International recommendations for electrocardiographic interpretation in athletes


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Received 17 May 2016; revised 11 July 2016; editorial decision 16 November 2016; accepted 8 December 2016

Sudden cardiac death (SCD) is the leading cause of mortality in athletes during sport. A variety of mostly hereditary, structural, or electrical cardiac disorders are associated with SCD in young athletes, the majority of which can be identified or suggested by abnormalities on a resting 12-lead electrocardiogram (ECG). Whether used for diagnostic or screening purposes, physicians responsible for the cardiovascular care of athletes should be knowledgeable and competent in ECG interpretation in athletes. However, in most countries a shortage of physician expertise limits wider application of the ECG in the care of the athlete. A critical need exists for physician education in modern ECG interpretation that distinguishes normal physiological adaptations in athletes from distinctly abnormal findings suggestive of underlying pathology. Since the original 2010 European Society of Cardiology recommendations for ECG interpretation in athletes, ECG standards have evolved quickly over the last decade; pushed by a growing body of scientific data that both tests proposed criteria sets and establishes new evidence to guide refinements. On 26–27 February 2015, an international group of experts in sports cardiology, inherited cardiac disease, and sports medicine convened in Seattle, Washington, to update contemporary standards for ECG interpretation in athletes. The objective of the meeting was to define and revise ECG interpretation standards based on new and emerging research and to develop a clear guide to the proper evaluation of ECG abnormalities in athletes. This statement represents an international consensus for ECG interpretation in athletes and provides expert opinion-based recommendations linking specific ECG abnormalities and the secondary evaluation for conditions associated with SCD.
Introduction
Cardiovascular-related sudden death is the leading cause of mortality in athletes during sport and exercise.1–3 The majority of disorders associated with an increased risk of sudden cardiac death (SCD) are suggested or identified by abnormalities on a resting 12-lead ECG. Whether used for the evaluation of cardiovascular-related symptoms, a family history of inheritable cardiac disease or premature SCD, or for screening of asymptomatic athletes, ECG interpretation is an essential skill for all physicians involved in the cardiovascular care of athletes.

The 2015 summit on ECG interpretation in athletes
Over the last decade, ECG interpretation standards have undergone several modifications to improve the accuracy of detecting potentially life threatening cardiac conditions in young athletes while also limiting false positive results.4–15 In February 2015, an international group of experts convened in Seattle, Washington, to update contemporary recommendations for ECG interpretation in asymptomatic athletes aged 12–35 years. The goals of the summit meeting were to: (i) update ECG interpretation standards based on new and emerging research and (ii) develop a clear guide to the appropriate evaluation of ECG abnormalities for conditions associated with SCD in athletes. In the presence of cardiac symptoms or a family history of inherited cardiovascular disease or premature SCD, a normal ECG should not preclude further assessment.

Limitations
While ECG increases the ability to detect underlying cardiovascular conditions associated with SCD, ECG as a diagnostic tool has limitations in both sensitivity and specificity. Specifically, ECG is unable to detect anomalous coronary arteries, premature coronary atherosclerosis, and aortopathies. In some instances patients with cardiomyopathies, particularly arrhythmogenic right ventricular cardiomyopathy (ARVC), may also reveal a normal ECG. Thus, an ECG will not detect all conditions predisposing to SCD. Furthermore, inter-observer variability among physicians remains a major concern,16–18 despite studies demonstrating that using standardized criteria improves interpretation accuracy.19,20

Normal ECG findings in athletes
Physiological cardiac adaptations to regular exercise
Regular and long-term participation in intensive exercise (minimum of 4h per week) is associated with unique electrical manifestations

Figure 1
International consensus standards for electrocardiographic interpretation in athletes. AV, atrioventricular block; LBBB, left bundle branch block; LVH, left ventricular hypertrophy; RBBB, right bundle branch block; RVH, right ventricular hypertrophy; PVC, premature ventricular contraction; SCD, sudden cardiac death.
**Table 1** International consensus standards for electrocardiographic interpretation in athletes: definitions of ECG criteria

<table>
<thead>
<tr>
<th>ECG abnormality</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal ECG findings in athletes</td>
<td>These ECG findings are unrelated to regular training or expected physiologic adaptation to exercise, may suggest the presence of pathologic cardiovascular disease, and require further diagnostic investigation.</td>
</tr>
<tr>
<td>T wave inversion</td>
<td>≥ 1 mm in depth in two or more contiguous leads; excludes leads aVR, III, and V1</td>
</tr>
<tr>
<td>• Anterior</td>
<td>V2–V4; includes: black athletes with J-point elevation and convex ST segment elevation followed by TWI in V2–V4; athletes &lt; age 16 with TWI in V1–V3; and biphase T waves in only V3</td>
</tr>
<tr>
<td>• Lateral</td>
<td>I and AVL, V5 and/or V6 (only one lead of TWI required in V5 or V6)</td>
</tr>
<tr>
<td>• Inferolateral</td>
<td>II and aVF, V5-V6, I and AVL</td>
</tr>
<tr>
<td>ST segment depression</td>
<td>≥ 0.5 mm in depth in two or more contiguous leads</td>
</tr>
<tr>
<td>Pathologic Q waves</td>
<td>Q/R ratio ≥ 0.25 or ≥ 40 ms in duration in two or more leads (excluding III and aVR)</td>
</tr>
<tr>
<td>Complete left bundle branch block</td>
<td>QRS ≥ 120 ms, predominantly negative QRS complex in lead V1 (QS or Rs), and upright notched or slurred R wave in leads I and V6</td>
</tr>
<tr>
<td>Profound nonspecific intra-ventricular conduction delay</td>
<td>Any QRS duration ≥ 140 ms</td>
</tr>
<tr>
<td>Epsilon wave</td>
<td>Distinct low amplitude signal (small positive deflection or notch) between the end of the QRS complex and onset of the T wave in leads V1-V3</td>
</tr>
<tr>
<td>Ventricular pre-excitation</td>
<td>PR interval &lt; 120 ms with a delta wave (slurred upstroke in the QRS complex) and wide QRS (≥ 120 ms)</td>
</tr>
<tr>
<td>Prolonged QT interval&lt;sup&gt;a&lt;/sup&gt;</td>
<td>QTc ≥ 470 ms (male)</td>
</tr>
<tr>
<td>Brugada Type 1 pattern</td>
<td>Coved pattern: initial ST elevation ≥ 2 mm (high take-off) with downsloping ST segment elevation followed by a negative symmetric T wave in ≥ 1 leads in V1–V3</td>
</tr>
<tr>
<td>Profound sinus bradycardia</td>
<td>&lt; 30 bpm or sinus pauses ≥ 3 sec</td>
</tr>
<tr>
<td>Profound 1° atrioventricular block</td>
<td>≥ 400 ms</td>
</tr>
<tr>
<td>Mobitz Type II 2° atrioventricular block</td>
<td>Intermittently non-conducted P waves with a fixed PR interval</td>
</tr>
<tr>
<td>3° atrioventricular block</td>
<td>Complete heart block</td>
</tr>
<tr>
<td>Atrial tachyarrhythmias</td>
<td>Supraventricular tachycardia, atrial fibrillation, atrial flutter</td>
</tr>
<tr>
<td>PVC</td>
<td>≥ 2 PVCs per 10 s tracing</td>
</tr>
<tr>
<td>Ventricular arrhythmias</td>
<td>Couplets, triplets, and non-sustained ventricular tachycardia</td>
</tr>
</tbody>
</table>

