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CURRENT OPINION

International recommendations for electrocardiographic interpretation in athletes

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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.

The opinions expressed in this article are not necessarily those of the Editors of the *European Heart Journal* or of the European Society of Cardiology.

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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.

This document provides the most updated evidence-based recommendations developed with thoughtful attention to balance sensitivity and specificity, while maintaining a clear and practical checklist of findings to guide ECG interpretation for physicians and the appropriate evaluation of ECG abnormalities. A summary of the consensus recommendations is presented in *Figure 1*, *Tables 1 and 2*.

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 4 h per week) is associated with unique electrical manifestations

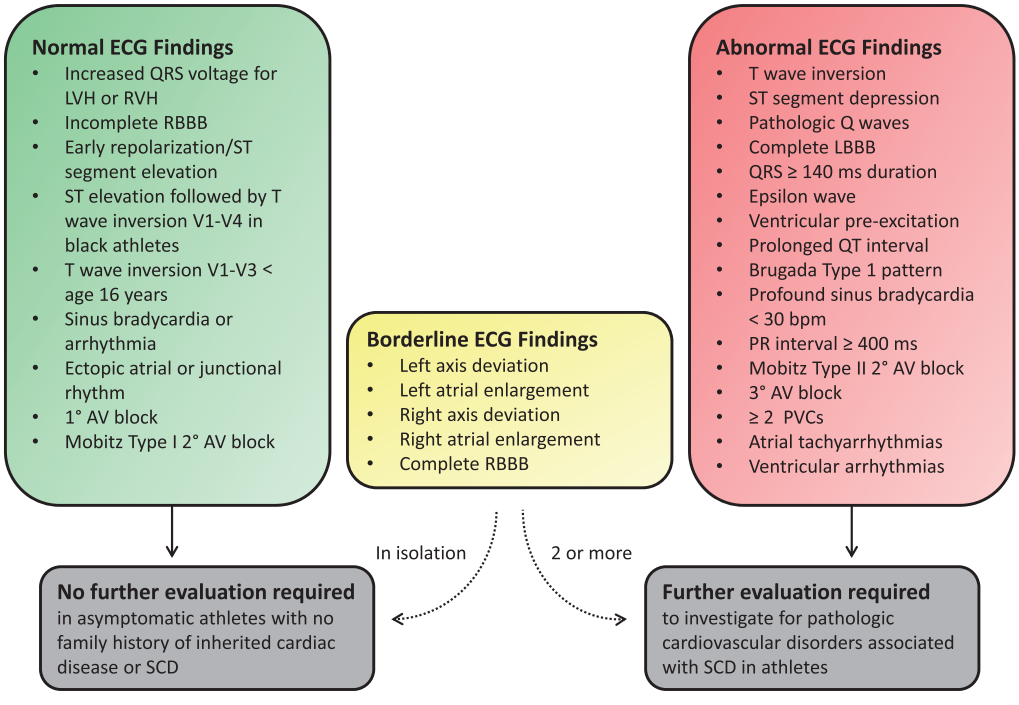

Figure 1 International consensus standards for electrocardiographic interpretation in athletes. AV, atrioventriular block; LBBB, left bundle branch block; LVH, left ventricular hypertrophy; RBBB, right bundle branch block; RVH, right ventricular hypertrophy; PVC, premature ventricular contraction; SCD, sudden cardiac death.

Table 1 International consensus standards for electrocardiographic interpretation in athletes: definitions of ECG criteria

Abnormal ECG findings in athletes

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.

ECG abnormality	Definition
T wave inversion	≥ 1 mm in depth in two or more contiguous leads; excludes leads aVR, III, and V1
• Anterior	• V2–V4
	– excludes: black athletes with J-point elevation and convex ST segment elevation followed by TWI in V2–V4; athletes < age 16 with TWI in V1-V3; and biphasic T waves in only V3
• Lateral	• I and AVL, V5 and/or V6 (only one lead of TWI required in V5 or V6)
• Inferolateral	• II and aVF, V5-V6, I and AVL
• Inferior	• II and aVF
ST segment depression	≥ 0.5 mm in depth in two or more contiguous leads
Pathologic Q waves	Q/R ratio ≥ 0.25 or ≥ 40 ms in duration in two or more leads (excluding III and aVR)
Complete left bundle branch block	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
Profound nonspecific intra-ventricular conduction delay	Any QRS duration ≥ 140 ms
Epsilon wave	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
Ventricular pre-excitation	PR interval < 120 ms with a delta wave (slurred upstroke in the QRS complex) and wide QRS (≥ 120 ms)
Prolonged QT interval[a]	QTc ≥ 470 ms (male) QTc ≥ 480 ms (female) QTc ≥ 500 ms (marked QT prolongation)
Brugada Type 1 pattern	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
Profound sinus bradycardia	< 30 bpm or sinus pauses ≥ 3 sec
Profound 1° atrioventricular block	≥ 400 ms
Mobitz Type II 2° atrioventricular block	Intermittently non-conducted P waves with a fixed PR interval
3° atrioventricular block	Complete heart block
Atrial tachyarrhythmias	Supraventricular tachycardia, atrial fibrillation, atrial flutter
PVC	≥ 2 PVCs per 10 s tracing
Ventricular arrhythmias	Couplets, triplets, and non-sustained ventricular tachycardia

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.

