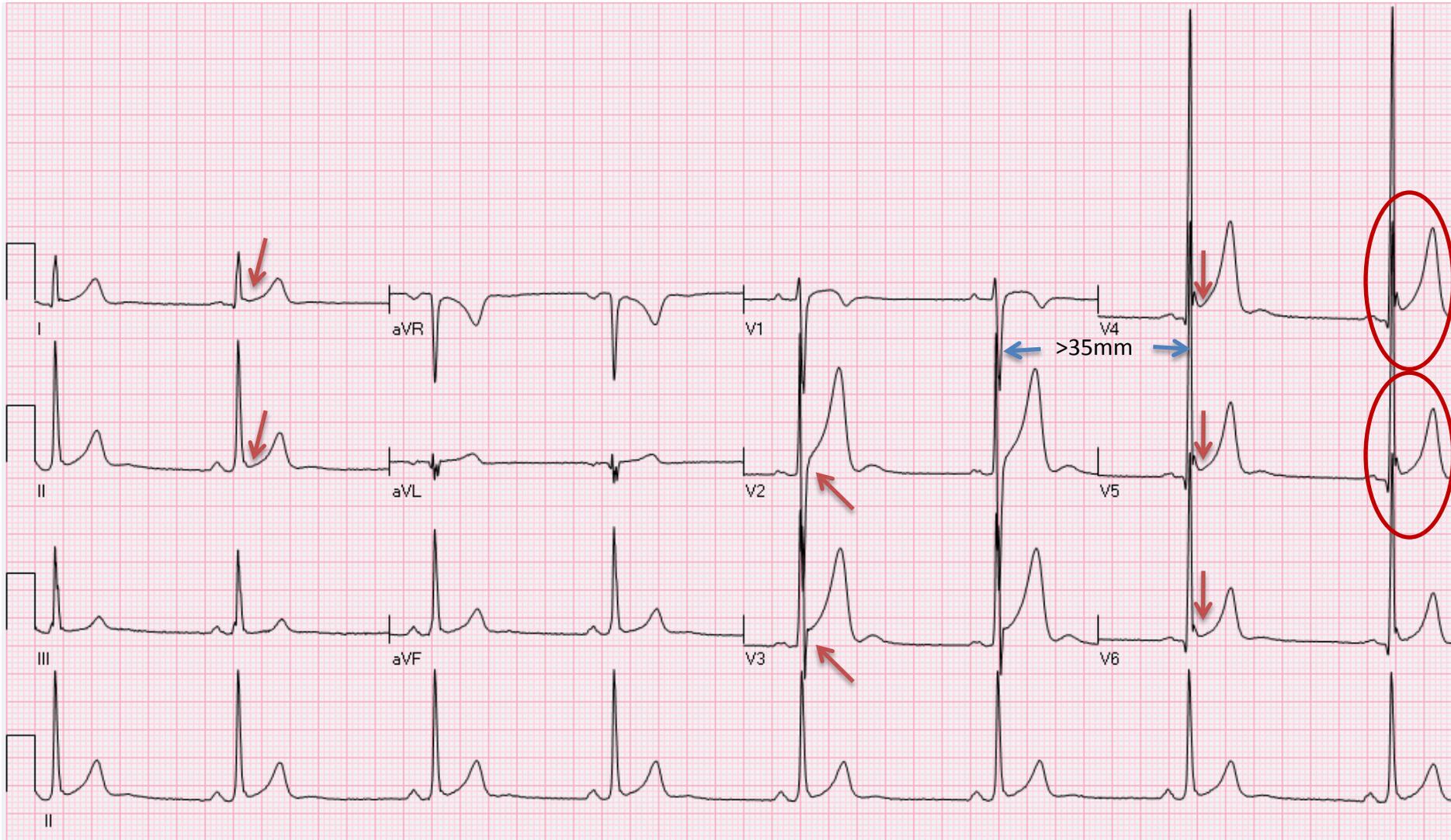# Normal ECG Findings in Athletes

Normal ECG Findings
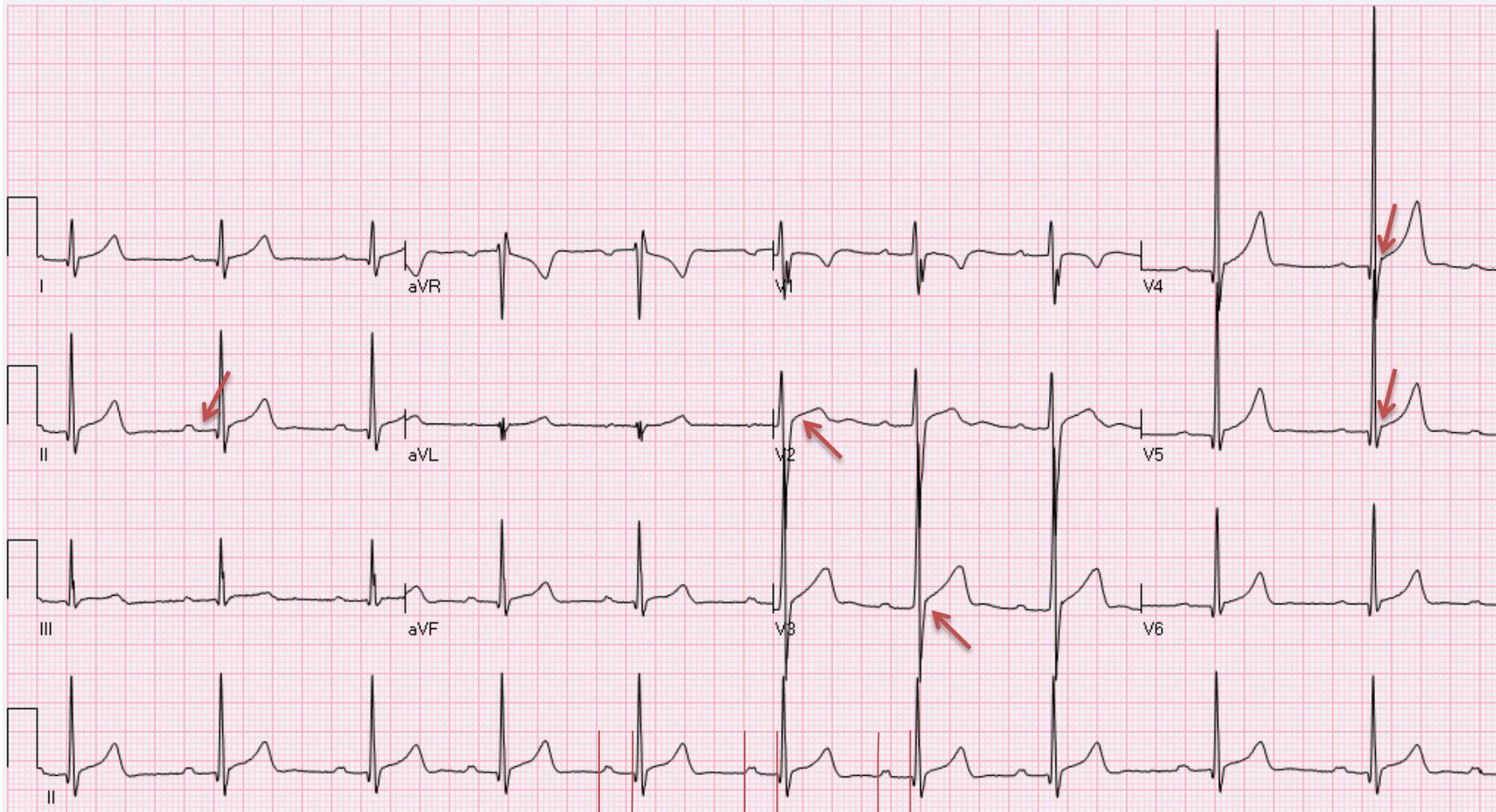
A 19-year old asymptomatic soccer player demonstrating sinus arrhythmia with 1st degree AV block (PR interval > 200 ms) and early repolarization (arrows).