**Borderline ECG findings in athletes**
These ECG findings in isolation likely do not represent pathologic cardiovascular disease in athletes, but the presence of two or more borderline findings may warrant additional investigation until further data become available.

<table>
<thead>
<tr>
<th>ECG abnormality</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left axis deviation</td>
<td>-30° to - 90°</td>
</tr>
<tr>
<td>Left atrial enlargement</td>
<td>Prolonged P wave duration of &gt; 120 ms in leads I or II with negative portion of the P wave ≥ 1 mm in depth and ≥ 40 ms in duration in lead V1</td>
</tr>
<tr>
<td>Right axis deviation</td>
<td>&gt; 120°</td>
</tr>
<tr>
<td>Right atrial enlargement</td>
<td>P wave ≥ 2.5 mm in II, III, or aVF</td>
</tr>
<tr>
<td>Complete right bundle branch block</td>
<td>rSR pattern in lead V1 and a S wave wider than R wave in lead V6 with QRS duration ≥ 120 ms</td>
</tr>
</tbody>
</table>

**Normal ECG findings in athletes**
These training-related ECG alterations are physiologic adaptations to regular exercise, considered normal variants in athletes, and do not require further evaluation in asymptomatic athletes with no significant family history.

**Normal ECG finding**

<table>
<thead>
<tr>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased QRS voltage</td>
</tr>
<tr>
<td>Incomplete RBBB</td>
</tr>
</tbody>
</table>

Continued
Early repolarization

- J point elevation, ST elevation, J waves, or terminal QRS slurring in the inferior and/or lateral leads
- J-point elevation and convex (‘domed’) ST segment elevation followed by T wave inversion in leads V1–V4 in black athletes
- T-wave inversion V1–V3 in athletes < age 16
- ≥ 30 bpm
- Heart rate variation with respiration: rate increases during inspiration and decreases during expiration
- P waves are a different morphology compared with the sinus P wave, such as negative P waves in the inferior leads (‘low atrial rhythm’)
- QRS rate is faster than the resting P wave or sinus rate and typically less than 100 beats/minute with narrow QRS complex unless the baseline QRS is conducted with aberrancy
- PR interval 200–400 ms
- PR interval progressively lengthens until there is a non-conducted P wave with no QRS complex; the first PR interval after the dropped beat is shorter than the last conducted PR interval

athletes and 63–91% of black athletes of African-Caribbean descent (hereafter referred to as ‘black athletes’).

Some studies on survivors of cardiac arrest and patients with primary ventricular fibrillation (VF) have suggested an association between early repolarization and the risk of VF. Although further studies are warranted to further elucidate the mechanisms and prognostic implications of early repolarization in competitive athletes, to date there are no data to support an association between inferior early repolarization and SCD in athletes. Based on current evidence, all patterns of early repolarization, when present in isolation and without clinical markers of pathology, should be considered benign variants in athletes.

Repolarization findings in black athletes

Ethnicity is a major determinant of cardiac adaptation to exercise with more than two-thirds of black athletes exhibiting repolarization changes. Black athletes also commonly demonstrate a repolarization variant consisting of J-point elevation and convex ST segment elevation in the anterior leads (V1–V4) followed by TWI (Figure 3 and Figure 4B and C) which is regarded as a normal variant and should not result in further investigation, in the absence of other clinical or ECG features of cardiomyopathy.

Considerations in athletes age 12–16 years: the ‘juvenile’ electrocardiogram pattern

TWI confined to the anterior precordial leads may be considered a normal age-related pattern in adolescent athletes up to the age of 16 years old. The term ‘juvenile’ ECG pattern is used to denote TWI or a biphasic T wave beyond lead V2 in adolescents who have not reached physical maturity and is present in 10–15% of white, adolescent athletes aged 12 years old but only in 2.5% of white athletes aged 14–15 years (Figure 4A). Anterior TWI that extends beyond lead V2 is rare (0.1%) in white athletes aged ≥ 16 years or
### Table 2: Evaluation of electrocardiographic abnormalities