ECG abnormality	Definition
Left axis deviation	-30° to - 90°
Left atrial enlargement	Prolonged P wave duration of > 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
Right axis deviation	> 120°
Right atrial enlargement	P wave ≥ 2.5 mm in II, III, or aVF
Complete right bundle branch block	rSR′ pattern in lead V1 and a S wave wider than R wave in lead V6 with QRS duration ≥ 120 ms

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	Definition
Increased QRS voltage	Isolated QRS voltage criteria for left (SV1 + RV5 or RV6 > 3.5 mV) or right ventricular hypertrophy (RV1 + SV5 or SV6 > 1.1 mV)
Incomplete RBBB	rSR′ pattern in lead V1 and a qRS pattern in lead V6 with QRS duration < 120 ms

Continued

Early repolarization	J point elevation, ST elevation, J waves, or terminal QRS slurring in the inferior and/or lateral leads
Black athlete repolarization variant	J-point elevation and convex ('domed') ST segment elevation followed by T wave inversion in leads V1-V4 in black athletes
Juvenile T wave pattern	T-wave inversion V1–V3 in athletes < age 16
Sinus bradycardia	≥ 30 bpm
Sinus arrhythmia	Heart rate variation with respiration: rate increases during inspiration and decreases during expiration
Ectopic atrial rhythm	P waves are a different morphology compared with the sinus P wave, such as negative P waves in the inferior leads ('low atrial rhythm')
Junctional escape 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
1° atrioventricular block	PR interval 200–400 ms
Mobitz Type I 2° atrioventricular block	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

ECG, electrocardiogram; PVC, premature ventricular contraction; RBBB, right bundle branch block.
[a]The QT interval corrected for heart rate is ideally measured using Bazett's formula with heart rates between 60 and 90 bpm; preferably performed manually in lead II or V5 using the teach-the-tangent method[1] to avoid inclusion of a U wave (please see text for more details). Consider repeating the ECG after mild aerobic activity for a heart rate-< 50 bpm, or repeating the ECG after a longer resting period for a heart rate > 100 bpm, if the QTc value is borderline or abnormal.

that reflect enlarged cardiac chamber size and increased vagal tone. These ECG findings in athletes are considered normal, physiological adaptations to regular exercise and do not require further evaluation (*Figure 1*; *Table 1*).

Left and right ventricular hypertrophy

The presence of isolated QRS voltage criterion for left ventricular hypertrophy (LVH) (*Figure 2*) does not correlate with pathology in athletes and is present in isolation (without other associated ECG abnormalities) in less than 2% of patients with hypertrophic cardiomyopathy (HCM).[21–27] Conversely, pathological LVH is commonly associated with additional ECG features such as T-wave inversion (TWI) in the inferior and lateral leads, ST segment depression, and pathological Q waves.[28,29] Therefore, the isolated presence of high QRS voltages fulfilling voltage criterion for LVH in the absence of other ECG or clinical markers suggestive of pathology are considered part of normal and training-related ECG changes in athletes and does not require further evaluation.

Voltage criterion for right ventricular hypertrophy (RVH) is also common in athletes with up to 13% of athletes fulfilling the Sokolow–Lyon index.[30,31] QRS voltages for RVH, when present in isolation, do not correlate with underlying pathology in athletes.[31] Similar to voltage criteria for LVH, isolated QRS voltage for RVH is part of the normal spectrum of ECG findings in athletes and does not require further evaluation.

Early repolarization

Early repolarization is defined as elevation of the QRS-ST junction (J-point) by ≥ 0.1 mV often associated with a late QRS slurring or notching (J wave) affecting the inferior and/or lateral leads.[32–34] Early repolarization is common in healthy populations (2–44%) and is more prevalent in athletes, young individuals, males, and black ethnicity.[32,35–39] Early repolarization consisting of J-point elevation with concave ST-segment elevation and a peaked TWI (*Figure 2*) is present in up to 45% of Caucasian athletes and 63–91% of black athletes of African-Caribbean descent (hereto referred to as 'black' athletes).[22,30]

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.[33,40] Although further studies are warranted to fully 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.[41]

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.[29,30,42,43] 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.[30,42–44]

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*).[22,45,46] Anterior TWI that extends beyond lead V2 is rare (0.1%) in white athletes aged ≥ 16 years or

