Athlete’s ECG: What You Should Not Miss?

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moustaghfir64@gmail.com
High level sports and "cardiac remodeling"

75% of the theoretical maximum frequency

> 8 to 10 hours per week

Endurance: dilatation

Resistance: hypertrophy

Genetic predisposition?

Difference between regions and races

Diagram:

- Resistance
- Mixed
- Endurance

Legend:

- Resistance
- Mixed
- Endurance
## Main etiology: sport doctor

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>Autosomal dominant inheritance, generalized or localized hypertrophy of the left ventricle, heart failure, atrial and ventricular arrhythmia, syncope, sudden death</td>
<td>No competitive sports, consider defibrillator implantation</td>
</tr>
<tr>
<td>Commotio cordis</td>
<td>Blunt force injury to the chest resulting in ventricular fibrillation</td>
<td>Work-up to rule out underlying heart disease</td>
</tr>
<tr>
<td>Anomalous origin of coronary arteries</td>
<td>Variant site of origin of right or left coronary artery, myocardial hypoperfusion, ventricular arrhythmia</td>
<td>No competitive sports, consider referral for surgery if myocardial hypoperfusion is demonstrated</td>
</tr>
<tr>
<td>Arrhythmogenic right ventricular dysplasia/cardiomopathy</td>
<td>Enlargement, dysfunction, and fibro-fatty replacement of the right ventricle, T-wave inversions in leads V1-3 or Epsilon waves on baseline electrocardiogram of some but not all patients, ventricular tachycardia (with left bundle branch like morphology), exertional presyncope, syncope, or sudden death</td>
<td>No competitive sports, defibrillator implantation, beta-blockers or catheter ablation for ventricular tachycardia suppression and shock reduction</td>
</tr>
<tr>
<td>Idiopathic ventricular tachycardia</td>
<td>Monomorphic ventricular tachycardia, normal cardiac structure and function</td>
<td>Consider catheter ablation</td>
</tr>
<tr>
<td>Atrial fibrillation/flutter</td>
<td>Most common arrhythmia in athletes, associated with endurance training</td>
<td>Competitive sports ok if structural heart disease and rapid ventricular rate are absent, consider catheter ablation</td>
</tr>
<tr>
<td>The long QT syndrome</td>
<td>Prolonged and abnormal cardiac repolarization, acquired or inherited</td>
<td>No competitive sports, consider defibrillator implantation</td>
</tr>
<tr>
<td>Wolff-Parkinson-White syndrome</td>
<td>Delta wave, atrioventricular reentry tachycardia, sudden death risk due to antegrade conduction of atrial fibrillation</td>
<td>Electrophysiology study and catheter ablation in symptomatic athletes, investigate pathway effective refractory period if asymptomatic</td>
</tr>
<tr>
<td>Brugada syndrome</td>
<td>Autosomal dominant inheritance, RSR' and ST segment elevation in right precordial leads, syncope, ventricular fibrillation</td>
<td>No competitive sports, defibrillator implantation</td>
</tr>
<tr>
<td>Catecholaminergic polymorphic ventricular tachycardia</td>
<td>Autosomal dominant inheritance, polymorphic ventricular tachycardia or fibrillation triggered by exercise</td>
<td>No competitive sports, defibrillator implantation, beta-blocker to reduce events</td>
</tr>
<tr>
<td>The short QT syndrome</td>
<td>Shortened and abnormal cardiac repolarization, atrial fibrillation, sudden death</td>
<td>No competitive sports, consider defibrillator implantation</td>
</tr>
</tbody>
</table>
Comparison of Electrocardiographic Criteria for the Detection of Cardiac Abnormalities in Elite Black and White Athletes

Nabeel Sheikh, MRCP; Michael Papadakis, MRCP; Saqib Ghani, MRCP; Abbas Zaidi, MRCP; Sabiha Gati, MRCP; Paolo Emilio Adami, MD; François Carré, PhD; Frédéric Schnell, PhD; Mathew Wilson, PhD; Paloma Avila, MD; William McKenna, MD, DSc, FESC; Sanjay Sharma, MD, FRCP, FESC (UK)

**Background**—Recent efforts have focused on improving the specificity of the European Society of Cardiology (ESC) criteria for ECG interpretation in athletes. These criteria are derived predominantly from white athletes (WAs) and do not account for the effect of Afro-Caribbean ethnicity or novel research questioning the relevance of several isolated ECG patterns. We assessed the impact of the ESC criteria, the newly published Seattle criteria, and a group of proposed refined criteria in a large cohort of black athletes (BAs) and WAs.