<table>
<thead>
<tr>
<th>ECG abnormality</th>
<th>Potential cardiac disease*</th>
<th>Recommended evaluationb</th>
<th>Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>T wave inversion in the lateral or inferolateral leads</td>
<td>HCM</td>
<td>Echocardiography</td>
<td>Lateral or inferolateral T wave inversion is common in primary myocardial disease. CMR should be a routine diagnostic test for this ECG phenotype and is superior to echocardiography for detecting apical HCM, LVH localized to the free lateral wall, ARVC with predominant left ventricular involvement, and myocarditis. If CMR is not available, echocardiography with contrast should be considered as an alternative investigation for apical HCM in patients with deep T wave inversion in leads V5–V6. Consider family evaluation if available and genetic screening. Annual follow-up testing is recommended throughout athletic career in athletes with normal results.</td>
</tr>
<tr>
<td>T wave inversion isolated to the inferior leads</td>
<td>HCM DCM LVNC Myocarditis</td>
<td>Echocardiography</td>
<td>Consider CMR based on echo findings or clinical suspicion.</td>
</tr>
<tr>
<td>T wave inversion in the anterior leads*</td>
<td>ARVC</td>
<td>Echocardiography</td>
<td>The extent of investigations may vary based on clinical suspicion for ARVC and results from initial testing.</td>
</tr>
<tr>
<td>ST segment depression</td>
<td>HCM DCM LVNC ARVC Myocarditis</td>
<td>Echocardiography</td>
<td>Consider CMR and additional testing based on echo findings or clinical suspicion.</td>
</tr>
<tr>
<td>Pathologic Q waves</td>
<td>HCM DCM LVNC Myocarditis</td>
<td>Echocardiography</td>
<td>Consider CMR (with perfusion study if available) based on echo findings or clinical suspicion. In the absence of CMR, consider exercise stress testing, dobutamine stress echocardiogram, or a myocardial perfusion scan for evaluation of coronary artery disease in athletes with suspicion of prior MI or multiple risk factors for CAD.</td>
</tr>
<tr>
<td>Complete left bundle branch block</td>
<td>DCM HCM LVNC Sarcoïdosis Myocarditis</td>
<td>Echocardiography</td>
<td>A comprehensive cardiac evaluation to rule out myocardial disease should be considered.</td>
</tr>
<tr>
<td>Profound nonspecific intraventricular conduction delay ≥ 140 ms</td>
<td>DCM HCM LVNC ARVC</td>
<td>Echocardiography</td>
<td>Consider additional testing based on echo findings or clinical suspicion.</td>
</tr>
<tr>
<td>Epsilon wave</td>
<td>ARVC</td>
<td>Echocardiography</td>
<td>An epsilon wave in leads V1-V3 is a highly specific ECG maker and a major diagnostic criterion for ARVC.</td>
</tr>
</tbody>
</table>

Continued
<table>
<thead>
<tr>
<th>ECG abnormality</th>
<th>Potential cardiac disease*</th>
<th>Recommended evaluation**</th>
<th>Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple premature ventricular contractions</td>
<td>HCM</td>
<td>Echocardiography</td>
<td>If &gt; 2000 PVC’s or non-sustained ventricular tachycardia are present on initial testing, comprehensive cardiac testing inclusive of CMR is warranted to investigate for myocardial disease. Consider signal averaged ECG (SAECG).</td>
</tr>
<tr>
<td></td>
<td>DCM</td>
<td>24 h ECG monitor</td>
<td>Abrupt cessation of the delta wave (pre-excitation) on exercise ECG denotes a low risk pathway. EP study for risk assessment should be considered if a low risk accessory pathway cannot be confirmed by non-invasive testing. Consider EP study for moderate to high intensity sports.</td>
</tr>
<tr>
<td></td>
<td>LVNC</td>
<td>Exercise ECG test</td>
<td>Acute ECG of 1st degree relatives if possible</td>
</tr>
<tr>
<td></td>
<td>ARVC</td>
<td>Echocardiography</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Myocarditis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sarcoidosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventricular pre-excitation</td>
<td>WPW</td>
<td>Exercise ECG test</td>
<td>Abrupt cessation of the delta wave (pre-excitation) on exercise ECG denotes a low risk pathway. EP study for risk assessment should be considered if a low risk accessory pathway cannot be confirmed by non-invasive testing. Consider EP study for moderate to high intensity sports.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Echocardiography</td>
<td></td>
</tr>
<tr>
<td>Prolonged QTc</td>
<td>LQTS</td>
<td>Repeat resting ECG on separate day</td>
<td>Consider exercise ECG test, laboratory (electrolyte) screening, family screening and genetic testing when clinical suspicion is high. Consider direct referral to a heart rhythm specialist or sports cardiologist for a QTc ≥ 500 ms.</td>
</tr>
<tr>
<td>Brugada Type 1 pattern</td>
<td>Brugada syndrome</td>
<td>Referral to cardiologist or heart rhythm specialist</td>
<td>Consider high precordial lead ECG with leads V1 and V2 in 2nd and 3rd intercostal space or sodium channel blockade if Brugada pattern is indeterminate. Consider genetic testing and family screening.</td>
</tr>
<tr>
<td>Profound sinus bradycardia &lt; 30 BPM</td>
<td>Myocardial or electrical disease</td>
<td>Repeat ECG after mild aerobic activity</td>
<td>Consider additional testing based on clinical suspicion.</td>
</tr>
<tr>
<td>Profound 1^st^ AV block ≥ 400 ms</td>
<td>Myocardial or electrical disease</td>
<td>Repeat ECG after mild aerobic activity</td>
<td>Consider additional testing based on clinical suspicion.</td>
</tr>
<tr>
<td>Advanced 2^nd^ or 3^rd^ atrioventricular block</td>
<td>Myocardial or electrical disease</td>
<td>Exercise ECG test</td>
<td></td>
</tr>
<tr>
<td>Atrial tachyarrhythmias</td>
<td>Myocardial or electrical disease</td>
<td>Echocardiography</td>
<td>Consider laboratory screening and CMR based on echo findings.</td>
</tr>
<tr>
<td>Minimum 24 h ECG monitor</td>
<td>Exercise ECG test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventricular arrhythmias^e</td>
<td>Myocardial or electrical disease</td>
<td>Echocardiography</td>
<td>A comprehensive cardiac evaluation to rule out myocardial disease and primary electrical disease should be considered.</td>
</tr>
<tr>
<td>Minimum 24 h ECG monitor</td>
<td>CMR</td>
<td>Exercise ECG test</td>
<td></td>
</tr>
<tr>
<td>Two or more borderline ECG findings</td>
<td>Myocardial disease</td>
<td>Echocardiography</td>
<td>Consider additional testing based on clinical suspicion.</td>
</tr>
</tbody>
</table>

ARVC, arrhythmogenic right ventricular cardiomyopathy; CAD, coronary artery disease; CMR, cardiovascular magnetic resonance; DCM, dilated cardiomyopathy; ECG, electrocardiogram; EP, electrophysiological; HCM, hypertrophic cardiomyopathy; LQTS, long QT syndrome; LVNC, left ventricular noncompaction; PVC, premature ventricular complex; SAECG, signal averaged ECG; WPW, Wolff-Parkinson-White syndrome.