Normal ECG Findings

A 29-year old asymptomatic soccer player demonstrating sinus bradycardia, early repolarization (ST elevation) in leads I, II, aVF, V2-V6 (arrows), and voltage criteria for LVH.

Normal ECG Findings

A 24 year old athlete with sinus bradycardia (45 bpm), early repolarization in I, II, V2-V6 (red arrows), voltage criteria for LVH, and peaked T-waves (circles). These are common, training-related ECG changes in athletes.

The abnormal ECGs in athletes
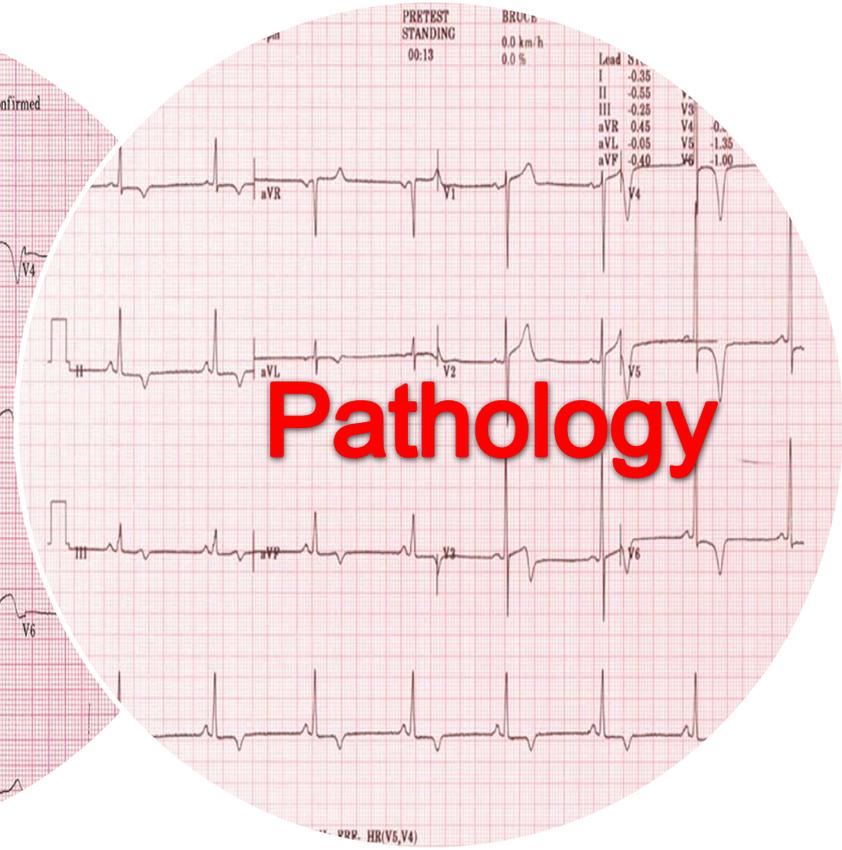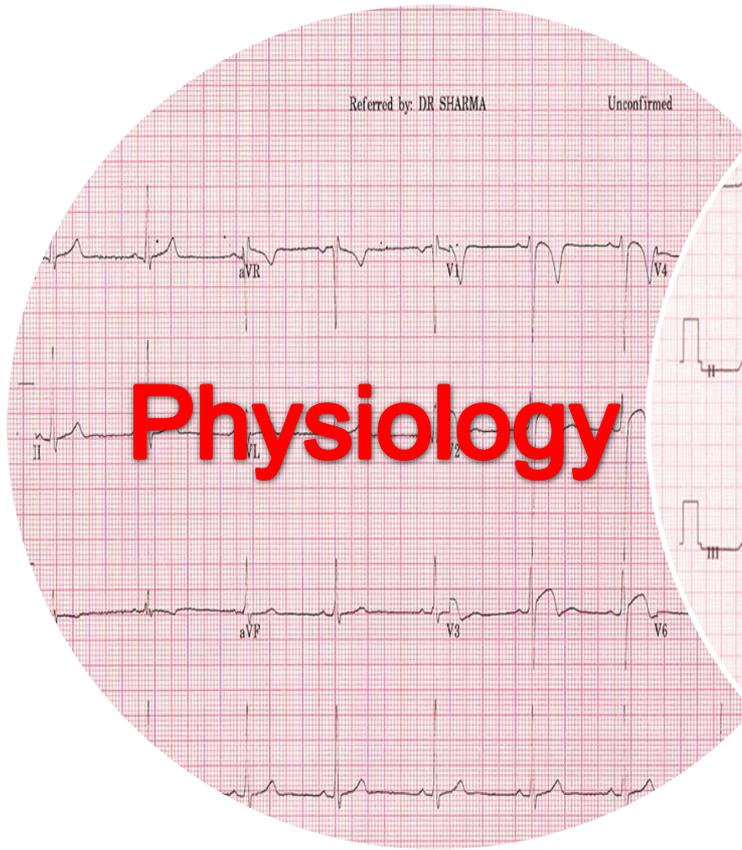
```
                    ECG abnormalities
                      in the athlete


         (Group 1)                        (Group 2)
    common (up to 80%)               Uncommon (< 5%)


 -Sinus bradycardia               - T-wave inversion