**Methods and Results**—Between 2000 and 2012, 1208 BAs were evaluated with history, examination, 12-lead ECG, and further investigations as appropriate. ECGs were retrospectively analyzed according to the ESC recommendations, Seattle criteria, and proposed refined criteria, which exclude several specific ECG patterns when present in isolation. All 3 criteria were also applied to 4297 WAs and 103 young athletes with hypertrophic cardiomyopathy. The ESC recommendations raised suspicion of a cardiac abnormality in 40.4% of BAs and 16.2% of WAs. The Seattle criteria reduced abnormal ECGs to 18.4% in BAs and 7.1% in WAs. The refined criteria further reduced abnormal ECGs to 11.5% in BAs and 5.3% in WAs. All 3 criteria identified 98.1% of athletes with hypertrophic cardiomyopathy. Compared with ESC recommendations, the refined criteria improved specificity from 40.3% to 84.2% in BAs and from 73.8% to 94.1% in WAs without compromising the sensitivity of the ECG in detecting pathology.

**Conclusion**—Refinement of current ECG screening criteria has the potential to significantly reduce the burden of false-positive ECGs in athletes, particularly BAs. *(Circulation. 2014;129:1637-1649.)*

**Key Words:** cardiomyopathies ■ echocardiography ■ electrocardiography ■ ethnic groups ■ exercise ■ hypertrophy ■ mass screening
Comparison of Electrocardiographic Criteria for the Detection of Cardiac Abnormalities in Elite Black and White Athletes

Nabeel Sheikh, MRCP; Michael Papadakis, MRCP; Saqib Ghani, MRCP; Abbas Zaidi, MRCP; Sabiha Gati, MRCP; Paolo Emilio Adami, MD; François Carré, PhD; Frédéric Schnell, PhD; Mathew Wilson, PhD; Paloma Avila, MD; William McKenna, MD, DSc, FESC; Sanjay Sharma, MD, FRCP, FESC (UK)

Refined Criteria Training Related Normal Variants
Not Warranting Further Investigation*

- Sinus bradycardia
- First-degree AV block
- Incomplete RBBB
- Early repolarisation
- Isolated QRS voltage criteria for LVH

Refined Criteria Borderline Variants
Potentially Warranting Further Investigation

- Left atrial enlargement
- Right atrial enlargement
- Left axis deviation
- Right axis deviation
- Right ventricular hypertrophy
- TWI up to V4 in BAs†

Refined Criteria Training Unrelated Changes
Warranting Further Investigation

- ST-segment depression
- Pathological Q-waves
- Ventricular pre-excitation
- TWI beyond V1 in WAs beyond V4 in BAs
- Complete LBBB or RBBB
- QTc ≥470 ms in males
- ≥480 ms in females
- Brugada-like ER
- Atrial or vent. arrhythmias
- ≥2 PVCs per 10 sec tracing

If present in ISOLATION* If TWO OR MORE present

Figure 2. The number of positive ECGs produced by the 3 different ECG screening criteria.
Athlete's heart: differences between races
International recommendations for electrocardiographic interpretation in athletes


**Normal ECG Findings**
- Increased QRS voltage for LVH or RVH
- Incomplete RBBB
- Early repolarization/ST segment elevation
- ST elevation followed by T wave inversion V1-V4 in black athletes
- T wave inversion V1-V3 < age 16 years
- Sinus bradycardia or arrhythmia
- Ectopic atrial or junctional rhythm
- 1° AV block
- Mobitz Type I 2° AV block

**Abnormal ECG Findings**
- T wave inversion
- ST segment depression
- Pathologic Q waves
- Complete LBBB
- QRS ≥ 140 ms duration
- Epsilon wave
- Ventricular pre-excitation
- Prolonged QT interval
- Brugada Type 1 pattern
- Profound sinus bradycardia < 30 bpm
- PR interval ≥ 400 ms
- Mobitz Type II 2° AV block
- 3° AV block
- ≥ 2 PVCs
- Atrial tachyarrhythmias
- Ventricular arrhythmias