*This list of disorders for each ECG abnormality represents the primary cardiac disorders of concern and is not intended to be exhaustive.

**Initial evaluation of ECG abnormalities should be performed under the direction of a cardiologist. Additional testing will be guided by initial findings and clinical suspicion based on the presence of symptoms or a family history of inherited cardiac disease or SCD.

^Excludes black athlete repolarization variant and juvenile pattern in adolescents < 16 years.

^CT coronary angiography if stress perfusion with CMR is unavailable.

^Includes couplets, triplets, accelerated ventricular rhythm, and non-sustained ventricular tachycardia.
Figure 2  Electrocardiogram of a 29-year-old male asymptomatic soccer player showing sinus bradycardia (44 bpm), early repolarization in I, II, aVF, V5–V6 (arrows), voltage criterion for left ventricular hypertrophy (S-V1 + R-V5 > 35 mm), and tall, peaked T waves (circles). These are common, training related findings in athletes and do not require more evaluation.

Figure 3  Electrocardiogram from a black athlete demonstrating voltage criterion for left ventricular hypertrophy, J point elevation and convex (‘domed’) ST segment elevation followed by T-wave inversion in V1–V4 (circles). This is a normal repolarization pattern in black athletes.
Based on current evidence, TWI in the anterior leads (V1–V3) in adolescent athletes < 16 years of age should not prompt further evaluation in the absence of symptoms, signs, or a family history of cardiac disease.

**Physiological arrhythmias in athletes**

Common consequences of increased vagal tone include sinus bradycardia and sinus arrhythmia. Other, less common markers of increased vagal tone are junctional or ectopic atrial rhythms, first degree atrioventricular (AV) block, and Mobitz Type I second degree AV block (Wenckebach phenomenon). In the absence of symptoms, heart rates ≥ 30 bpm are considered normal in highly trained athletes. Sinus rhythm should resume and bradycardia should resolve with the onset of physical activity.

**Borderline electrocardiogram findings in athletes**

Recent data suggest that some ECG findings previously categorized as abnormal may represent normal variants or the result of physiological cardiac remodelling in athletes and do not usually represent...
pathological cardiac disease. These ECG findings have been categorized as ‘borderline’ findings in athletes (Figure 1; Table 1).

**Axis deviation and voltage criteria for atrial enlargement**

Atrial deviation and voltage criteria for atrial enlargement account for >40% of abnormal ECG patterns in athletes but do not correlate with cardiac pathology. In a large study of 2533 athletes aged 14–35 years old and 9997 controls of similar age, echocardiographic evaluation of the 579 athletes and controls with isolated axis deviation or voltage criteria for atrial enlargement failed to identify any major structural or functional abnormalities.

**Complete right bundle branch block**

Although incomplete right bundle branch block (RBBB) is common in young athletes, the significance of complete RBBB is less certain. Complete RBBB is detected in approximately 1% of the general population and large datasets in young adult athletes reveal a prevalence of 0.5–2.5%. In a study of 510 U.S. collegiate athletes, RBBB was reported in 2.5% and compared with athletes with normal QRS complexes or incomplete RBBB, athletes with complete RBBB exhibited larger right ventricular dimensions and a lower right ventricular ejection fraction but preserved fractional area change. None of the athletes with complete RBBB or incomplete RBBB was found to have pathological structural cardiac disease. These patterns among trained athletes could represent a spectrum of structural and physiological cardiac remodelling characterized by RV dilation with resultant QRS prolongation and a relative reduction in the RV systolic function at rest.

Based on the aforementioned considerations, left axis deviation, left atrial enlargement, right axis deviation and right atrial enlargement and complete RBBB are considered borderline variants in athletes. The presence of any one of these findings in isolation or with other recognized physiological electrical patterns of athletic training does not warrant further assessment in asymptomatic athletes without a family history of premature cardiac disease or SCD. Conversely, the presence of more than one of these borderline findings places the athlete in the abnormal category warranting additional investigation.

**Abnormal electrocardiogram findings in athletes**

The abnormal findings defined in this section are not recognized features of athletic training and always require further assessment to exclude the presence of intrinsic cardiac disease (Figure 1; Tables 1 and 2). Temporary restriction from athletic activity should be considered for athletes with abnormal ECGs of uncertain clinical significance until secondary investigations are completed.

**Abnormal T-wave inversion**

T-wave ≥1 mm in depth in two or more contiguous leads (excluding leads aVR, III, and V1) in an anterior, lateral, inferolateral, or inferior territory is abnormal and should prompt further evaluation for underlying structural heart disease (Tables 1 and 2). Normal exceptions include T-wave confined to leads V1–V4 in black athletes when preceded by J point and/or ST segment elevation, and T waves in leads V1–V3 in athletes aged <16 years.

**Clinical considerations**

The relationship between abnormal T-wave inversion and several forms of structural heart disease is well documented. Twi in the inferior or lateral leads is common in HCM, whereas Twi in the right precordial leads (V1–V3) or beyond in the absence of a complete RBBB is common in ARVC (Figure 4D). There are no data relating to the significance of flat or biphasic T waves in athletes but similar to TWI, this panel would recommend further evaluation of biphasic T waves where the negative portion is ≥1 mm in depth in ≥2 leads.

**Evaluation**

Lateral or inferolateral T-wave inversion

There is mounting evidence that T-wave inversion in the lateral or inferolateral leads is associated with the presence of quiescent cardiomyopathy in a considerable proportion of athletes. Recommendations for the evaluation of abnormal TWI and other clinical considerations are presented in Table 2.

T-wave affecting the lateral leads (V5–V6, I and aVL) (Figure 4E) should prompt a comprehensive investigation to exclude cardiomyopathy. If echocardiography is not diagnostic, cardiac magnetic resonance imaging (MRI) with gadolinium should be utilized. Cardiac MRI provides superior assessment of myocardial hypertrophy, especially the left ventricular apex and the lateral free wall and may also demonstrate late gadolinium enhancement (LGE), a non-specific marker suggesting myocardial fibrosis. If cardiac MRI is not available, echocardiography with contrast should be considered. Exercise ECG testing and Holter monitoring also should be considered in the evaluation of lateral or inferolateral T-wave inversion, especially for athletes with ‘grey zone’ hypertrophy (males with maximal LV wall thickness 13–16 mm) without LGE, where the diagnosis of HCM remains uncertain. In such cases, the presence of ventricular tachycardia during exercise or Holter may support HCM and is also useful in risk stratification.