 -First degree AV block           - ST-segment depression
 -Notched QRS in V1 or            - Pathological Q waves
  incomplete RBBB                 - Left atrial enlargement
 -Early repolarization            - Left axis deviation/left anterior
 -Isolated QRS voltage criteria     hemiblock
  for left ventricular hypertrophy - Right axis deviation/left posterior
                                    hemiblock
                                  - Right ventricular hypertrophy
                                  - Complete LBBB or RBBB
                                  - Long or short QT interval
                                  - Brugada-like early repolarization
                                  - Ventricular arrhythmias
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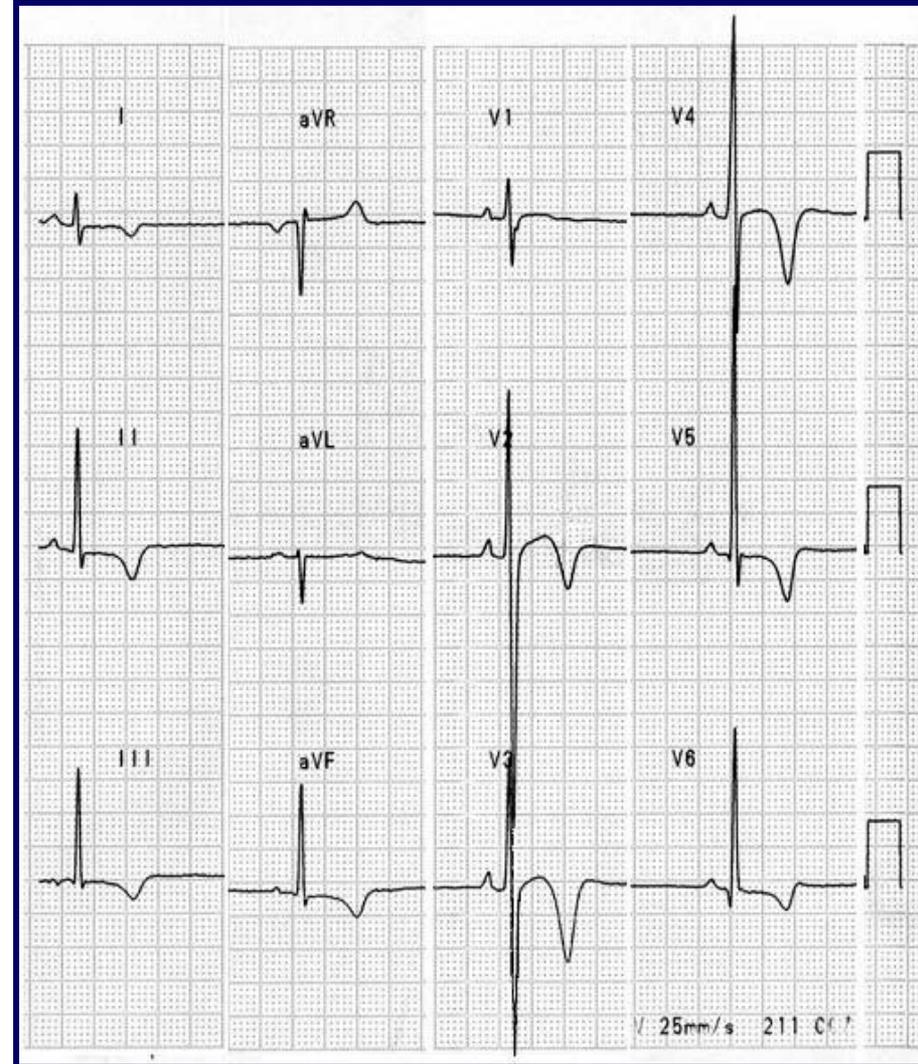
ECG abnormalities in HCM

- *Diffuse T-wave inversion in precordial (V_{4-6}) and standard (II,III,VF) leads (60%)*

- *Pathologic Q-waves (30%)*

- *Left atrial enlargement (25%)*

- *Conduction abnormalities (25%)*

- *Left axis deviation (5%)*

- *Isolated increase in R/S voltages or Normal Patterns (5%)*

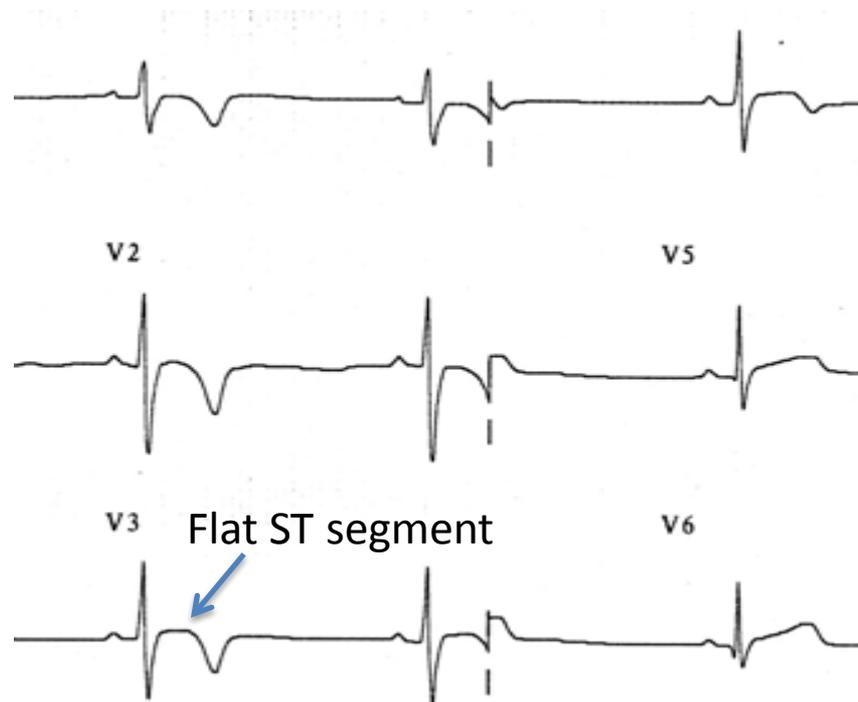# Pathologic T-wave Inversion in Black/African Athletes
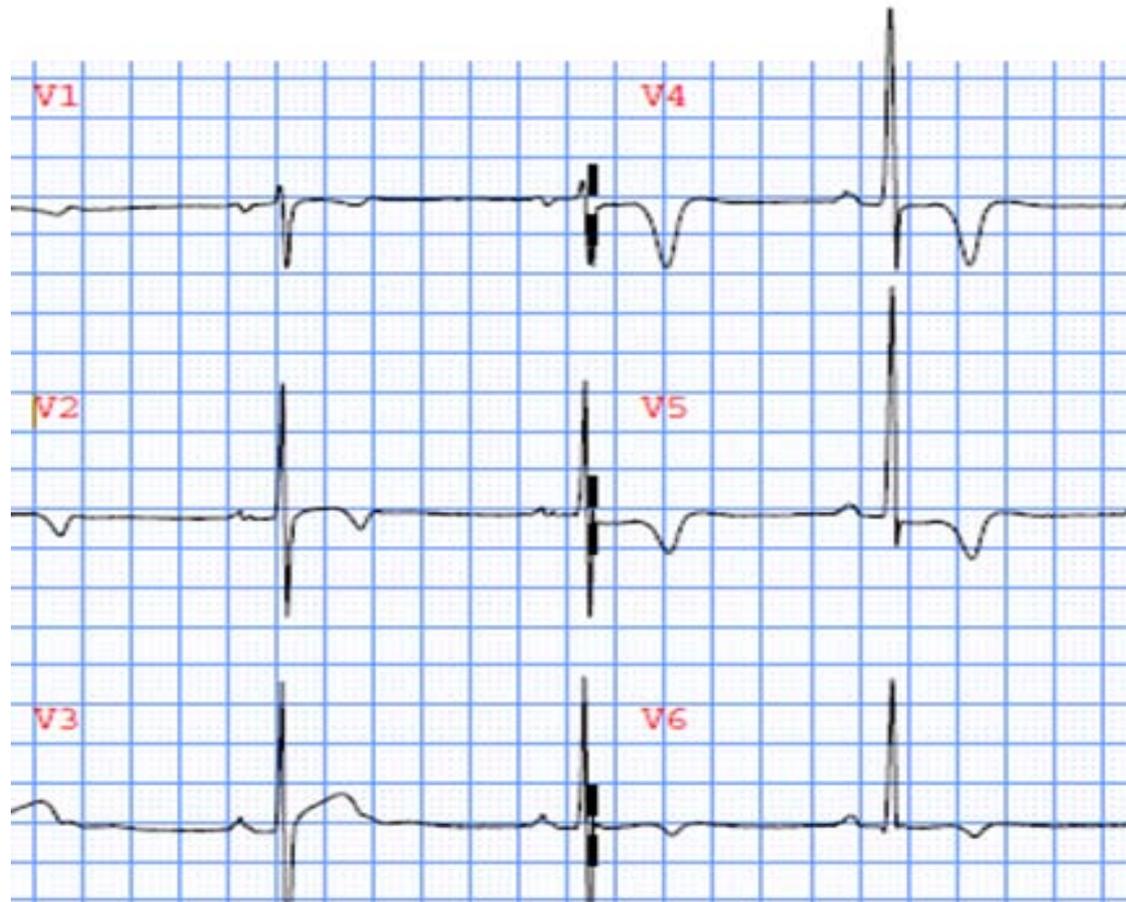
ECG shows pathologic T-wave inversion in the lateral leads. T-wave inversion in V5-V6 is always an abnormal finding and requires additional testing to rule out cardiomyopathy.

ECG abnormalities in ARVC

- Inverted T-wave in anterior precordial leads V_1 to V_3 (V_4) in individuals >14 yrs (without RBBB)