**Borderline ECG Findings**
- Left axis deviation
- Left atrial enlargement
- Right axis deviation
- Right atrial enlargement
- Complete RBBB

**Short PR without Preexcitation**

- In isolation
- 2 or more

**Further evaluation required**
- To investigate for pathologic cardiovascular disorders associated with SCD in athletes

**No further evaluation required**
- In asymptomatic athletes with no family history of inherited cardiac disease or SCD
normal ECG

HVG, Early repolarization

Incomplete RBBB

Coronary sinus rhythm

First degree AV block

Sinus bradycardia

TW inversion D2, D3 and VF
Questionable ECG

Right atrial enlargement

Left atrial enlargement

Right ventricular hypertrophy

Left or right axis deviation

If two or more present
ECG abnormalities and sport

1. High degree conduction disorders
   - Profound sinus bradycardia
   - Profound 1° atrioventricular block >400 ms
   - Mobitz Type II 2° atrioventricular block
   - 3° atrioventricular block

2. Depolarization disorders
   - WPW
   - LBBB
   - Anormal Q waves

3. Repolarization disorders
   - ST abnormalities
   - T wave inversion: V5,V6, D1,VL
   - J Wave syndromes
   - QT syndrome

4. Premature Beat
   - atrial
   - ventricular +++

Sports doctors
And ECG
1. High degree conduction disorders
Disturbing ECG

Left ECG:
- Date: 27.12.1956
- HR: 43 /min
- Axis P: 47°
- Axis QRS: 47°
- Axis T: -6°
- RR: 1399 ms
- PQ: 128 ms
- QRS: 246 ms
- QT: 452 ms
- QTc: 383 ms

Right ECG:
- Date: 07.04.2016
- HR: 73 /min
- Axis P: 44°
- Axis QRS: -78°
- Axis T: 30°
- RR: 827 ms
- PQ: 130 ms
- QRS: 188 ms
- QT: 134 ms
- QTc: 416 ms
Second AVB Mobitz 1
WPW syndrome
WPW and sudden death
Left bundle block
<table>
<thead>
<tr>
<th>Prénom</th>
<th>Hassan</th>
<th>Sexe : Male</th>
<th>Axe P : 37°</th>
<th>P : 124 ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>N° patient</td>
<td>1507</td>
<td>Taille : -- m</td>
<td>Axe QRS : +11°</td>
<td>PQ : 152 ms</td>
</tr>
<tr>
<td>Visite</td>
<td>--</td>
<td>Poids : -- kg</td>
<td>Axe T : 20°</td>
<td>QRS : 162 ms</td>
</tr>
<tr>
<td>Date</td>
<td>09.05.2016</td>
<td>PA : -- / -- mmHg</td>
<td>OT : 398 ms</td>
<td></td>
</tr>
<tr>
<td>Heure</td>
<td>15:46:06</td>
<td>Origine : Undefined</td>
<td>QTc : 427 ms</td>
<td></td>
</tr>
</tbody>
</table>

Rem : Moy :

### ECG

**RBBB with Wide QRS**

- **V1**
- **V2**
- **V3**
- **V4**
- **V5**
- **V6**

**Diagramme**

- **0.05-35 Hz, Onde crochetée 50Hz**
- **10 mm/mV , 25 mm/s**
<table>
<thead>
<tr>
<th>Nom</th>
<th>Prénom :</th>
<th>Né(e) le :</th>
<th>Sexe :</th>
<th>Taille :</th>
<th>Poids :</th>
<th>PA :</th>
<th>Origine :</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mohammed</td>
<td>Male</td>
<td>09.03.2005</td>
<td>Male</td>
<td>-- m</td>
<td>-- kg</td>
<td>-- / --</td>
<td>Undefined</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Date</th>
<th>Visite :</th>
<th>PA :</th>
<th>Poids :</th>
<th>Taille :</th>
</tr>
</thead>
<tbody>
<tr>
<td>19.08.2016</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>-- kg</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Heure :</th>
<th>Origine :</th>
</tr>
</thead>
<tbody>
<tr>
<td>15:48:32</td>
<td>Undefined</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>HR :</th>
<th>RR :</th>
</tr>
</thead>
<tbody>
<tr>
<td>56 /min</td>
<td>1063 ms</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Axe P :</th>
<th>Axe QRS :</th>
</tr>
</thead>
<tbody>
<tr>
<td>-- °</td>
<td>52 °</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Axe T :</th>
<th>QRS :</th>
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<tbody>
<tr>
<td>43 °</td>
<td>92 ms</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>QT :</th>
<th>QTc :</th>
</tr>
</thead>
<tbody>
<tr>
<td>454 ms</td>
<td>439 ms</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Validé par :</th>
</tr>
</thead>
<tbody>
<tr>
<td>--</td>
</tr>
</tbody>
</table>