For athletes with lateral or inferolateral T-wave inversion, regular follow-up with serial cardiac imaging is necessary even when the initial evaluation is normal, in order to monitor for the development of a cardiomyopathy phenotype.

**Anterior T-wave inversion**

Anterior T-wave inversion is a normal variant in asymptomatic adolescent athletes age <16 years, in black athletes when preceded by J-point elevation and convex ST segment elevation, and in some endurance athletes. However, anterior T-wave inversion in leads V1–V2/V3 also is a recognized pattern in patients with ARVC and rarely HCM.

There are discrepancies among existing guidelines relating to the extent of anterior T-wave inversion before considering further investigations. A large study of over 14 000 white adults aged 16–35 years old, including over 2500 athletes showed that anterior T-wave had a prevalence of 2.3%. Anterior T-wave inversion was more common in females and athletes and was confined to leads V1–V2 in almost all individuals, and only exceeded beyond V2 in 1% of females and 0.2% of males.

None of the individuals with anterior T-wave inversion were diagnosed with a cardiomyopathy following comprehensive investigation indicating that this particular ECG pattern is non-specific in low-risk populations. Based on this report, it is justifiable to only investigate non-black athletes with anterior T-wave inversion in the absence of other clinical or electrical features of ARVC.

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**Tables and Figures**

- **Table 1**: Abnormal electrocardiogram findings in athletes
- **Table 2**: Clinical considerations
- **Figure 1**: Abnormal electrocardiogram findings in athletes
- **Figure 4D**: Complete right bundle branch block
- **Figure 4E**: Lateral or inferolateral T-wave inversion
Specific information about the J-point and preceding ST segment may help differentiate between physiological adaptation and cardiomyopathy in athletes with anterior TWI affecting leads V3 and/or V4. A recent study comparing anterior TWI in a series of black and white healthy athletes, and patients with HCM and ARVC, showed that in athletes with anterior TWI, the combination of J-point elevation $\geq$ 1 mm and TWI confined to leads V1–V4 excluded either cardiomyopathy with 100% negative predictive value, regardless of ethnicity.66 Conversely, anterior TWI associated with minimal or absent J-point elevation (< 1 mm) could reflect a cardiomyopathy.66 These data require duplication in larger studies but may prove useful in the assessment of a small proportion of white endurance athletes who exhibit anterior TWI and in athletes of black/mixed ethnicity.69

In most non-black athletes age $\geq$ 16 years, anterior TWI beyond lead V2 should prompt further evaluation given the potential overlap with ARVC. In these athletes, concurrent findings of J-point elevation, ST segment elevation, or biphasic T waves more likely represents athlete’s heart, while the absence of J-point elevation or a coexistent depressed ST segment is more concerning for ARVC (Figure 5).66 Other ECG findings suggestive of ARVC include low limb lead voltages, prolonged S wave upstroke, ventricular ectopy with LBBB morphology, and epsilon waves.61 A combination of tests is needed to diagnose ARVC including echocardiography, cardiac MRI, Holter monitoring, exercise ECG test, and signal averaged ECG.

**Figure 5** Examples of physiological (A) and pathological T-wave inversion (B). Panel A demonstrates T-wave inversion in V1–V4 preceded by J-point elevations and convex ‘domed’ ST segment elevation (green circles). This should not be confused with pathological T-wave inversion (Panel B) which demonstrates T-wave inversion in V1–V6 with absent J-point elevation and a downsloping ST segment (red circles).

In inferior leads, the significance of TWI confined to the inferior leads is unknown. However, this finding cannot be attributed to physiological remodeling and thus warrants further investigation with, at minimum, an echocardiogram. Cardiac MRI should be considered based on the echocardiographic findings or clinical suspicion.

**ST segment depression**

While ST segment depression is common among patients with cardiomyopathy, it is not a feature of athletic training.28,59,70,71 ST segment depression (relative to the isoelectric PR segment) in excess of 0.05 mV (0.5 mm) in two or more leads should be considered an abnormal finding requiring definitive evaluation for underlying structural heart disease.

**Evaluation**

Echocardiography is the minimum evaluation for athletes with ST segment depression to investigate for underlying cardiomyopathy. Cardiac MRI should be considered based on the echocardiographic findings or clinical suspicion.

**Pathological Q waves**

Several pathological disorders including HCM, ARVC, infiltrative myocardial diseases, accessory pathways and transmural myocardial
Physiological underpinnings of IVCD in athletes remain incompletely understood but likely include some combination of neurologically mediated conduction fibre slowing and increased myocardial mass. In patients with LVH, left ventricular mass seems to be closely related to QRS duration. 

While the exact cut-off to trigger more investigation in athletes with a nonspecific IVCD remains unclear, this panel recommends that marked nonspecific IVCD > 140 ms in athletes, regardless of QRS morphology, is abnormal and should prompt further evaluation.

**Evaluation**

In asymptomatic athletes with profound non-specific IVCD, an echocardiogram is recommended to evaluate for myocardial disease. Other cardio testing may be indicated depending on echocardiographic findings or clinical suspicion.

**Ventricular pre-excitation**

Ventricular pre-excitation occurs when an accessory pathway bypasses the AV node resulting in abnormal conduction to the ventricle (pre-excitation) with shortening of the PR interval and widening of the QRS. This is evident on the ECG as the Wolf–Parkinson–White (WPW) pattern defined as a PR interval < 120 ms, the presence of a delta wave (slurring of the initial QRS), and a QRS duration > 120 ms. The WPW pattern occurs in up to 1 in 250 athletes. 

The presence of an accessory pathway can predispose an athlete to sudden death because rapid conduction of atrial fibrillation across the accessory pathway can result in VF.

**Evaluation**

A short PR interval in isolation without a widened QRS or delta wave in an asymptomatic athlete should not be considered for further assessment. The WPW pattern warrants further assessment of the refractory period of the accessory pathway. Non-invasive risk stratification begins with an exercise stress test, where abrupt, complete loss of pre-excitation at higher heart rates suggests a low-risk accessory pathway. 