Table 2 Evaluation of electrocardiographic abnormalities

ECG abnormality	Potential cardiac disease[a]	Recommended evaluation[b]	Considerations
T wave inversion in the lateral or inferolateral leads	HCM DCM LVNC ARVC (with predominant LV involvement) Myocarditis	Echocardiography CMR Exercise ECG test Minimum 24 h ECG monitor	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.
T wave inversion isolated to the inferior leads	HCM DCM LVNC Myocarditis	Echocardiography	Consider CMR based on echo findings or clinical suspicion.
T wave inversion in the anterior leads[c]	ARVC DCM	Echocardiography CMR Exercise ECG test Minimum 24 h ECG monitor SAECG	The extent of investigations may vary based on clinical suspicion for ARVC and results from initial testing.
ST segment depression	HCM DCM LVNC ARVC Myocarditis	Echocardiography	Consider CMR and additional testing based on echo findings or clinical suspicion.
Pathologic Q waves	HCM DCM LVNC Myocarditis Prior MI	Echocardiography CAD risk factor assessment Repeat ECG for septal (V1–V2) QS pattern; above investigations recommended if septal Q waves are persistent	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.
Complete left bundle branch block	DCM HCM LVNC Sarcoidosis Myocarditis	Echocardiography CMR (with stress perfusion study)[d]	A comprehensive cardiac evaluation to rule out myocardial disease should be considered.
Profound nonspecific intraventricular conduction delay ≥ 140 ms	DCM HCM LVNC	Echocardiography	Consider additional testing based on echo findings or clinical suspicion.
Epsilon wave	ARVC	Echocardiography CMR Exercise ECG test Minimum 24 h ECG monitor SAECG	An epsilon wave in leads V1-V3 is a highly specific ECG maker and a major diagnostic criterion for ARVC.

Continued

Table 2 *Continued*

ECG abnormality	Potential cardiac disease[a]	Recommended evaluation[b]	Considerations
Multiple premature ventricular contractions	HCM DCM LVNC ARVC Myocarditis Sarcoidosis	Echocardiography 24 h ECG monitor Exercise ECG test	If > 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).
Ventricular pre-excitation	WPW	Exercise ECG test Echocardiography	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.
Prolonged QTc	LQTS	Repeat resting ECG on separate day Review for QT prolonging medication Acquire ECG of 1st degree relatives if possible	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.
Brugada Type 1 pattern	Brugada syndrome	Referral to cardiologist or heart rhythm specialist	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.
Profound sinus bradycardia < 30 BPM	Myocardial or electrical disease	Repeat ECG after mild aerobic activity	Consider additional testing based on clinical suspicion.
Profound 1° AV block ≥ 400 ms	Myocardial or electrical disease	Repeat ECG after mild aerobic activity Exercise ECG test	Consider additional testing based on clinical suspicion.
Advanced 2° or 3° atrioventricular block	Myocardial or electrical disease	Echocardiography Minimum 24 h ECG monitor Exercise ECG test	Consider laboratory screening and CMR based on echo findings.
Atrial tachyarrhythmias	Myocardial or electrical disease	Echocardiography Minimum 24 h ECG monitor Exercise ECG test	Consider CMR or EP study based on clinical suspicion.
Ventricular arrhythmias[e]	Myocardial or electrical disease	Echocardiography CMR Minimum 24 h ECG monitor Exercise ECG test	A comprehensive cardiac evaluation to rule out myocardial disease and primary electrical disease should be considered.
Two or more borderline ECG findings	Myocardial disease	Echocardiography	Consider additional testing based on clinical suspicion.

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

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

[b]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.

[c]Excludes black athlete repolarization variant and juvenile pattern in adolescents < 16 years.

[d]CT coronary angiography if stress perfusion with CMR is unavailable.

[e]Includes couplets, triplets, accelerated ventricular rhythm, and non-sustained ventricular tachycardia.