**Rem :**

**Moy :**

**N° patient :** 1993

**N° ordre :**

**Prénom :** CHRAIBI

**Moy :**

**Né(e) le :**

**N° ordre :**

**Prov. ord. :**

**N° patient :** 1993

**Né(e) le :**

**N° ordre :**

**Prov. ord. :**

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Imprimé le 19.08.2016
Repolarization disorders
Under offset ST segment
MHC and negative T waves

D1, VL, V5 and V6: Predictive values of T-wave inversion for MHC in black athletes.
**ST segment and J wave**

<table>
<thead>
<tr>
<th>Prénom</th>
<th>Bouazza</th>
<th>Sexe</th>
<th>Male</th>
<th>Axe P</th>
<th>51°</th>
<th>P : 114 ms</th>
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</thead>
<tbody>
<tr>
<td>N° patient</td>
<td>1502</td>
<td>Taille</td>
<td>-- m</td>
<td>Axe QRS</td>
<td>12°</td>
<td>PQ : 154 ms</td>
</tr>
<tr>
<td>Visite</td>
<td>--</td>
<td>Poids</td>
<td>-- kg</td>
<td>Axe T</td>
<td>129°</td>
<td>QRS : 94 ms</td>
</tr>
<tr>
<td>Date</td>
<td>27.05.2016</td>
<td>PA :</td>
<td>-- / mmHg</td>
<td>QT : 422 ms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heure</td>
<td>18:04:05</td>
<td>Origine</td>
<td>Undefined</td>
<td>QTc : 456 ms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N° ordre</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prov. ord.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rem</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Acute IM**

Acute IM

ST segment and J wave

[Electrocardiogram with annotations]
ARVD and surface ECG

QRS duration beyond 140 ms

Rarely normal ECG in proven DAVD
<table>
<thead>
<tr>
<th>Nom :</th>
<th>Né(e) le :</th>
<th>11.11.1982</th>
<th>HR :</th>
<th>71/min</th>
<th>RR :</th>
<th>843 ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prénom :</td>
<td>Sexe :</td>
<td>Male</td>
<td>Axe P :</td>
<td>11 °</td>
<td>P :</td>
<td>80 ms</td>
</tr>
<tr>
<td>N° patient :</td>
<td>Taille :</td>
<td>-- m</td>
<td>Axe QRS :</td>
<td>14 °</td>
<td>PQ :</td>
<td>154 ms</td>
</tr>
<tr>
<td>Visite :</td>
<td>Poids :</td>
<td>-- kg</td>
<td>Axe T :</td>
<td>186 °</td>
<td>QRS :</td>
<td>106 ms</td>
</tr>
<tr>
<td>Date :</td>
<td>PA :</td>
<td>-- / -- mmHg</td>
<td>QT :</td>
<td>454 ms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heure :</td>
<td>Origine :</td>
<td>Undefined</td>
<td>QTc :</td>
<td>494 ms</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Validé par : --

N° ordre : --
Rem : --
Moy : --

**Électrocardiogramme**

- **HR** : 71/min
- **RR** : 843 ms
- **PQ** : 80 ms
- **QRS** : 154 ms
- **QT** : 454 ms
- **QTc** : 494 ms

**Mise en place**

- **Origine** : Undefined

**Né(e) le :** 11.11.1982

**N° patient :** 1460

**Visite :** --

**Date :** 07.11.2016

**Heure :** 15:51:54

**Prénom :** Abdelhamid

**Nom :** DIOURI

**N° ordre :** 1460

**Prov. ord. :**

---

**Remarques**

- **Moy** :
- **Nom** :
- **Prénom** :
- **N° patient** :
- **Visite** :
- **Date** :
- **Heure** :
- **Né(e) le** :