An echocardiogram also should be considered due to the association of WPW with Ebstein’s anomaly and cardiomyopathy. Intermittent pre-excitation during sinus rhythm on a resting ECG is also consistent with a low-risk pathway and may obviate the need for an exercise test. If non-invasive testing cannot confirm a low-risk pathway or is inconclusive, an electrophysiological study should be considered to determine the shortest pre-excited RR interval during atrial fibrillation. If the shortest pre-excited RR interval is < 250 ms (240 bpm), then the accessory pathway is deemed high risk and transcatheter ablation is recommended. Some physicians may choose to subject all competitive athletes involved in moderate or high-intensity sport to electrophysiological studies irrespective of the results of the exercise test or 24 h ECG on the premise that high catecholamine concentrations during very intensive exercise may modify the refractory period of an accessory pathway in a fashion that cannot be reproduced during laboratory tests.

**Prolonged QT interval**

Congenital Long QT syndrome (LQTS) is a potentially lethal, genetically mediated ventricular arrhythmia syndrome with the hallmark electrocardiographic feature of QT prolongation. LQTS is estimated to affect 1 in 2000 individuals, and this may be underestimated given the subpopulation of so-called ‘normal QT interval’ or ‘concealed’
LQTS. Autopsy negative sudden unexplained death represents 25–40% of sudden unexpected deaths in persons under age 40 years. In such cases, cardiac ion channelopathies have been implicated by post-mortem genetic testing as the probable cause in up to 25–40% of cases.

Calculating the corrected QT interval
Accurate measurement and manual confirmation of the computer derived QT interval corrected for heart rate (QTc) is critical as the accuracy of computer generated QTc values is about 90–95%. Studies have suggested the ability of cardiologists to accurately measure the QTc is suboptimal. However, accurate assessment of the QTc can be achieved by adhering to the following six principles:

1. Use Bazett’s heart rate correction formula (QTc = QT/√RR; note the RR interval is measured in seconds) as population-based QTc distributions most frequently use Bazett-derived QTc values.
2. Bazett’s formula underestimates the QTc at heart rates < 50 bpm, and overestimates the QTc at heart rates > 90 bpm. Accordingly, for a heart rate < 50 bpm, a repeat ECG after mild aerobic activity is recommended to achieve a heart rate closer to 60 bpm. For heart rates > 90 bpm, a repeat ECG after additional resting time may help achieve a lower heart rate.
3. If sinus arrhythmia is present with beat to beat variation in heart rate, an average QT interval and average RR interval should be used.
4. Leads II and V5 usually provide the best delineation of the T wave.
5. Low amplitude U waves, which are common in the anterior precordial leads, should not be included in the QT calculation. The ‘Teach-the-Tangent’ or ‘Avoid-the-Tail’ method to delineate the end of the T wave should be followed (Figure 6).
6. The morphology of the T wave, not just the length of the QT interval, also can suggest the presence of LQTS. For instance, a notched T wave in the lateral precordial leads where the amplitude of the second portion of the T wave following the notch is greater than the first portion of the T wave may represent LQT-2 even in the absence of overt QT prolongation.

The easiest and most efficient way to confirm the computer-derived QTc is to examine lead II and/or V5 and determine if the manually measured QT interval matches the computer’s QT measurement. If there is concordance within about 10 ms, one can trust that the computer can derive accurately an average RR interval and complete the Bazett’s calculation. If, however, the manually measured QT interval is > 10 ms different than the computer’s QT measurement, an average RR interval should be determined and the QTc recalculated using the Bazett’s formula.

Corrected QT cut-offs
Given the overlap between QTc distributions in population-derived cohorts of healthy individuals compared with patients with genetically confirmed LQTS, the QTc cut-off value compelling further evaluation must be chosen carefully to balance the frequency of abnormal results and the positive predictive value for LQTS.

Recent consensus statements on ECG interpretation in athletes have recommended that male athletes with a QTc ≥ 470 ms and female athletes with a QTc ≥ 480 ms undergo further evaluation for LQTS to better balance false positive and false negative findings. These cut-off values are around the 99th percentile and consistent with thresholds defined by the American Heart Association and American College of Cardiology.
American College of Cardiology. This consensus group also recommends QTc values of $\geq 470$ ms in males and $\geq 480$ ms in females to define the threshold of QT prolongation that warrants further assessment in asymptomatic athletes.

**Short QT Interval**

The precise cut-off and clinical significance of a short QT interval in athletes is unknown. Data from over 18,000 asymptomatic young British individuals found that the prevalence of a QTc $\leq 320$ ms is 0.1%, suggesting an abnormal cut-off value of $< 320$ ms is pragmatic. However, over a mean follow up period of 5.3 years, none of the individuals with a short QT $< 320$ ms experienced any adverse events, syncope, or sudden death. Based on the rarity of this finding and absence of data to suggest long-term morbidity in asymptomatic athletes, this panel recommends that a short QT interval only be investigated in the context of concerning clinical markers.

**Evaluation**

It is critical that an athlete with a single prolonged QTc reading not be obligated a diagnosis of LQTS, but rather that these cut-off values trigger the need for additional evaluation. The importance of additional evaluation but not a premature diagnosis of LQTS was demonstrated in a study of 2000 elite athletes in which 7 (0.4%) had a prolonged QTc (range 460–570 ms). A QTc of $< 500$ ms in the absence of symptoms or familial disease was unlikely to represent LQTS. In contrast, a QTc $> 500$ ms was highly suggestive of LQTS as all three athletes with a QTc value of $> 500$ ms exhibited one of paradoxical prolongation of the QTc during exercise, a confirmatory genetic mutation, or prolonged QTc in a first-degree relative.

A personal history of syncope or seizures and a family history of exertional syncope, ‘epilepsy’, postpartum-timed syncope/seizure, unexplained motor vehicle accidents, unexplained drowning, and premature, unexplained sudden death $< 50$ years of age should be reviewed. If the personal/family history is positive, the athlete should be referred to an electrophysiologist for further evaluation. If the personal/family history is negative, a repeat ECG should be obtained (ideally on a different day). If the follow-up ECG is below the QTc cut-off values, then no additional evaluation is needed and the athlete should be reassured.