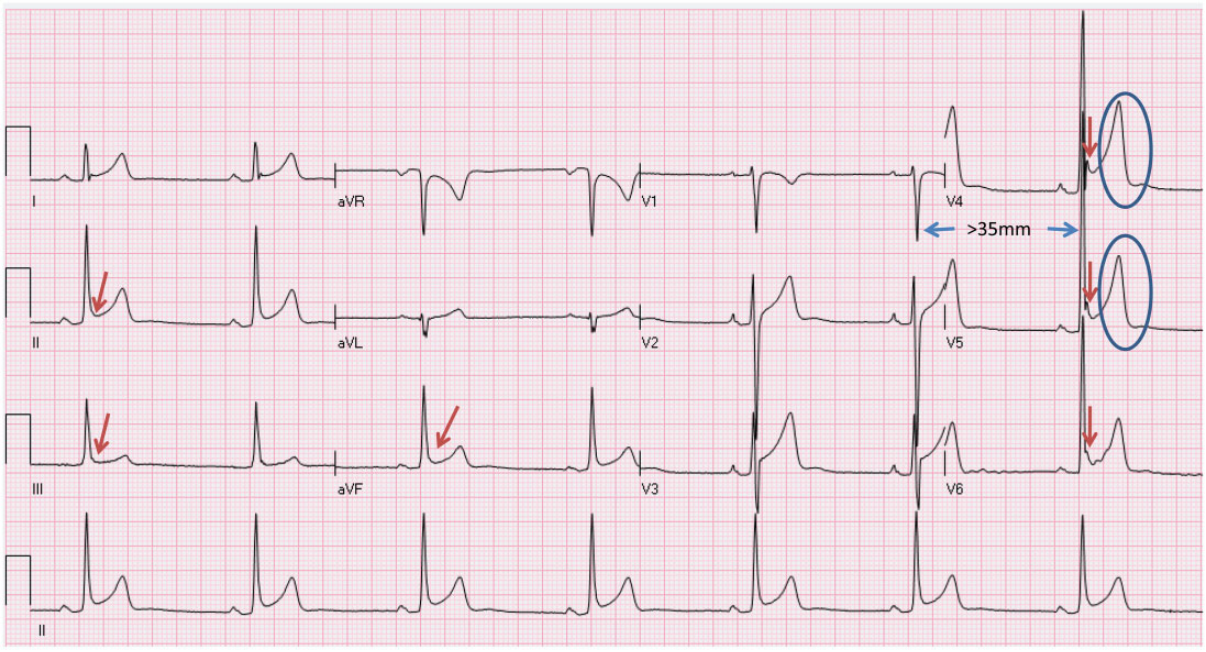

Figure 2 Electrocardiogram of a 29-year-old male asymptomatic soccer player showing sinus bradycardia (44 bpm), early repolarization in I, II, aVF, V5–V6 (arrows), voltage criterion for left ventricular hypertrophy (S-V1 + R-V5 > 35 mm), and tall, peaked T waves (circles). These are common, training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

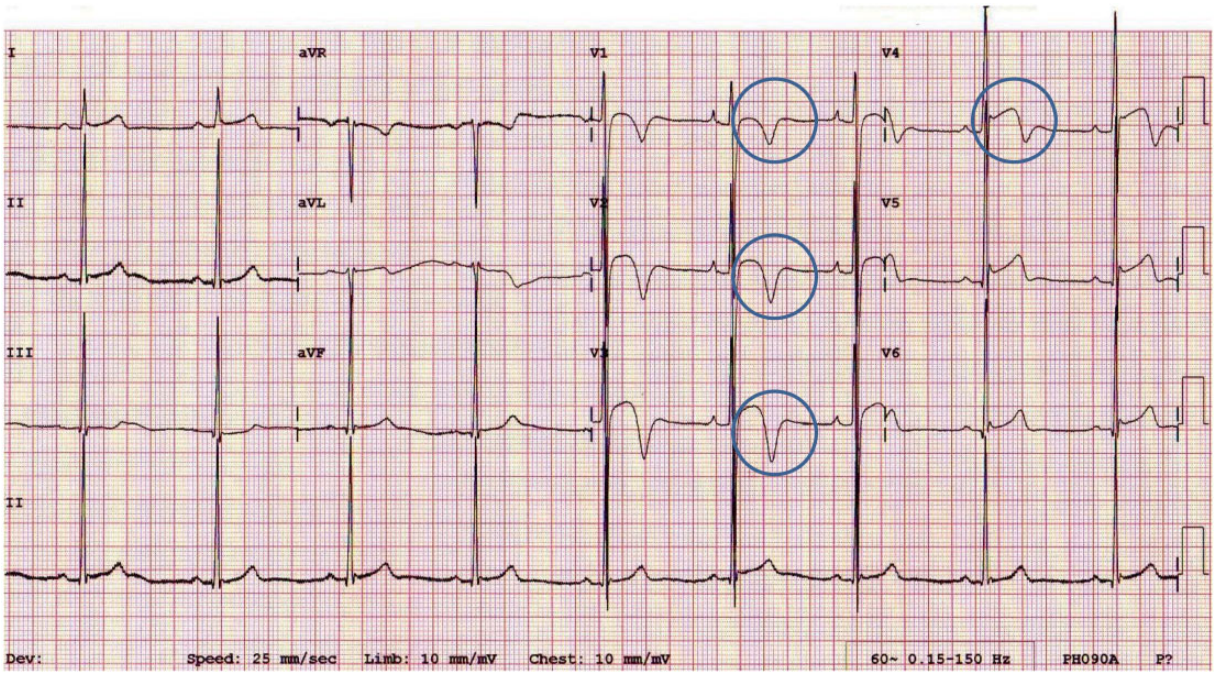

Figure 3 Electrocardiogram from a black athlete demonstrating voltage criterion for left ventricular hypertrophy, J point elevation and convex ('domed') ST segment elevation followed by T-wave inversion in V1–V4 (circles). This is a normal repolarization pattern in black athletes.

