ECG in the Athlete

Alessandro Biffig
Institute of Sports Medicine and Science
Italian Olympic Committee – Rome, Italy
60 Milioni Italiani

11 Milioni fanno sport occasionalmente

6 Milioni S’allennano regolarmente e gareggiano

~ 2.000 Atleti di alto livello

Obiettivo della Legge

Dati CONI-ISTAT 2011
Protocolli cardiologici per il giudizio di idoneità allo sport agonistico 2009

Edizione del Ventennale
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 an *appropriate* 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.
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)
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
Asymptomatic water-polo player, 27 yrs
Davis Cup International Tennis Player, 22 yrs!
Exercise session during 24-h Holter monitoring
Carissimo Dalla Volta,

da alcuni anni seguiamo un giovane tennista di alto livello, [nome sconosciuto], 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 elettrofisico 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

Risposta di Dalla Volta

(Studio EFG)
Electrophysiologic study

Supra-hissian high-degree AV block
12-lead ECG after 27 years
Holter monitoring after 27 years
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 (STJ) and after 80 ms (ST80), and STJ/ST80 ratio.
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.
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.
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

- 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.

AHA Scientific Sessions 2007
<table>
<thead>
<tr>
<th>STUDY POPULATIONS &amp; DEMOGRAPHICS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HCM</strong> (n. 260)</td>
</tr>
<tr>
<td>Age: 42 years (3 – 82 ys)</td>
</tr>
<tr>
<td>Sex: 184 male (71%)</td>
</tr>
<tr>
<td>DNA analysis: 48 pts (18%)</td>
</tr>
<tr>
<td>Max LV WT: 22 mm ± 6 (range 16 – 46 mm)</td>
</tr>
<tr>
<td>LV EDD: 43 mm ± 10</td>
</tr>
<tr>
<td>Obstruction: 22% (57 pts)</td>
</tr>
<tr>
<td><strong>ATHLETES</strong> (n. 1005)</td>
</tr>
<tr>
<td>Age: 23 years (9 - 55 ys)</td>
</tr>
<tr>
<td>Sex: 745 male (74%)</td>
</tr>
<tr>
<td>Race: 1003 Europeans, 2 Africans</td>
</tr>
<tr>
<td>Sport discipline: 38</td>
</tr>
<tr>
<td>Training program: median of 7 years (2-30 ys)</td>
</tr>
<tr>
<td>Max LV WT: 10.7±4</td>
</tr>
<tr>
<td>LV EDD: 54 mm ± 6</td>
</tr>
</tbody>
</table>
## RESULTS: ECG PATTERNS IN HCM PTS AND IN ELITE ATHLETES

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

• 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

- Genotype +
- Phenotype -
- Abnormal ECG
- LV Hypertrophy

Sudden death may occur in every period

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
- LV Dd 47 mm
- AVS 12 mm
- Pfw 11 mm
- LA 45 mm
- EF 70%
Athlete’s heart

HCM
12-lead ECG in 2011
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%
- ARVC/D diagnosis (Echo/cardiac MR): 3 of 127 (2.3%)
- ARVC/D prevalence in this population: 0.1% (p<0.001)
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.
Sinus bradycardia 36 bpm

35 yrs, elite marathon runner
Bazett Formula

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

- QTc = 400 msec
- U wave
- 1.54 sec \( \sqrt{1.24} \)
- 490 msec
29 yrs., elite cyclist

HR 42 bpm, QT 0.53 sec, QTc 0.435

U wave
QT interval in athletes and sedentary subjects

Marathon run, Cyclists, Soccer pl., Sedentary

Da P. Zeppilli, Cardiologia dello Sport, 2007
QTc in athletes and sedentary subjects

Da P. Zeppilli, Cardiologia dello Sport, 2007
**LQT1**
(42-55%)

- "normal" T wave
- KCNQ1 gene
- \( K^+ \) channel, slow current
- Loss of function
- Syncope or SD during exercise (swimming)

**LQT2**
(35-45%)

- "notched" T wave
- KCNH2 gene (Herg)
- \( K^+ \) channel, fast current
- Loss of function
- Syncope or SD during emotional stress (alarm-clock)

**LQT3**
(8-10%)

- "late" T wave
- SCN5A gene
- \( Na^+ \) channel,
- Gain of function
- SD during sleeping
Practical guidelines for suspected long QT in Athletes

- **To exclude acquired long QT**
  (drugs, ipokalemia, 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 Borggreue, 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

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 Borggreve, MD

QT = 280 msec
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
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 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". 
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