If the repeat ECG still exceeds the QTc cut-off values, then a screening ECG of the athlete’s first degree relatives (parents and siblings) should be considered and the athlete should be referred to an electrophysiologist for the possibility of newly discovered LQTS. Reversible, extrinsic factors, such as electrolyte abnormalities (hypokalaemia) or the presence of QT prolonging medications, must also be evaluated. If an athlete’s ECG shows a QTc $> 500$ ms and no reversible causes are identified, then the athlete should be referred immediately to an electrophysiologist as the probability of LQTS and future adverse events has increased. The Schwartz-Moss scoring system, electrocardiographic features, stress ECG, provocative testing, and genetic testing may be needed to clarify the diagnosis and should be performed and interpreted by a cardiologist familiar with the disease.

**Brugada Type 1 pattern**

Brugada syndrome (BrS) is an inherited primary electrical disease which predisposes to ventricular tachyarrhythmias and sudden death during states of enhanced vagal tone. It is characterized by the distinctive Brugada ECG pattern which consists of a coved rSr’ pattern, ST-segment elevation $\geq 2$ mm, and inversion of the terminal portion of the T wave in leads V1, V2, and V3 (Figure 4F). Although three types were described, only the Type 1 Brugada pattern is now considered diagnostic.

The coved ST segment elevation in Type 1 Brugada pattern results in a broad r’ and should be distinguishable from the upsloping ST segment elevation of early repolarization in an athlete. In this regard, the ‘Corrado index’ measures the ST elevation at the start of the ST segment (STj) and 80 ms after the start of the ST segment (ST80). In Type 1 Brugada pattern, the downsloping ST segment will have a STj/ST80 ratio $> 1$, while the initial upsloping of the ST segment found in early repolarization patterns in an athlete will produce an STj/ST80 ratio $< 1$ (Figure 7).

**Evaluation**

The Type 1 Brugada ECG pattern should be investigated regardless of symptoms. If the pattern is unclear, confirm correct lead placement, repeat the ECG if necessary, and perform a high precordial lead ECG with V1 and V2 placed in the 2nd or 3rd intercostal space. If the Type 1 pattern is seen on a high-precordial lead ECG, then referral to an electrophysiologist is indicated. Consideration should be given to potential accentuating factors for a Brugada-like ECG pattern, such as hyperkalaemia, fever, medications with sodium ion channel blocking properties, and lead placement.

**Profound sinus bradycardia or first degree atrioventricular block**

Sinus bradycardia and moderate prolongation of the PR interval (200–399 ms) are recognized features of athletic conditioning. Although a resting heart rate $\leq 30$ bpm or a PR interval $\geq 400$ ms may be normal in a well-trained athlete, it should prompt further evaluation for cardiac conduction disease.

**Evaluation**

Evaluation of profound sinus bradycardia or a markedly increased PR interval should include assessing the chronotropic response to mild aerobic activity, such as running on the spot or climbing stairs. Exercise testing is useful in this situation to provide an objective measure of the PR interval and heart rate response to aerobic activity. If the heart rate increases appropriately and the PR interval normalizes, and the athlete is asymptomatic, no further testing is necessary. Conversely, further evaluation should be performed if the heart rate does not increase or the PR interval does not shorten appropriately on exertion, the athlete experiences pre-syncope/syncope, or in athletes with a family history of cardiac disease or sudden death. Depending on the clinical scenario, an echocardiogram or ambulatory ECG monitor may be indicated.

**High grade atrioventricular block**

Mobitz Type II second degree AV block and third degree (complete) AV block are abnormal findings in athletes. Complete heart block can be confused with AV dissociation without block; a situation where the junctional pacemaker is faster than the sinus node, leading to more QRS complexes than P waves. Intermittent ventricular capture
by sinus P waves (resulting in an irregular ventricular response) excludes complete AV block. AV dissociation without block is the expression of autonomic mismatch between AV and sinus nodal modulation, but is not pathological. Like all other functional disturbances, a small exercise load with repeat ECG recording will show resolution of the ECG findings in AV dissociation.

Evaluation
If Mobitz II AV block or complete AV block is detected, further evaluation includes an echocardiogram, ambulatory ECG monitor, and exercise ECG test. Based on these results, laboratory testing and cardiac MRI may be considered. Referral to an electrophysiologist is essential.

Multiple premature ventricular contractions
Multiple (> 2) premature ventricular contractions (PVCs) are uncommon and present in < 1% of 12-lead ECGs in athletes. Although multiple PVCs are usually benign, their presence may be the hallmark of underlying heart disease. PVCs originating from the right ventricular outflow tract (LBBB and inferior axis origin) are considered particularly benign when associated with a normal ECG, however this PVC morphology can also be present in patients with early ARVC particularly when the QRS exceeds 160 ms. Therefore, the finding of > 2 PVCs on an ECG should prompt more extensive evaluation to exclude underlying structural heart disease.

Evaluation
The extent of evaluation for > 2 PVCs is controversial and excluding pathology may be difficult. At a minimum, an ambulatory Holter monitor, echocardiogram, and exercise stress test should be performed. The availability of modern small, leadless ambulatory recorders allows for longer electrocardiographic monitoring, including during training and competition, to exclude complex ventricular arrhythmias. If the Holter and echocardiogram are normal and the PVCs suppress with exercise, no further evaluation is recommended for an asymptomatic athlete. A previous study has shown that among athletes with > 2000 PVCs per 24 h, up to 30% were found to have underlying structural heart disease, compared with 3% and 0% in those with < 2000 and < 100 PVCs per day, respectively. Therefore, in athletes with > 2000 PVCs per 24 h or with episodes of non-sustained ventricular tachycardia, or with an increasing burden of ectopy during an incremental exercise test, additional evaluation may include contrast-enhanced cardiac MRI and more invasive electrophysiology study. Although some studies have suggested that regression of the PVC burden with detraining indicates a good prognosis, other studies have not confirmed this. Thus, detraining as a diagnostic or therapeutic measure is not recommended.