**Imprimé le 07.11.2016**

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Page 1 de 1
Brugada syndrome

Eight men for 1 woman
Syncope and sudden TV or FV death, often in a vagal setting
Higher incidence of SV tachycardias
First symptoms between 20 and 30 years old, but also rare cases described in children

- fast sodium current inhibitors
- Unmasking or worsening ECG abnormalities
- Intravenous injection on 5 mn of 1 mg/kg of ajmaline or on 10 mn of 2 mg/kg of flecainide
- Perform an ECG before, during and every 5 mn for 20 mn

Type 1 Brugada pattern

Miyazaki et al. J Am Coll Cardiol 1996
<table>
<thead>
<tr>
<th>Prénom :</th>
<th>Khadija</th>
<th>Sexe :</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>N° patient :</td>
<td>1262</td>
<td>Taille :</td>
<td>-- m</td>
</tr>
<tr>
<td>Visite :</td>
<td>--</td>
<td>Poids :</td>
<td>-- kg</td>
</tr>
<tr>
<td>Date :</td>
<td>25.03.2016</td>
<td>PA :</td>
<td>-- / -- mmHg</td>
</tr>
<tr>
<td>Heure :</td>
<td>16:01:08</td>
<td>Origine :</td>
<td>Undefined</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Axe P :</th>
<th>34 °</th>
<th>P :</th>
<th>98 ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axe QRS :</td>
<td>62 °</td>
<td>PQ :</td>
<td>150 ms</td>
</tr>
<tr>
<td>Axe T :</td>
<td>77 °</td>
<td>QRS :</td>
<td>114 ms</td>
</tr>
<tr>
<td>QT :</td>
<td>380 ms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>QTc :</td>
<td>428 ms</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

N° ordre : --
Rem : --
Moy : --- 3ème EIC

Préposé par : --
Né(e) le : --
N° ordre : 1262
Prov. ord. : 25.03.2016
Imprimé le : 25.03.2016

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J-Point Elevation in Survivors of Primary Ventricular Fibrillation and Matched Control Subjects

Incidence and Clinical Significance

JACC 2008

Raphael Rosso, MD,* Evgeni Kogan, MD,* Bernard Belhassen, MD,* Uri Rozowski, MD,* Melvin M. Scheinman, MD,§ David Zeitser, MD,* Amir Halkin, MD,* Arie Steinvil, MD,* Karin Heller, MD,* Michael Gilison, MD,† Amos Katz, MD,‡ Sami Viskin, MD*

Tel Aviv and Beer-Sheva, Israel, and San Francisco, California

---

A. J wave

- Slur
- Notched

B. ST segment pattern

- Ascending/up-sloping
- Horizontal
- Descending

---

<table>
<thead>
<tr>
<th></th>
<th>Any J-point elevation</th>
<th>J-point elevation &gt; 1 mm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
</tr>
<tr>
<td>Control Adults</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Young Athletes</td>
<td>**</td>
<td>**</td>
</tr>
<tr>
<td>Idiopathic VF</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

Outward shift of balance of currents during early phases of the action potential

- J Wave Syndromes
- Brugada Syndrome
- Early Repolarization Syndrome
- RVOT

---

Increasing risk

- Short coupled VPBs
- Family history of sudden cardiac death
- Dynamic J point elevation; augmented at slower heart rates
- Associated pathology: Brugada syndrome, SQTS, fragmented QRS
- More widespread J wave distribution
- Increased J wave amplitude
- Horizontal/descending ST segment

Heart rhythm 2015
Early repolarization syndrome

<table>
<thead>
<tr>
<th>Sexe</th>
<th>Taille</th>
<th>Poids</th>
<th>IMC</th>
<th>PA</th>
<th>Axis</th>
<th>P</th>
<th>PQ</th>
<th>QRS</th>
<th>QT</th>
<th>QTC</th>
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<tbody>
<tr>
<td>M</td>
<td>0 cm</td>
<td>0 kg</td>
<td>--</td>
<td>--</td>
<td>63°</td>
<td>138 ms</td>
<td>196 ms</td>
<td>110 ms</td>
<td>402 ms</td>
<td>391 ms</td>
</tr>
</tbody>
</table>

