Cardiac preparticipation screening for the young athlete: still a matter of controversy

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Regular physical activity is recommended as part of cardiovascular prevention and reduction of cardiovascular morbidity and mortality. However, vigorous exertion may increase the risk of sudden cardiac death in predisposed individuals: the risk of sudden cardiac death (SCD) is 2- to 3-fold higher in athletes compared to nonathletes.1

Most of these events in the young athletes less than 35 years are due to malignant tachyarrhythmias, usually ventricular fibrillation (VF) or ventricular tachycardia (VT) degenerating into VF, occurring in individuals with arrhythmogenic disorders (e.g. hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, channelopathies).2

Although the incidence of SCD is not accurately known, it is estimated in 0.5 and 1 per 100 000 below the age of 35 years. A comprehensive literature search review by Harmon et al found that this incidence is probably underestimated, which may imply the need of more effective prevention strategies.3 The causes of cardiac sudden death in the different series also depend on the characteristics of the population studied (age and geographical localization) (table 1).2

It is generally accepted that screening to detect potential diseases and prevent sudden death is justified and potentially beneficial. The strategies of screening are different across the countries. A medical detailed history (personal and family history to exclude genetic diseases) and a thorough physical examination are the basis of the evaluation for sports participation. Various protocols of preparticipation evaluation have been developed. In Portugal the questionnaire form can be assessed at the site of Instituto Português de Juventude e Desporto (Portuguese Institute of Sport and Youth); the Portuguese screening includes the history and physical examination, an ECG and a thorax X-ray.

The inclusion of the ECG to identify athletes at risk is still a matter of debate and controversy. The European Society of Cardiology recommends the addition of the ECG on the basis that medical history and physical examination alone have a limited ability to detect potentially lethal cardiac conditions, often silent. The ECG can manifest abnormalities in cardiomyopathies or channelopathies and contribute to the early recognition of these diseases.2,4,7

This argument is supported by a recent meta analysis of available studies comparing screening strategies (history, physical examination and ECG) that demonstrated the efficacy of the twelve lead ECG in the detection of cardiac disease.8 The use of modern criteria for interpretation of the ECG has reduced the number of false positives and the associated cost of further investigation (table 2).9-11

The ESC statement does not recommend the use of transthoracic echocardiography as a first line of screening or other imaging techniques. Exercise ECG test should be reserved for symptomatic athletes or those presenting high risk of CAD. The ambulatory ECG recording remains a second-line test. The most common indications for ambulatory ECG monitoring are unexplained syncope and palpitations, the investigation of bradyarrhythmias, to quantify premature ventricular contractions (PVC) density after initial preparticipation tests or to assess QT in patients with suspected LQTS.2

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Table 1 - Recent studies reporting causes of SCD in young athletes and their relative prevalence (Modified from Mont et al.)

<table>
<thead>
<tr>
<th>Study</th>
<th>Target population</th>
<th>Methodology</th>
<th>Site, year</th>
<th>SCD incidence</th>
<th>Causes</th>
</tr>
</thead>
</table>
| Corrado et al<sup>4</sup> | Young competitive athletes 12-35 years | Prospective declaration forms | Veneto Italy 1979-2004 | 1.9/100,000 person years | - Cardiomyopathies 25%  
- CAD 20%  
- Myocarditis 13%  
- Coronary congenital anomalies 30%  
- Mitral valve prolapse 11%  
- Conduction diseases 7% |
| Harmon et al<sup>5</sup> | NCAA athletes 17-24 years | Retrospective data collection from lists and registries owned by the NAAA and a non profit organization | USA, 2003-2013 | 1.9/100,000 person years | - Coronary congenital anomalies 11%  
- Myocarditis 9%  
- CAD 9%  
- HCM 8%  
- Idiopathic LVH 8%  
- Non specific cardiomyopathies 8%  
- ARVC 5%  
- Aortic dissection 5%  
- WPW 3%  
- DCM 3%  
- Kawasaki disease 2%  
- LQTS 1% |
| Maron et al<sup>6</sup> | Young competitive athletes <40 years | Prospectively and retrospectively collected for the US National Registry of Sudden Death through several sources | USA, 1990-2006 | 0.61/100,000 person-years (for the 2001-2006 period) | - HCM 36%  
- Coronary congenital anomalies 17%  
- Myocarditis 6%  
- ARVC 4%  
- Channelopathies 4% CAD 2%  
- MVP 2.5%  
- DCM 1.5%  
- Aortic stenosis 1.5% |

ARVC- arrhythmogenic right ventricular cardiomyopathy, CAD - coronary artery disease, ARVC- arrhythmogenic right ventricular cardiomyopathy, DCM - dilated cardiomyopathy, HCM - hypertrophic cardiomyopathy, LQTS - long QT syndrome, LVH - left ventricle hypertrophy, WPW- Wolf - Parkinson Syndrome.