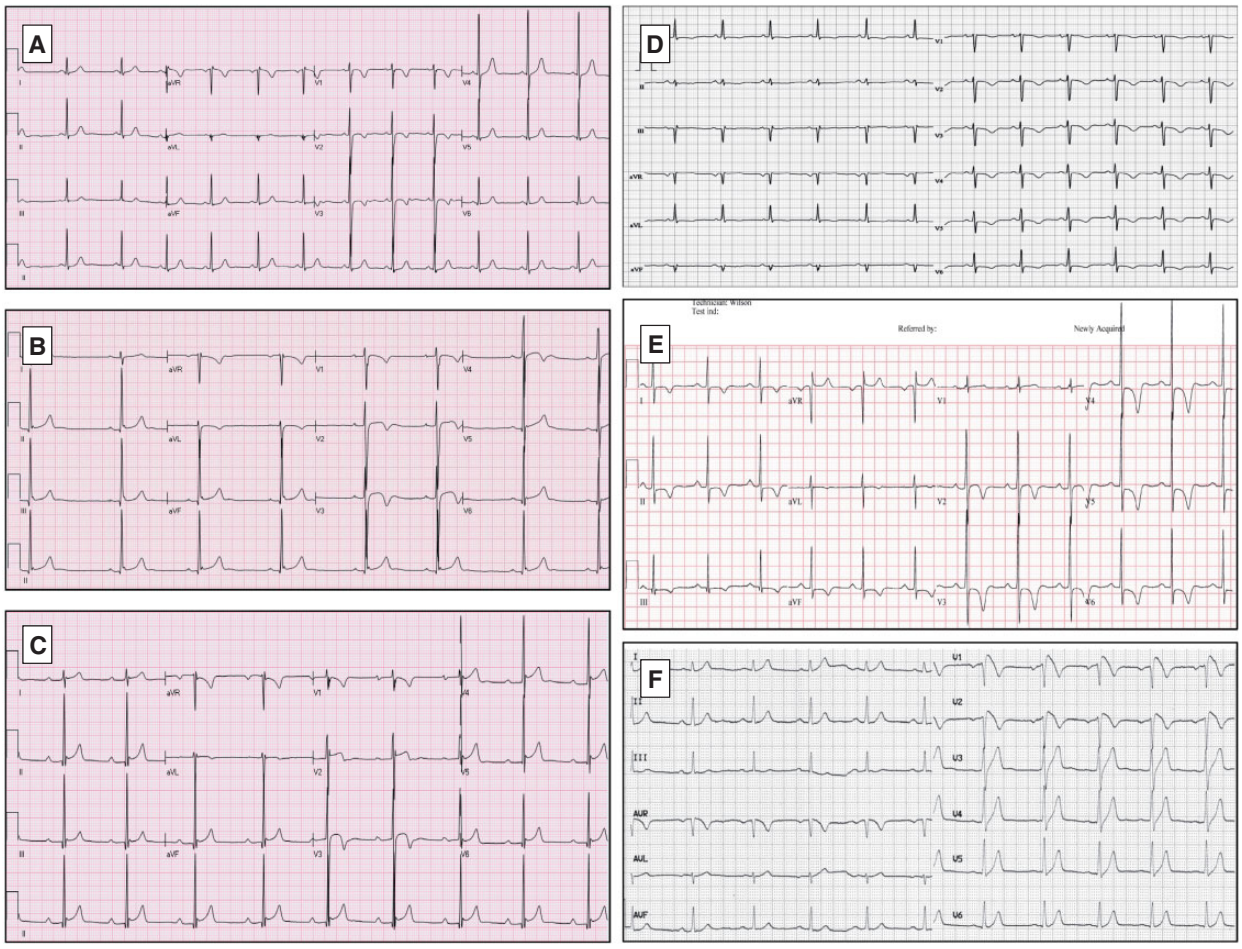

Figure 4 Normal and abnormal patterns of T-wave inversion. (*A*) Anterior T-wave inversion in V1–V3 in a 12-year-old asymptomatic athlete without a family history of sudden cardiac death considered a normal 'juvenile' pattern. (*B*) T-wave inversion in V1–V4 in a 17-year-old asymptomatic mixed race (Middle-Eastern/black) athlete without a family history of sudden cardiac death. This is a normal repolarization pattern in black athletes. (*C*) Biphasic T-wave inversion in V3 in a 31-year-old asymptomatic black athlete without a family history of sudden cardiac death. Anterior biphasic T waves are considered normal in adolescents < 16 years old and in adults when found in a single lead, most commonly V3. (*D*) Abnormal T-wave inversion in V1–V6 in an adult symptomatic former soccer player with genetically confirmed arrhythmogenic right ventricular cardiomyopathy and a positive family history of sudden cardiac death (brother died at 26 years of age). (*E*) Inferolateral T-wave inversion in leads I, II, III, aVF, V2–V6, and ST segment depression in leads II, aVF, V4–V6 in a 31-year-old asymptomatic professional soccer referee. These markedly abnormal findings require a comprehensive evaluation to exclude cardiomyopathy. (*F*) An electrocardiogram demonstrating the Type 1 Brugada pattern with high take-off ST elevation ≥ 2 mm with downsloping ST segment elevation followed by a negative symmetric T wave in V1–V2.