Traitement méd.:
LQT syndromes

PA : LQT3

Ikr: rapide
Iks: lent

80%

<table>
<thead>
<tr>
<th>QTL</th>
<th>Gène</th>
<th>Protéine</th>
<th>Courant ionique</th>
<th>Fréquence</th>
</tr>
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<tbody>
<tr>
<td>LQT1</td>
<td>KCNQ1</td>
<td>K_v 7.1\alpha</td>
<td>I_{Ks}</td>
<td>30-35 %</td>
</tr>
<tr>
<td>LQT2</td>
<td>KCNH2</td>
<td>K_v 7.1\alpha</td>
<td>I_{Kr}</td>
<td>25-30 %</td>
</tr>
<tr>
<td>LQT3</td>
<td>SCN5A</td>
<td>Na_v 1.5\alpha</td>
<td>I_{Na} up</td>
<td>5-10 %</td>
</tr>
<tr>
<td>LQT4</td>
<td>ANK2</td>
<td>Ankyrin-B</td>
<td>I_{K_{Na,LK},I_{K_{Ca}}}</td>
<td>1-2 %</td>
</tr>
<tr>
<td>LQT5</td>
<td>KCNE1</td>
<td>minK\beta</td>
<td>I_{Kr}</td>
<td>1 %</td>
</tr>
<tr>
<td>LQT6</td>
<td>KCNE2</td>
<td>MIRP1\beta</td>
<td>I_{Kr}</td>
<td>Rare</td>
</tr>
<tr>
<td>LQT7*</td>
<td>KCNJ2</td>
<td>Kir2.1\alpha</td>
<td>I_{Kr}</td>
<td>Rare</td>
</tr>
<tr>
<td>LQT8F</td>
<td>CACNA1C</td>
<td>Ca_v 1.2\alpha1c</td>
<td>I_{Ca}1\alpha</td>
<td>Rare</td>
</tr>
<tr>
<td>LQT9</td>
<td>CAV3</td>
<td>Caveolin-3</td>
<td>I_{Na} up</td>
<td>Rare</td>
</tr>
<tr>
<td>LQT10</td>
<td>SCN4B</td>
<td>Na_v1.5\beta</td>
<td>I_{Na} up</td>
<td>Rare</td>
</tr>
<tr>
<td>LQT11</td>
<td>AKA9</td>
<td>Yotiao</td>
<td>I_{Ks}</td>
<td>Rare</td>
</tr>
<tr>
<td>LQT12</td>
<td>SNTA1</td>
<td>A1-syntrophin</td>
<td>I_{Na} up</td>
<td>Rare</td>
</tr>
</tbody>
</table>

i_{Ca}: courant calcique entrant dépolarisant (lent) ; i_{Kr}: courant rectifiant entrant ; i_{Ks}: courant potassique sortant repolarisant rapide ; i_{Na}: courant potassique sortant repolarisant lent ; i_{Na}: courant entrant sodique rapide ; *Syndrome d’Andersen-Tawil ; 1Syndrome de Timothy

LQT syndrome

QTc duration of more than 460 ms in children < 15 years of age, more than 450 ms in adult men, or more than 470 ms in adult women using the Bazett formula identifies QT prolongation.

The QT is best measured in lead II, lead V5, or lead V6
If you have U wave on T wave you must include it in the measurement
SQT syndrome

< 300 ms

PA : SQTS
Premature ventricular beat
PVB: QRS morphology

More the QRS is “broad and dwarf”, more we have heart disease...

Ample (tall) fine late pouring monomorphic sinus QRS are normal

L’ESV bénigne est généralement ample et fine à couplage tardif monomorphe à type de retard gauche (à prédominance négative V1) et axe inférieur (positive en D2, D3 et AVF) (figure n°65). La transition du QRS précoce (V1-V2) ou tardive (V3-V4) renseigne sur la topographie respectivement droite ou gauche. Elle ne s’aggrave pas à l’effort et elle ne s’accompagne pas d’onde T négative de V1 à V3 sur les complexes QRS normaux. Ce dernier cas impose d’éliminer formellement une DAVD quand il est associé à des ESV.