Atrial tachyarrhythmias
Sinus tachycardia is the most common atrial tachyarrhythmia but is very rarely due to intrinsic cardiac disease. Supraventricular tachycardia (SVT), atrial fibrillation, and atrial flutter are rarely seen on a resting ECG in athletes and require investigation. Atrial tachyarrhythmias are rarely life threatening but can be associated with other conditions that can lead to SCD, including LQTS, WPW, BrS, myocarditis, congenital heart disease, and the cardiomyopathies.

Evaluation
If resting sinus tachycardia > 120 bpm is seen, a repeat ECG should be considered after a period of rest as recent exercise or anxiety may be the cause. Other underlying aetiologies may be sought, including fever, infection, dehydration, stimulant use, anaemia, hyperthyroidism, or, rarely, underlying cardiac or pulmonary disease. For paroxysmal SVT, a repeat ECG when not in SVT should be obtained if possible. If the Valsalva maneuver, carotid sinus massage, or the diving reflex is used to terminate the arrhythmia, a rhythm strip should be obtained which can help elucidate the mechanism of the SVT. An echocardiogram, ambulatory ECG monitor, and exercise treadmill...
test should be completed. Referral to an electrophysiologist may be indicated for consideration of electrophysiology study and ablation. If atrial fibrillation or flutter is found, an echocardiogram should be completed to assess for structural heart disease and anti-coagulation considered based on standard guidelines. An ambulatory ECG monitor should be used to assess if the rhythm is paroxysmal or persistent and what the ventricular rate is throughout the day. A thorough family history may elucidate an underlying genetic cause. Depending on what these results show, cardiac MRI, electrophysiology study with possible ablation, and/or genetic testing may be considered.

**Ventricular arrhythmias**

Ventricular couplets, triplets, and non-sustained ventricular tachycardia always require investigation as they can be a marker for underlying cardiac pathology or lead to sustained ventricular tachycardia which may cause SCD.

**Evaluation**

If ventricular arrhythmias are seen, the evaluation should include a thorough family history, an echocardiogram to evaluate for structural heart disease, cardiac MRI to assess for ARVC or other cardiomyopathies, ambulatory ECG monitor and exercise ECG test. Depending on these results, further evaluation may be needed including electrophysiology study or genetic testing.

**Considerations in athletes ≥ 30 years of age**

In athletes ≥ 30 years of age, CAD is the most common cause of SCD. In addition, older athletes may be less fit compared with 20–30 years ago, increasing the possibility of underlying CAD. While resting ECGs have a low sensitivity for CAD, some ECG patterns may suggest underlying CAD such as TWI, pathological Q waves, ST segment depression, left or RBBB, abnormal R wave progression, left anterior hemiblock, and atrial fibrillation.

**Evaluation**

The main role of a resting ECG in older athletes is to identify those athletes who may potentially be at high risk for CAD and warrant further testing. Initial testing should include an exercise stress test, resting echocardiogram, and assessment of traditional risk factors for CAD. When indicated, this evaluation may be complemented by coronary CT angiography or a functional stress test.

**Electrocardiogram patterns requiring serial evaluation**

 Several common heritable cardiomyopathies may present with ECG abnormalities prior to the onset of overt heart muscle pathology. Therefore, athletes with abnormal ECGs suggestive of cardiomyopathy and initially normal clinical evaluations should be followed with serial evaluation during and after their competitive athletic careers. Evaluations may be conducted annually or more frequently depending on individual circumstances. These athletes may be permitted to participate in competitive athletics without restriction contingent on longitudinal follow-up.

**Conclusion**

Accurate ECG interpretation in athletes requires adequate training and an attention to detail to distinguish physiological ECG findings from abnormal ECG findings that might indicate the presence of cardiac pathology. The international consensus standards presented on ECG interpretation and the evaluation of ECG abnormalities serve as an important foundation for improving the quality of cardiovascular care of athletes. As new scientific data become available, revision of these recommendations may be necessary to further advance the accuracy of ECG interpretation in the athletic population.

**Supplementary material**

Supplementary material is available at European Heart Journal online.

**Acknowledgements**

The 2nd Summit on Electrocardiogram Interpretation in Athletes was sponsored by the American Medical Society for Sports Medicine (AMSSM), the FIFA Medical Assessment and Research Center (F-MARC), and the National Collegiate Athletic Association (NCAA). Participating medical societies included the American College of Cardiology (ACC) Sports & Exercise Council, and 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). This statement has been endorsed by the following societies: American Medical Society for Sports Medicine (AMSSM), Austrian Society of Sports Medicine and Prevention, Brazilian Society of Cardiology - Department of Exercise and Rehabilitation (SBC - DERCE), British Association for Sports and Exercise Medicine (BASEM), Canadian Academy of Sport and Exercise Medicine (CASEM), European College of Sports and Exercise Physicians (ECOSEP), European Society of Cardiology (ESC) Section of Sports Cardiology, Fédération Internationale de Football Association (FIFA), German Society of Sports Medicine and Prevention, International Olympic Committee (IOC), Norwegian Association of Sports Medicine and Physical Activity (NIMF), South African Sports Medicine Association (SASMA), Spanish Society of Cardiology (SEC) Sports Cardiology Group, Sports Doctors Australia, and the Swedish Society of Exercise and Sports Medicine (SFAIM). The American College of Cardiology (ACC) affirms the value of this document (ACC supports the general principles in the document and believes it is of general benefit to its membership).

**Conflict of interest:** S.S. reports grants from Cardiac Risk in the Young, grants from British Heart Foundation, outside the submitted work. M.P. reports grants from Cardiac Risk in the Young outside the submitted work. M.J.A. reports personal fees from Boston Scientific, personal fees from Gilead Sciences, personal fees from Invitae, personal fees from Medtronic, personal fees from St. Jude Medical, other from Transgenomic, outside the submitted work. In addition, M.J.A. has a patent QT and T Wave Analytics pending. V.F.F. reports other from Insightinc, from null, outside the submitted work; this is an entity that manufactures and develops ECG software and devices for screening purposes. H.H. reports other from Biotronik, during the conduct of the study; other from Biotronik, personal fees from Biotronik, personal fees from Pfizer/BMS, personal fees from Daichi-
Sankyo, personal fees from Bayer, personal fees from Boehringer-Ingelheim, personal fees from Cardiome, submitted the worked out.

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