younger athletes who have completed puberty.[22,45] 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.[22,47–49] 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).[22,47,50] 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

Axis deviation and voltage criteria for atrial enlargement account for >40% of abnormal ECG patterns in athletes but do not correlate with cardiac pathology.[51] 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.[51]

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%.[12,52–54] 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.[55] 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.[55]

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

TWI ≥ 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 TWI confined to leads V1–V4 in black athletes when preceded by J point and/or ST segment elevation, and TWI in leads V1–V3 in athletes aged < 16 years.

Clinical considerations

The relationship between abnormal TWI and several forms of structural heart disease is well documented.[56] TWI in the inferior or lateral leads is common in HCM.[56–59] Whereas TWI in the right precordial leads (V1–V3) or beyond in the absence of a complete RBBB is common in ARVC (*Figure 4D*).[60,61]

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 TWI in the lateral or inferolateral leads is associated with the presence of quiescent cardiomyopathy in a considerable proportion of athletes.[30,62–64] Recommendations for the evaluation of abnormal TWI and other clinical considerations are presented in *Table 2*.

TWI 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 TWI, 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.[65]

For athletes with lateral or inferolateral TWI, 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.[62,63]

Anterior T-wave inversion

Anterior TWI 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.[66,67] However, anterior TWI 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 TWI inversion before considering further investigations.[5,6,14,29] A large study of over 14 000 white adults aged 16–35 years old, including over 2500 athletes showed that anterior TWI had a prevalence of 2.3%.[68] Anterior TWI 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.[68] None of the individuals with anterior TWI 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 TWI beyond V2 in the absence of other clinical or electrical features of ARVC.

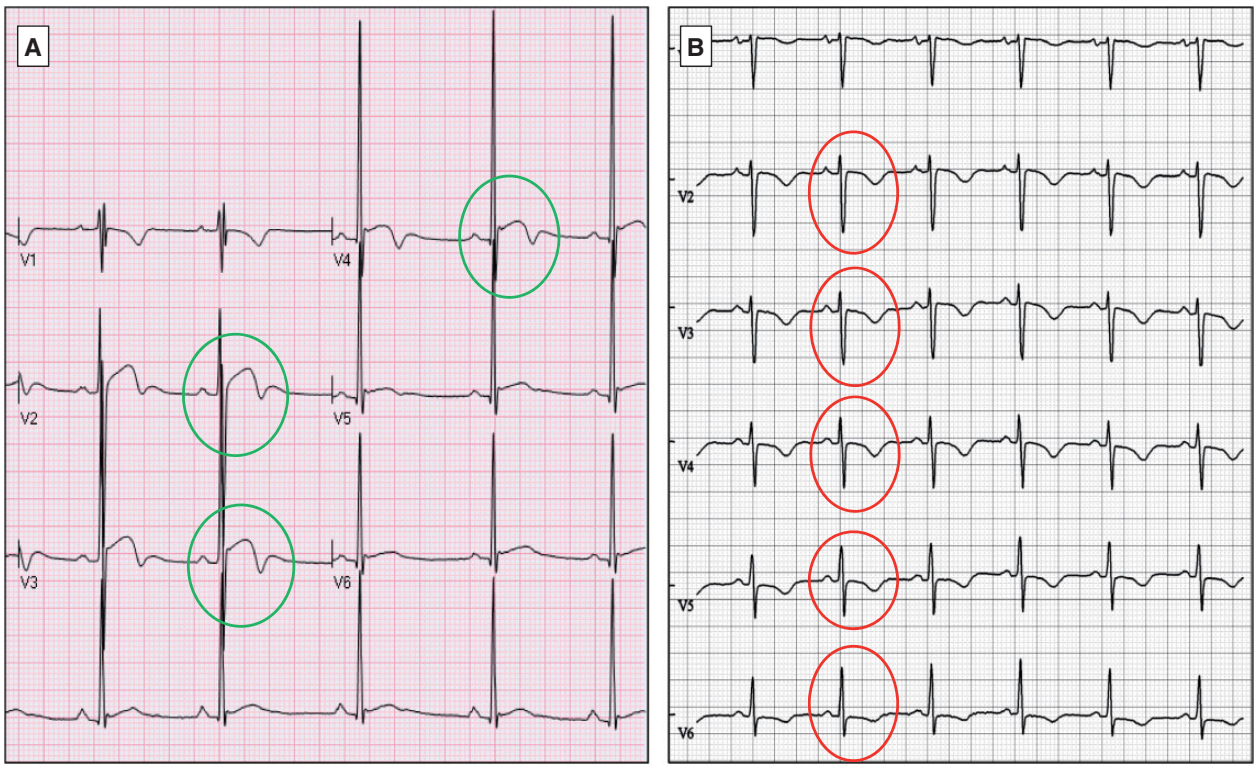

Figure 5 Examples of physiological (*A*) and pathological T-wave inversion (*B*). Panel A demonstrates T-wave inversion in V1–V4 preceded by J-point elevations and convex 'domed' ST segment elevation (green circles). This should not be confused with pathological T-wave inversion (Panel B) which demonstrates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

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 ≥ 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 ≥ 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.