A. Moustaghfir, ECG du sportif de haut niveau. 2018
More the QRS is “broad and dwarf”, more we have heart disease...

Ample (tall), fine, late pouring, monomorphic sinus QRS are normal
Ventricular tachycardia in bursts
EXTRASYSTOLIE VENTRICULAIRE ET DYSPLASIE VENTRICULAIRE DROITE ARYTHMOGÈNE

ANALYSE CRITIQUE DE LA VALEUR DIAGNOSTIQUE DES EXAMENS NON INVASIFS

A. Moustagifir, J.C. Delaro, L. Fourcade, J.P. Van de Walle, B. Mafart, P. Djiane, J.E. Touze

Presse Med 1996;25:546-8

ESHV : épreuve du temps! PVB: Test of time!
ESV infundibulaires ?
Exercise ECG
How to evaluate premature ventricular beats in the athlete: critical review and proposal of a diagnostic algorithm

Domenico Corrado, Jonathan A Drezner, Flavio D’Ascenzi, Alessandro Zorzi

**Table 3** Classification and risk stratification of premature ventricular beats in the athlete

<table>
<thead>
<tr>
<th>Common</th>
<th>Uncommon</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PVB characteristics</strong></td>
<td></td>
</tr>
<tr>
<td>Ectopic QRS morphology</td>
<td>LBBB/inferior axis, typical RBBB and narrow QRS (&lt;130 ms)</td>
</tr>
<tr>
<td>Response to exercise testing</td>
<td>Decrease/suppression</td>
</tr>
<tr>
<td>Complexity of PVBs</td>
<td>Isolated, monomorphic</td>
</tr>
<tr>
<td>Short coupling interval*</td>
<td>No</td>
</tr>
<tr>
<td><strong>Clinical findings</strong></td>
<td></td>
</tr>
<tr>
<td>Symptoms</td>
<td>No</td>
</tr>
<tr>
<td>Family history of premature SCD† or cardiomyopathy</td>
<td>No</td>
</tr>
<tr>
<td>Other ECG abnormalities</td>
<td>No</td>
</tr>
<tr>
<td>Imaging abnormalities</td>
<td>No</td>
</tr>
</tbody>
</table>

*PVBs are superimposed on the preceding T-wave peak or earlier (ie, R on T).  †Premature sudden cardiac death (SCD) is defined as that occurring before 40 years of age in men and before 50 years old in women.  ‡Couplets, triplets or non-sustained ventricular tachycardia.

LBBB, left bundle branch block; PVBs, premature ventricular beats; RBBB, right bundle branch block.

**Figure 6** Proposed algorithm for evaluation of athletes with premature ventricular beats. *24-hour ECG monitoring should ideally have 12-lead configuration and include a training session. NEG, negative; POS, positive; PVBs, premature ventricular beats.
P « on » T wave : APB
Atrial fibrillation and APB
**ECG and sport in Africa**

### African heart study

**Morocco 2002**

Heart and sport modifications of electrocardiogram, late Potentials and echocardiography about 75 sport’s men and 45 whiteness

**Algeria 2009**

Cardiac findings in the precompetition medical assessment of football players participating in the 2009 African Under-17 Championships in Algeria.

**Table 1**

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>TA (mmHg)</th>
<th>FC (beats/min)</th>
<th>PR (ms)</th>
<th>QTc (ms)</th>
<th>Leaf SL (mm)</th>
<th>Serum T (ng/dl)</th>
<th>Baso-décalage Segment ST (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>25.75</td>
<td>88.2</td>
<td>174.35</td>
<td>127.884</td>
<td>88.05</td>
<td>155.33</td>
<td>377.04</td>
<td>38.27</td>
<td>8.00</td>
<td>V2</td>
</tr>
<tr>
<td>p</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.005</td>
<td>V3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 2**

<table>
<thead>
<tr>
<th>Speed (km/h)</th>
<th>Systolic Blood Pressure (mmHg)</th>
<th>Diastolic Blood Pressure (mmHg)</th>
<th>Heart Rate (beats/min)</th>
<th>QTc (ms)</th>
<th>Baso-décalage Segment ST (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>49.37</td>
<td>113</td>
<td>83</td>
<td>143</td>
<td>49</td>
<td>20</td>
</tr>
<tr>
<td>p</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Tunisia 2007**

Analysis of the electrocardiogram and of the echocardiography of 181 footballers professionals tunsiens

**Gabon 2013**

Screening athletes for cardiovascular disease in Africa: a challenging experience.