- Epsilon wave

- NSVT with LBB and superior axis configuration
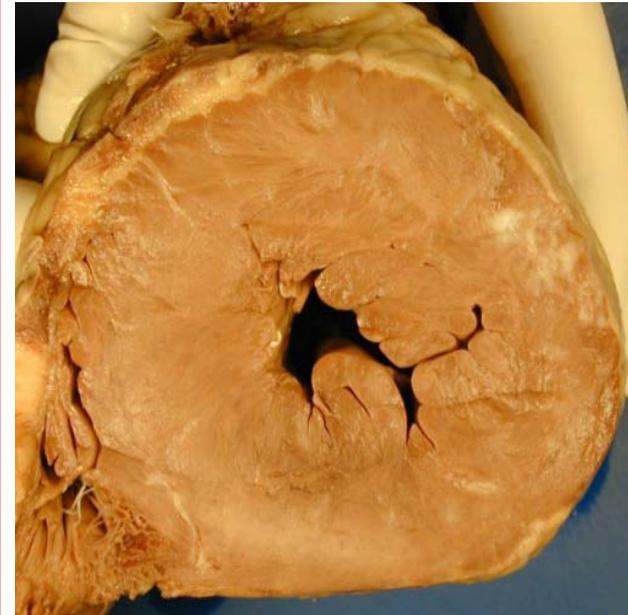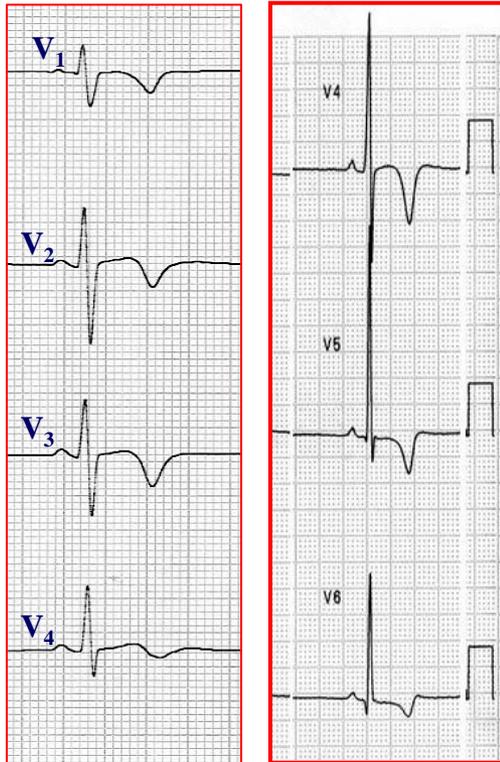
- Frequent (>500/24 h) PVBs with LLB configuration

ARVC-Pattern ECG

V2   V5

V3   Flat ST segment   V6
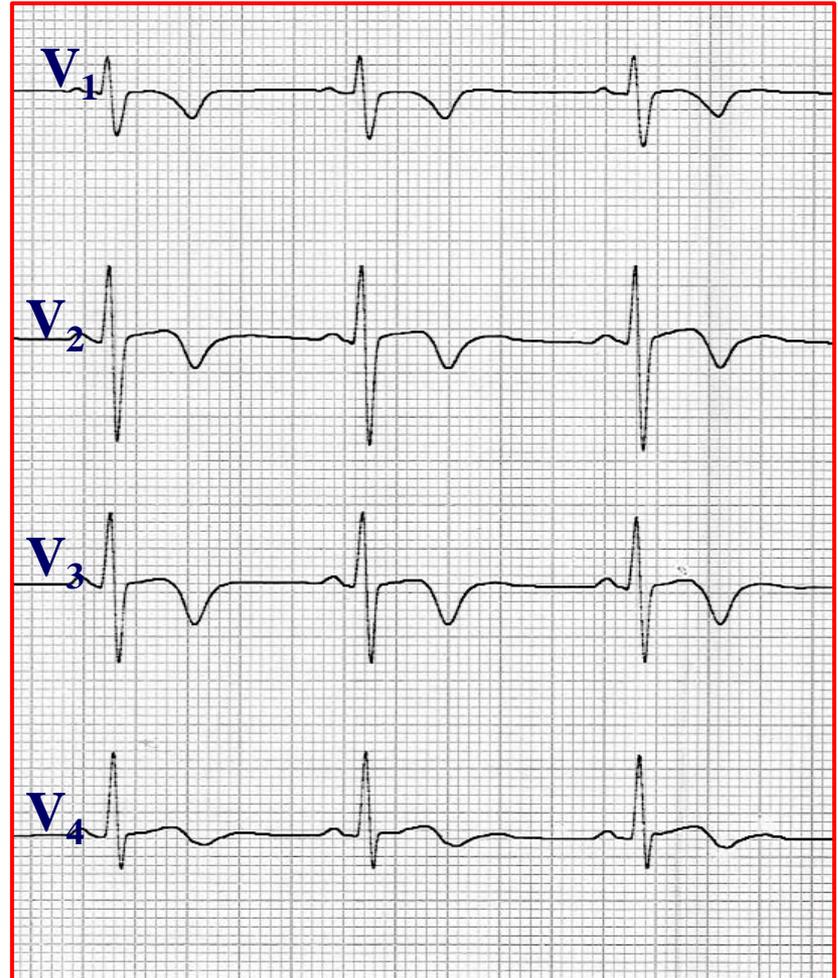
ECG shows pathologic T-wave inversion in V1-V3. Note the isoelectric ST segment. The absence of ST segment elevation prior to T-wave inversion makes this ECG abnormal. Additional testing is required to rule out ARVC.

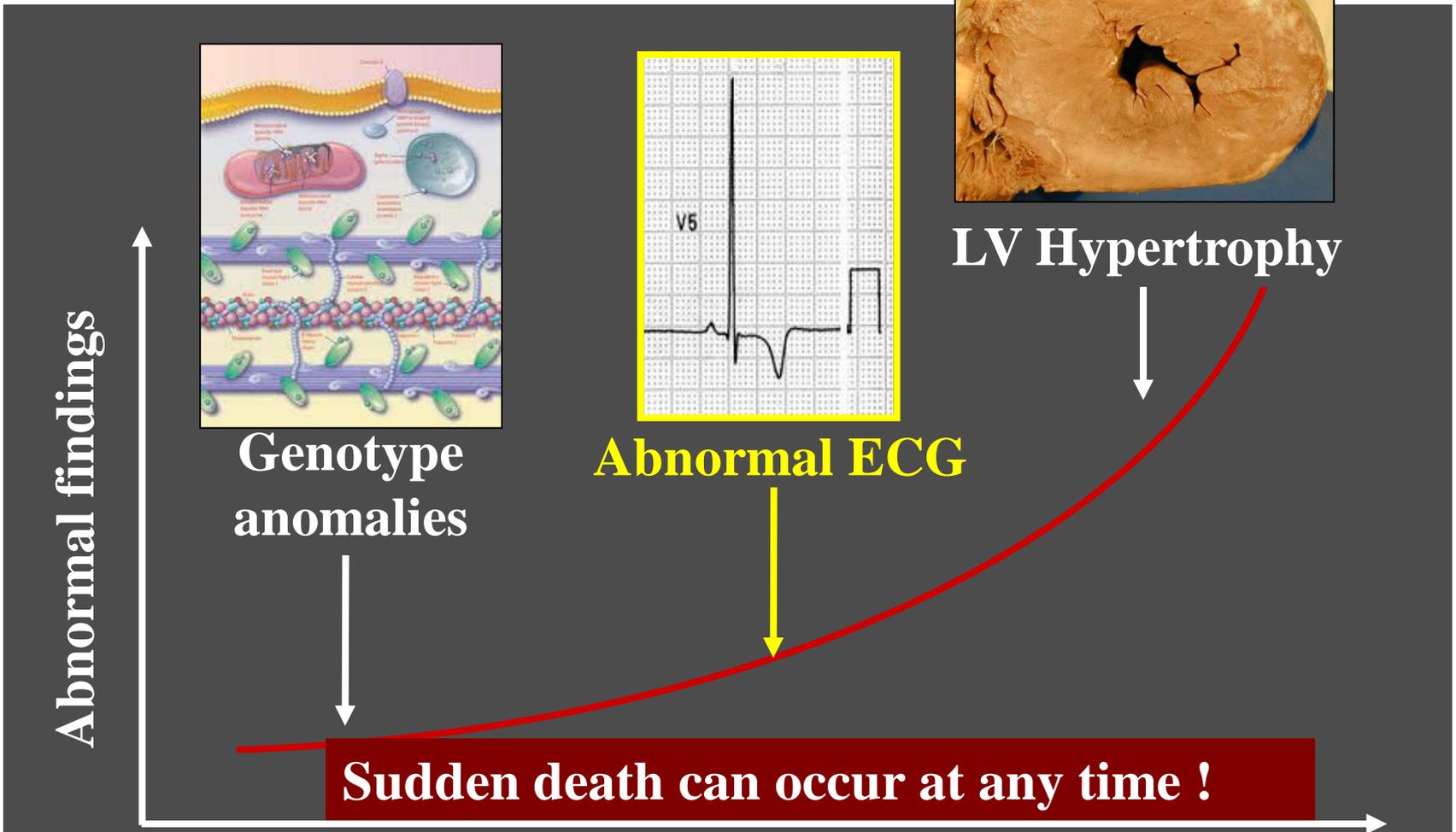# Prevalence of T-wave inversion in CMPs

up to 80%

40 to 60%

# The abnormal ECG precedes LVH in HCM

Abnormal findings

Genotype anomalies

Abnormal ECG

LV Hypertrophy

Sudden death can occur at any time !