Inferior T-wave inversion

The significance of TWI confined to the inferior leads is unknown. However, this finding cannot be attributed to physiological remodelling 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

infarction can lead to the development of exaggerated (deep or wide) Q waves or unexpected Q waves in atypical leads.[28,56] Pathological Q waves also may be a result of lead misplacement. In particular, a pseudo-septal infarct pattern with pathological Q waves in leads V1–V2 is commonly due to high-lead placement relative to cardiac position.[72]

Pathological Q waves have been reported in approximately 1–2% of all athletes, and may be higher in males and black athletes.[29,73] For asymptomatic athletes, pathological Q waves were previously defined as > 3 mm in depth or > 40 ms in duration in two or more leads (except III and aVR).[6,10] In practice, however, this criterion is a common source of false positive ECG results as trained athletes with physiological LVH and thin adolescent athletes may have increased precordial voltages and deep lateral or inferior Q waves.

The use of a Q/R ratio overcomes some of these issues by normalizing Q wave depth to the degree of proceeding R wave voltage. Case control analyses of athletes and HCM patients suggest that this will decrease the false positive rate without compromising sensitivity for the detection of cardiomyopathy.[29,74] Thus, this consensus panel has modified the definition for pathological Q waves in athletes as a Q/R ratio ≥ 0.25 or ≥ 40 ms in duration in two or more contiguous leads (except III and aVR).

Evaluation

An ECG with abnormal Q waves should be carefully examined for the possibility of an accessory pathway. If the pathological Q waves are isolated to leads V1–V2, the ECG should be repeated, including re-placing the ECG leads to ensure proper positioning. Persistence of pathological Q waves in two or more contiguous leads warrants further investigation with echocardiography to exclude cardiomyopathy. If the echocardiogram is normal and there are no other concerning clinical findings or ECG abnormalities, no additional testing is generally necessary. However, if there is a high index of clinical suspicion, additional evaluation with cardiac MRI should be considered. In athlete's age ≥ 30 years with suspicion of prior myocardial infarction or risk factors for coronary artery disease (CAD), stress testing may be warranted.

Complete left bundle branch block

LBBB is found in less than 1 in 1000 athletes but is common in patients with cardiomyopathy and ischaemic heart disease.[9,28,59,75,76] Thus, complete LBBB always should be considered an abnormal finding and requires a comprehensive evaluation to rule out a pathological cardiac disorder.

Evaluation

Athletes with complete LBBB require a thorough investigation for myocardial disease including echocardiography and a cardiac MRI with perfusion study.

Profound non-specific intra-ventricular conduction delay

Epidemiological studies of nonspecific intra-ventricular conduction delay (IVCD) in the general population have shown an increased risk of cardiovascular death and have been documented among patients with cardiomyopathy.[77,78] The significance of non-specific IVCD with normal QRS morphology in healthy, asymptomatic athletes is uncertain.[79] The physiology underlying IVCD in athletes remains incompletely understood but likely includes some combination of neurally mediated conduction fibre slowing and increased myocardial mass. In patients with LVH, left ventricular mass seems to be closely related to QRS duration.[80]

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 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.[81] The WPW pattern occurs in up to 1 in 250 athletes.[9,12,52,82] 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.[83,84] 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.[85] 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.[83] 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.[83,86] 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'