**Table 3**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pelliccia [26]</th>
<th>Sharma [32]</th>
<th>Somoanu [34]</th>
<th>None study</th>
</tr>
</thead>
<tbody>
<tr>
<td>n = 1005</td>
<td>n = 1000</td>
<td>n = 171</td>
<td>n = 181</td>
<td></td>
</tr>
<tr>
<td>Aspect of hypertrophic ventricular gauche</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
</tr>
<tr>
<td>Hypertrophy auriculaire gauche</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Hypertrophy auriculaire droite</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Ondes T negatives</td>
<td>2.7</td>
<td>4</td>
<td>5.9</td>
<td>8.0</td>
</tr>
<tr>
<td>Ondes de ST</td>
<td>14.3</td>
<td>43</td>
<td>30.3</td>
<td>20.3</td>
</tr>
<tr>
<td>Ondes Qz 2 mm</td>
<td>8.0</td>
<td>5.9</td>
<td>5.9</td>
<td>5.9</td>
</tr>
<tr>
<td>Bloc de branche droit complet</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Bloc de branche droit incomplet</td>
<td>13.0</td>
<td>5.9</td>
<td>3.9</td>
<td>3.9</td>
</tr>
<tr>
<td>Bloc de branche gauche incomplet</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Bloc auriculo-ventriculaire du premier degré</td>
<td>7.5</td>
<td>5</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Bloc auriculo-ventriculaire du 2e degré Mobitz</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Bruckhart sepalostal de 3e degré</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Bruckhart sinistral de 60 battements/mm</td>
<td>36.4</td>
<td>80</td>
<td>30.9</td>
<td>40.9</td>
</tr>
<tr>
<td>Outre Delta</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>FR court sans onde delta</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

**Abstract**

Preparticipation cardiovascular (CV) screening has been advocated as an efficient strategy to reduce sudden cardiac death in Caucasian athletes. At present, uncertainty remains if such strategy is feasible and efficient in native African athletes. To this scope, we performed a CV screening in an African setting.

**Methods**: 210 male Gabonian football players were examined with history, physical examination, ECG and echocardiography.

**Results**: On history, 19 players (9%) referred atypical chest discomfort/appression. Familial sudden death was referred by 36 (17%). No anomalies were detected at physical examination. ECG showed large proportions of ‘training-related’ abnormalities, that is, ST-segment elevation in precordial leads in 150 (71.4%), and isolated increase in R/S-wave voltage in 116 (55.2%). A substantial subset (12.4%) showed ‘training-unrelated’ abnormalities, that is, inverted T-waves in 10 (4.8%), left atrial enlargement in 8 (4%), deep Q-waves in 3 (1.4%). On echocardiography, one athlete meet criteria for hypertrophic cardiomyopathy (HCM); none showed evidence for arrhythmogenic right ventricular cardiomyopathy (ARVC) or dilated cardiomyopathy (DCM). Other abnormalities included mitral valve prolapse in three, atrial septal defect in two and pulmonary hypertension in one.

**Conclusions**: About 12% of native African athletes showed ECG abnormalities unrelated to training and requiring additional testing and periodical follow-up. Structural abnormalities were found, however, in a minority (5%), including HCM in one, but no ARVC or DCM. In conclusion, this study demonstrates that preparticipation CV screening is efficient to identify (or raise suspicion) for CV abnormalities in native African athletes, but challenging for conclusive identification of cardiac diseases in the difficult scenario of a developing African country.

**my friend Dr Mouyopa did not have his name on this article**
Learn to work together?

ECG and echocardiographic criteria?

Definition of an athlete's heart: black and white?

Etiology of sudden death in Africa: climate, valve disease and infections +++

What prevention strategy in Africa?

Screening in schools +++++
Screening strategy

Simple strategy = best strategy

- Family history sudden death. Technical staff
- Syncope. Physical exam.
- ECG
Initiate own studies to us on a large scale African sports is a strategic emergency.
Only how we can better try to establish the boundaries between normality and pathology in our continent and to better understand the causes of sudden death for Prevention