**Adolescence**

Adulthood

Brugada syndrome

- Genetic disease with autosomal dominant pattern of inheritance

- Affects the transmebranic ion channel exchange, in the absence of structural cardiac abnormalities

- 18-30% of cases associated with Gene SCN5A (deficit of Na+ conductance);

- Electrical abnormalities present in ECG leads exploring the RV outflow tract (V_2,V_3);
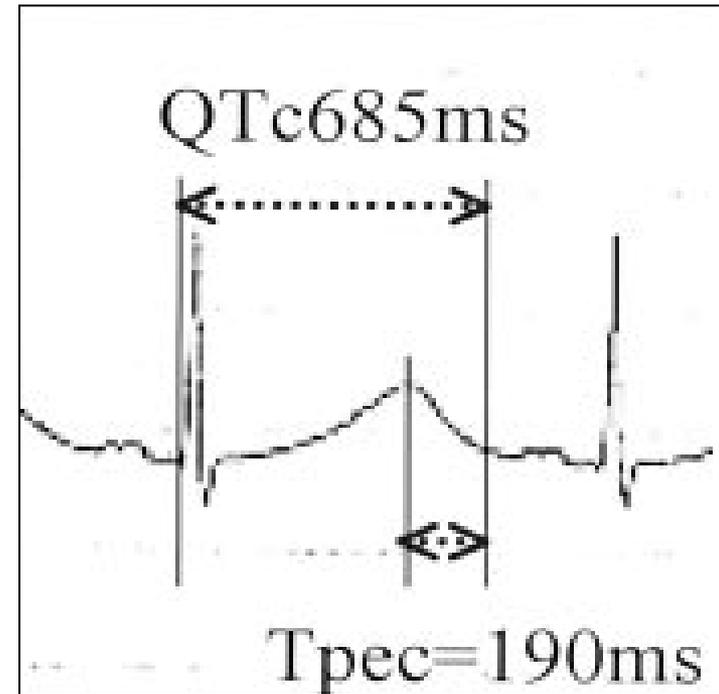
Brugada type 1
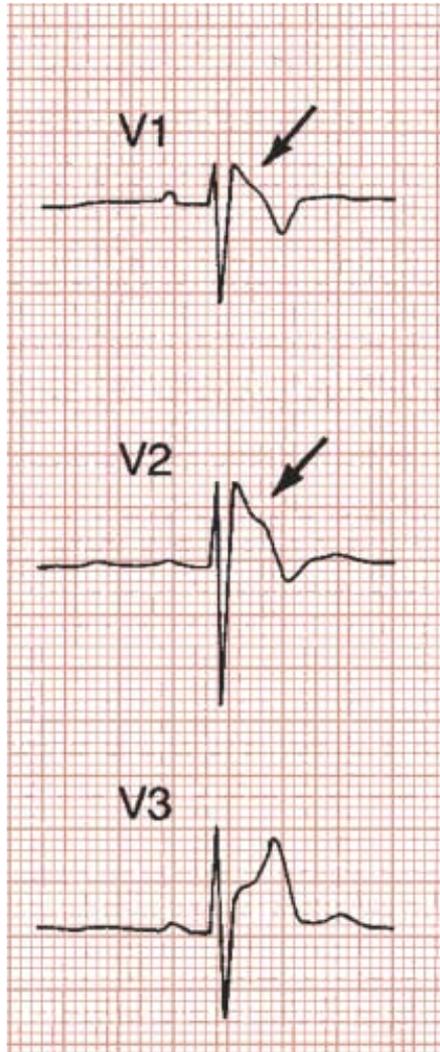
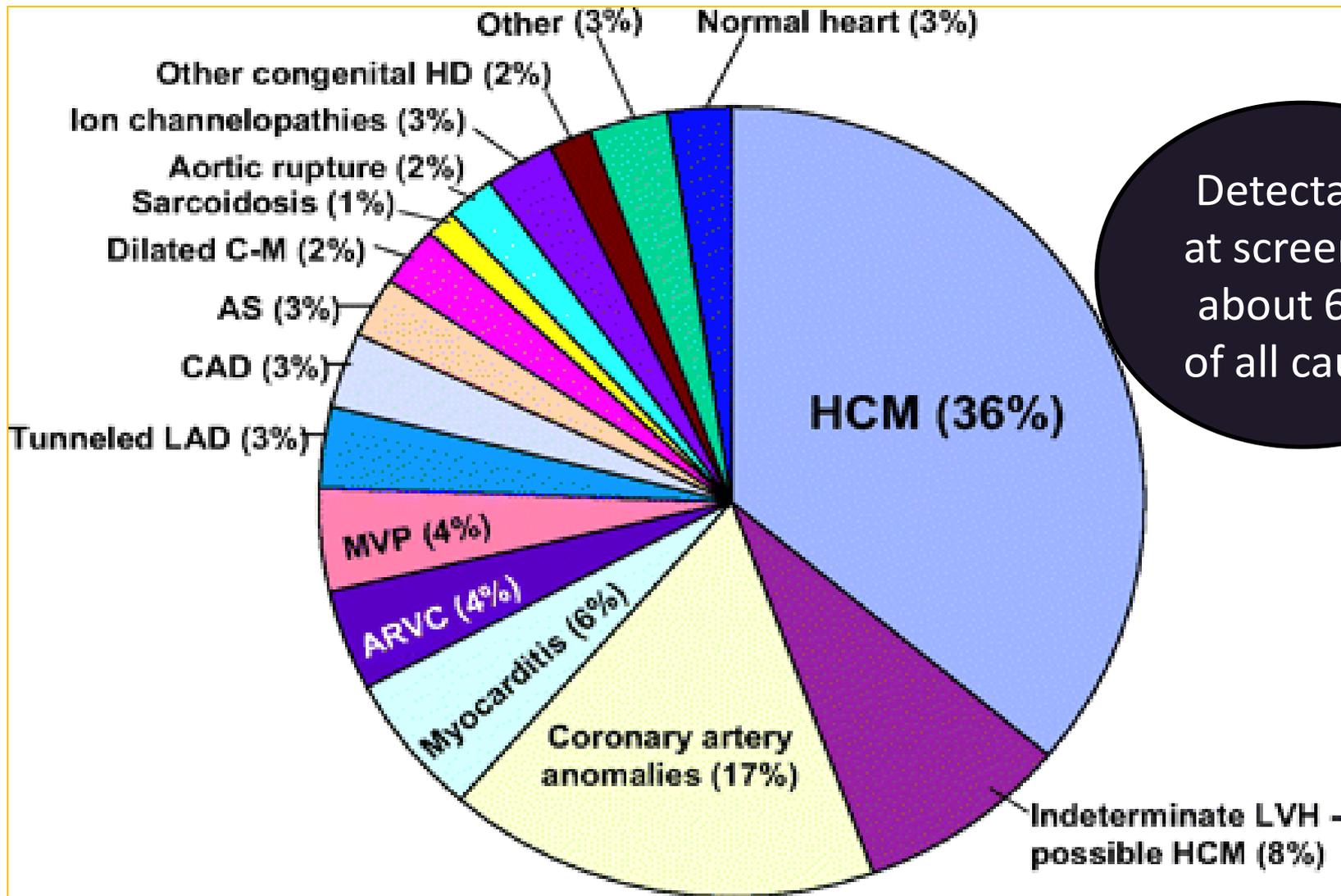# Early repolarization in trained athletes
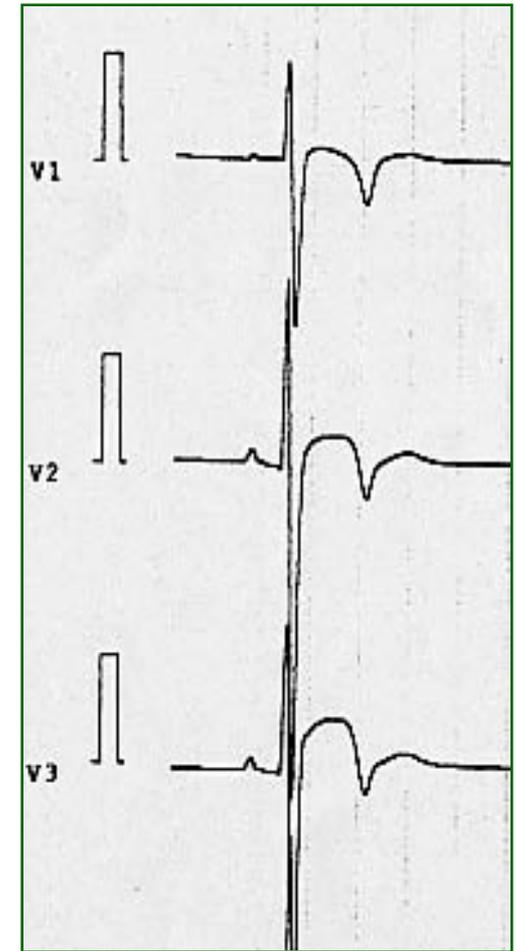
Measurement of QT interval

- The QT interval= interval between onset of QRS-complex and the end of T-wave