Table 2 - Electrocardiographic parameters used to define various ECG abnormalities in the European Society of Cardiology recommendations, Seattle Criteria and Refined Criteria<sup>9</sup>

<table>
<thead>
<tr>
<th>ECG abnormality</th>
<th>European Society of Cardiology (ESC) recommendations</th>
<th>Seattle Criteria</th>
<th>Refined Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left atrial enlargement</td>
<td>Negative portion of the P-wave in lead V1 ≥0.1 mV in depth and ≥40 ms in duration</td>
<td>Prolonged P-wave duration of &gt;120 ms in leads I or II with negative portion of the P-wave ≥0.1 mV in depth and ≥40 ms in duration in lead V1</td>
<td>As ESC</td>
</tr>
<tr>
<td>Right atrial enlargement</td>
<td>P-wave amplitude ≥2.5 mV in leads II, III or aVF</td>
<td>As ESC</td>
<td>As ESC</td>
</tr>
<tr>
<td>Left QRS-axis deviation</td>
<td>≤−30° to −90°</td>
<td>As ESC</td>
<td>As ESC</td>
</tr>
<tr>
<td>Right QRS-axis deviation</td>
<td>&gt;115°</td>
<td>&gt;120°</td>
<td>As ESC</td>
</tr>
<tr>
<td>RV hypertrophy</td>
<td>Sum of R-wave in V1 and S-wave in V5 or V6 ≥1.05 mV</td>
<td>Sum of R-wave in V1 and S-wave in V5&gt;1.05 mV and right axis deviation &gt;120°</td>
<td>As Seattle</td>
</tr>
<tr>
<td>Corrected QT interval</td>
<td>QRS ≥120 ms predominantly negative QRS complex in lead V1 (Q5 or Q5), and upright monophasic R-wave in leads I and V6</td>
<td>QRS duration &gt;120 ms including RBBB and LBBB</td>
<td>As Seattle</td>
</tr>
<tr>
<td>Complete left bundle branch block</td>
<td>Any QRS duration ≥120 ms or complete LBBB</td>
<td>Any QRS duration ≥140 ms or complete LBBB</td>
<td>As Seattle</td>
</tr>
<tr>
<td>Complete right bundle branch block</td>
<td>Any QRS duration ≥120 ms or complete RBBB</td>
<td>As Seattle</td>
<td>As Seattle</td>
</tr>
<tr>
<td>Intraventricular conduction delay</td>
<td>Any RSR pattern in anterior precordial leads with QRS duration ≥120 ms</td>
<td>Any QRS duration ≥140 ms or complete LBBB</td>
<td>As Seattle</td>
</tr>
<tr>
<td>Pathological Q-wave</td>
<td>≥0.04 mV in any lead except III, aV</td>
<td>≥0.03 mV and/or ≥40 ms in duration or ≥25% of the height of the ensuing R-wave</td>
<td>As Seattle</td>
</tr>
<tr>
<td>Significant T-wave inversion</td>
<td>≥2 mm in ≥2 adjacent leads (deep or minor) in ≥2 leads</td>
<td>≥1 mm in depth in two or more leads V2–6, II and aVF and I or aVL (excludes III, aVR and V1)</td>
<td>As Seattle</td>
</tr>
<tr>
<td>ST-segment depression</td>
<td>≥0.5 mm in ≥2 leads</td>
<td>As Seattle</td>
<td>As Seattle</td>
</tr>
<tr>
<td>Ventricular pre-excitation</td>
<td>PR interval ≥120 ms with or without delta wave</td>
<td>PR interval ≥120 ms with or without delta wave</td>
<td>As Seattle</td>
</tr>
</tbody>
</table>

LBBB, left bundle branch block; mm, millimeters; ms, milliseconds; RBBB, right bundle branch block.
The recommendations of the American Heart Association (AHA) for preparticipation screening for cardiovascular disease includes a comprehensive personal/family history and physical examination, using the AHA’s 14-point screening guidelines or those of other societies, such as the American Academy of Pediatrics’ Preparticipation Physical Evaluation (the latter available at http://www.aap.org/PPE). Mandatory and universal mass screening with 12-lead ECGs in large general populations of young healthy people 12 to 25 years of age is not recommended for athletes and nonathletes. The reasons for this position are based mainly on the low incidence of SCD, the difficulties in the interpretation of the ECG and the false negative and false positive results, depending on the criteria used and in cost-efficacy considerations.

No screening program is able to prevent sudden cardiac arrest/death (SCA/SCD) completely. To improve outcomes other preventive measures should be taken, namely an emergency action plan (EAP) and access to an automated external defibrillator (AED). Personal in schools, clubs and sports organizations should have training in cardiopulmonary resuscitation.

In summary, cardiac preparticipation screening for the young athlete is still a matter of controversy. The goal of cardiovascular screening of athletes is to detect cardiac conditions predisposing to SCD. The addition of 12-lead ECG to the clinical history and physical examination demonstrates to have superior diagnostic capability than just clinical history and physical examination. The ECG should be interpreted with modern standards that distinguish normal findings related to physiological cardiac remodelling in trained athletes from abnormalities associated to cardiac disease.

However, no screening program provides absolute protection against sudden cardiac arrest/death (SCA/SCD); an emergency action plan and access to an automated external defibrillator are essential to improve outcomes from SCA in athletes.

REFERENCES