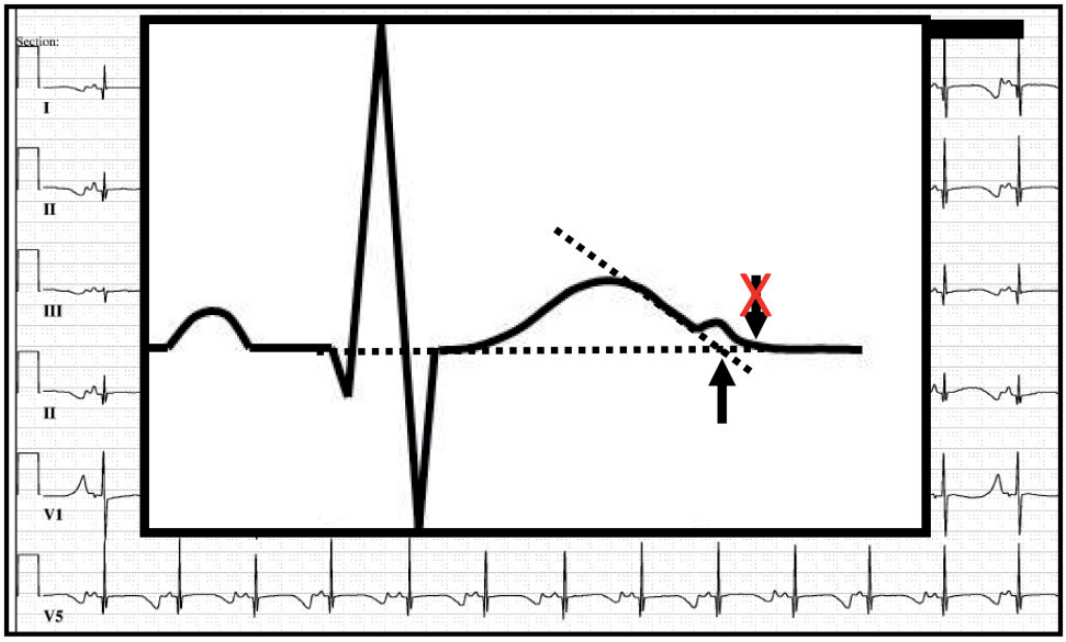

Figure 6 This figure illustrates the 'Teach-the-Tangent' or 'Avoid-the-Tail' method for manual measurement of the QT interval. A straight line is drawn on the downslope of the T wave to the point of intersection with the isoelectric line. The U wave is not included in the measurement.

LQTS.[87] Autopsy negative sudden unexplained death represents 25–40% of sudden unexpected deaths in persons under age 40 years.[3,88–90] 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.[91–94]

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.[95] However, accurate assessment of the QTc can be achieved by adhering to the following six principles:[96]

(1) Use Bazett's heart rate correction formula ($QTc = QT/\sqrt{RR}$; note the RR interval is measured in seconds) as population-based QTc distributions most frequently use Bazett-derived QTc values.[97]
(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*).[96]
(6) The morphology of the T wave, not just the length of the QT interval, also can suggest the presence of LQTS.[98] 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.[6,10] These cut-off values are around the 99th percentile and consistent with thresholds defined by the American Heart Association and

American College of Cardiology.[99] This consensus group also recommends QTc values of ≥ 470 ms in males and ≥ 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 < 320 ms is 0.1%; suggesting an abnormal cut-off value of < 320 ms is pragmatic.[100] 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.[100] 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).[101] 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.[101]

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.[102] 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.[103–106]

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 ≥ 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.[107–109]

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/J-point (STJ) and 80 ms after the start of the ST segment (ST80).[110] 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 ≤ 30 bpm or a PR interval ≥ 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

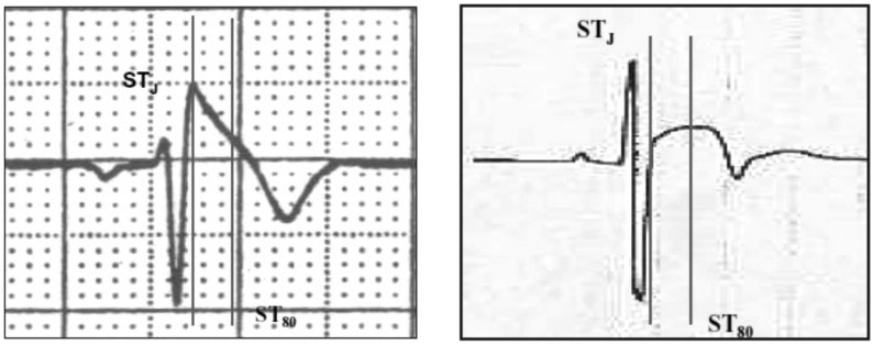

Figure 7 Brugada Type 1 electrocardiogram (left) should be distinguished from early repolarization with 'convex' ST segment elevation in a trained athlete (right). Vertical lines mark the J-point (STJ) and the point 80 ms after the J-point (ST80), where the amplitudes of the ST segment elevation are calculated. The 'downsloping' ST segment elevation in Brugada pattern is characterized by a STJ/ST80 ratio > 1. Early repolarization patterns in an athlete show an initial 'upsloping' ST segment elevation with STJ/ST80 ratio < 1.

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.[9,12] Although multiple PVCs are usually benign, their presence may be the hallmark of underlying heart disease.[111,112] 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.[113] 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.[112] 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.[114,115] Although some studies have suggested that regression of the PVC burden with detraining indicates a good prognosis, other studies have not confirmed this.[116–118] 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.[119] 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 $\geq$ 30 years of age

In athletes $\geq$ 30 years of age, CAD is the most common cause of SCD.[89,90] In addition, older athletes may be less fit compared with 20–30 years ago, increasing the possibility of underlying CAD.[120,121] 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.[122–124]

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.[123,125,126] 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.[62,63] 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.

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