- Lead II (of a 12-lead ECG) is the best lead
- Measurements performed by hand
- Traces with stable heart rate and costant RR intervals
- At least three separate measurements

QTc685ms

Tpec=190ms

Known causes of SCD

Other (3%)
Normal heart (3%)
Other congenital HD (2%)
Ion channelopathies (3%)
Aortic rupture (2%)
Sarcoidosis (1%)
Dilated C-M (2%)
AS (3%)
CAD (3%)
Tunneled LAD (3%)
MVP (4%)
ARVC (4%)
Myocarditis (6%)
Coronary artery anomalies (17%)
HCM (36%)
Indeterminate LVH - possible HCM (8%)

Detectable at screening about 60% of all causes

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)
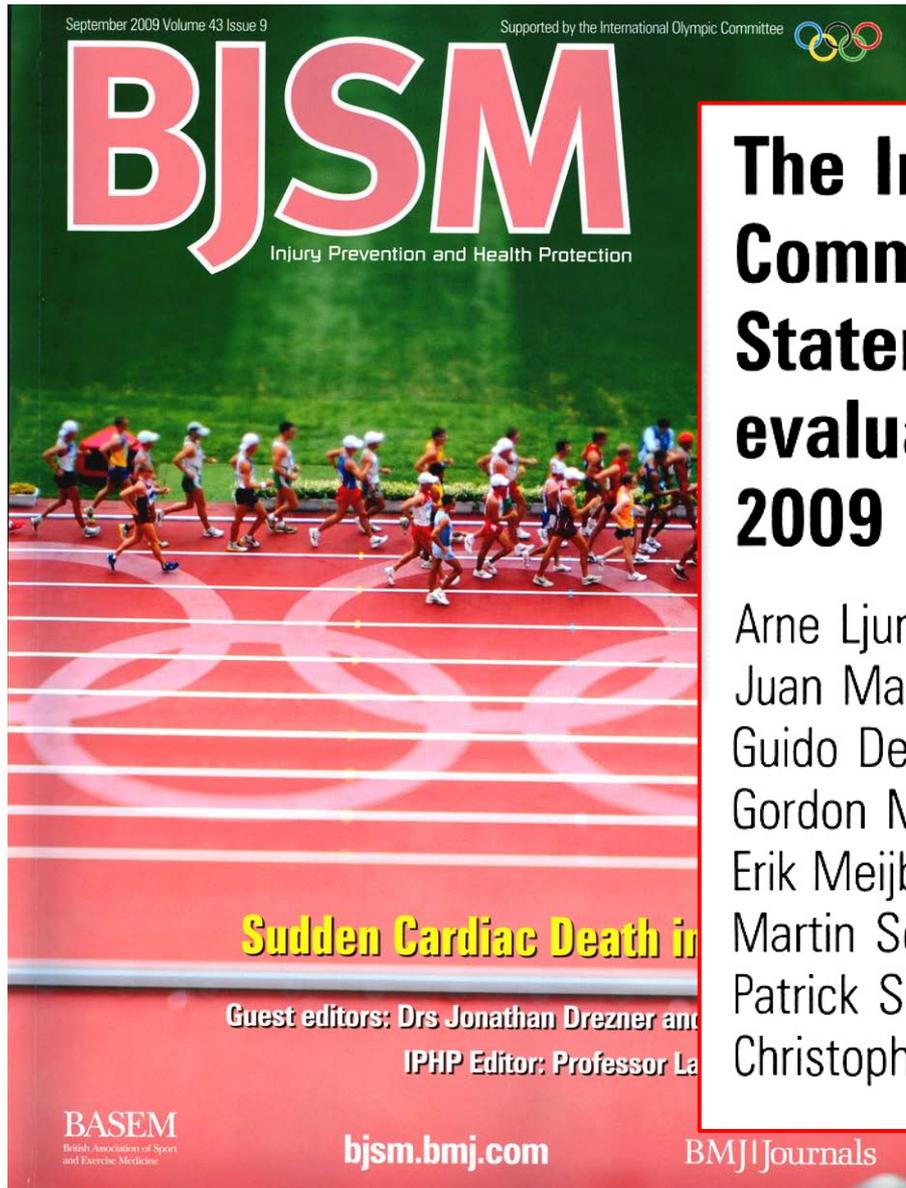

P for trend <0.001
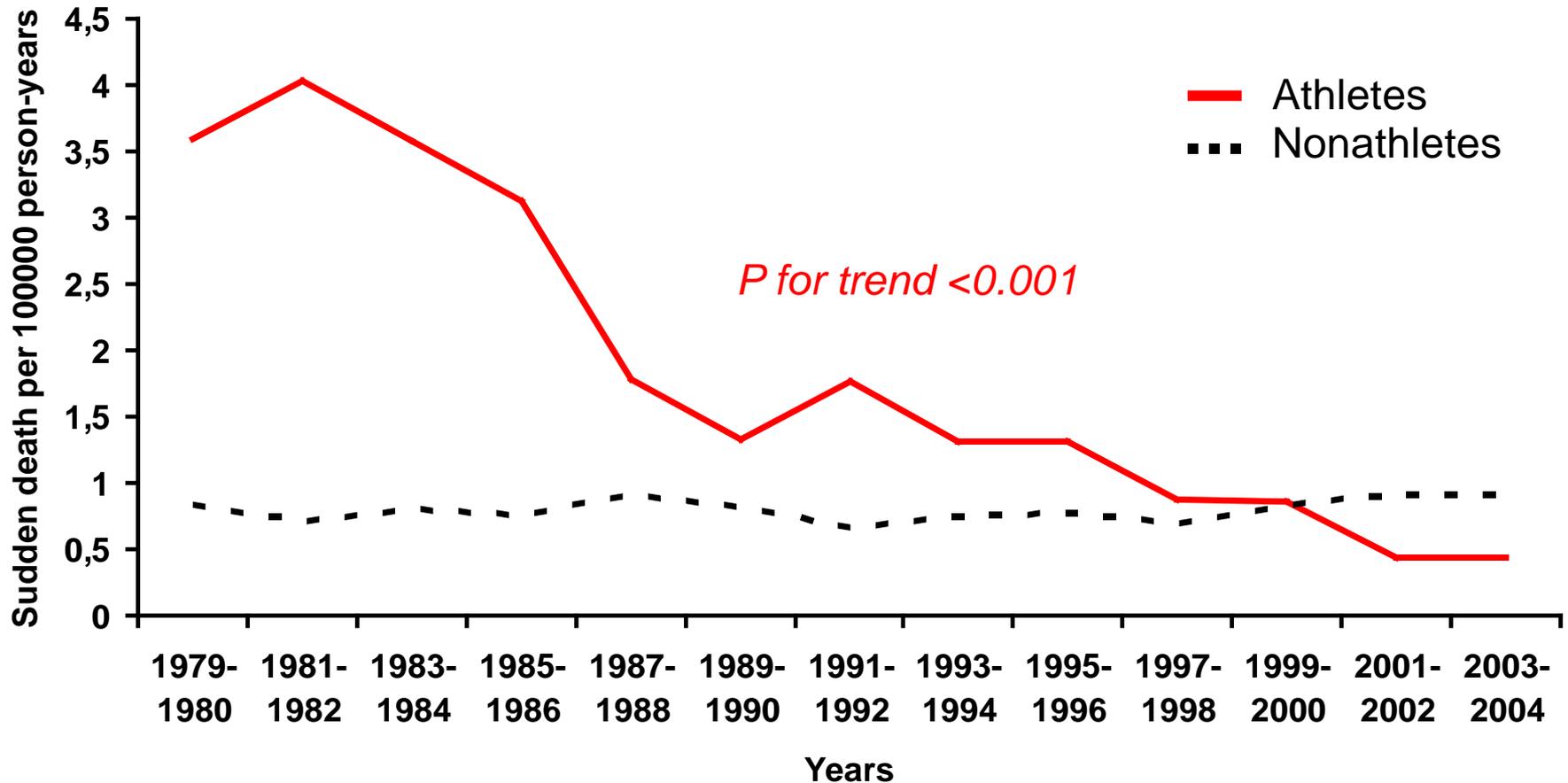
ESC Report

Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol

Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology

Domenico Corrado[1]*, Antonio Pelliccia[2], Hans Halvor Bjørnstad[3], Luc Vanhees[4], Alessandro Biffi[2], Mats Borjesson[5], Nicole Panhuyzen-Goedkoop[6], Asterios Deligiannis[7], Erik Solberg[8], Dorian Dugmore[9], Klaus P. Mellwig[10], Deodato Assanelli[11], Pietro Delise[12], Frank van-Buuren[10], Aris Anastasakis[13], Hein Heidbuchel[4], Ellen Hoffmann[14], Robert Fagard[4], Silvia G. Priori[15], Cristina Basso[19], Eloisa Arbustini[16], Carina Blomstrom-Lundqvist[17], William J. McKenna[18], and Gaetano Thiene[19]

The 2009 IOC Consensus Statement

Thank you for your attention

See also :www.antoniopelliccia.it