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International recommendations for electrocardiographic interpretation in athletes

# International Consensus Standards for Electrocardiographic Interpretation in Athletes

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## ABSTRACT

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 February 26-27, 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), such as cardiomyopathies and primary electrical diseases (channelopathies), are suggested or identified by abnormalities present on a resting 12-lead ECG. Interpretation of an ECG in athletes requires careful analysis to properly distinguish physiological changes related to athletic training from findings suggestive of an underlying pathological condition. 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

On February 26-27, 2015, an international group of experts in sports cardiology, inherited cardiac diseases, and sports medicine convened in Seattle, Washington, to update contemporary standards for ECG interpretation in athletes through development of an "international consensus". This summit meeting served as the critical foundation for subsequent work done by the larger writing group that ultimately generated this document. The goals of the summit meeting were to: 1) update ECG interpretation standards based on new and emerging research; and 2) develop a clear guide to the appropriate evaluation of ECG abnormalities for conditions associated with SCD in athletes.

The standards presented were developed with consideration of ECG interpretation in the context of an asymptomatic athlete aged 12-35 years. An athlete is defined as an individual who engages in regular exercise or training for sport or general fitness, typically with a premium on performance, and often engaged in individual or team competition. The prevalence of specific ECG findings in athletes may vary based on age, gender, ethnicity, type of sport and level of conditioning. Training-related physiological changes are more common in athletes exercising intensively at least 4-8 hours per week; thus prudent application of the criteria should occur in individuals at lower levels of regular exercise. Novel to these standards are specific considerations presented for young adolescent athletes age 12-16 years, as well as for older athletes ≥ 30 years where the prevalence of occult coronary artery disease (CAD) sharply increases. In the presence of cardiac symptoms or a family history of inherited cardiovascular disease or premature SCD, the interpretation standards may require modification.

The recommendations presented in this statement were 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 consensus recommendations from this panel is presented in Figure 1, Table 1, and Table 2. Physicians may choose to deviate from these consensus standards based on their experience or practice setting and according to the individual characteristics of the athlete. Ideally, the evaluation of ECG abnormalities is performed in consultation with a specialist with knowledge and experience in training-related cardiac adaptations and disorders associated with SCD in young athletes.

**Distinguishing Normal from Abnormal**

A challenge in the interpretation of an athlete's ECG is the ability to accurately differentiate findings suggestive of a potentially serious cardiovascular disorder from benign physiological adaptations occurring as the result of regular, intense training (i.e., athlete's heart). Several reports have outlined contemporary ECG criteria intended to distinguish normal ECG findings in athletes from ECG abnormalities requiring additional evaluation.[4-10]

**Evolution of ECG Interpretation Standards**

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. In 2005, criteria for an abnormal ECG were defined by the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology (ESC).[11] In 2010, driven by new research and large-population clinical investigations,[12,13] the ESC updated recommendations for interpretation of an athlete's ECG in a landmark statement which pioneered "modern" ECG interpretation standards.[5] The 2010 ESC criteria were the first to divide ECG findings into two groups – common and training-related (Group 1) versus uncommon and training-unrelated (Group 2) – based on the prevalence of ECG findings, relation to exercise training, and association with pathological conditions associated with SCD requiring further clinical investigation to confirm (or exclude) underlying cardiovascular disease.[5] Since then, other investigators and international panels have proposed updated guidelines for ECG interpretation in young athletes.[6,9,14] In 2012, leading sports medicine, inherited cardiac disease specialists, and sports cardiology physicians with expertise in the cardiovascular evaluation of young competitive athletes convened in Seattle, Washington, to redefine contemporary standards and develop an online training course (http://learning.bmj.com/ECGathlete) for ECG interpretation in athletes.[10,15-17]

Each revision of the ECG standards has demonstrated improved specificity while maintaining the sensitivity for ECG detectable pathological conditions associated with SCD. For example, in a study of 1,078 elite Australian athletes, the false-positive rate decreased from 17% using the 2010 ESC criteria to 4.2% with the Seattle criteria, with no change in sensitivity.[18] In another study of 2,017 high school athletes from the U.S., the Seattle criteria produced a low false-positive rate (2.8%) with 100% sensitivity for SCD-associated conditions.[19] In a study of 1,417 high school, college, and professional athletes comparing three published ECG interpretation algorithms for athlete pre-participation screening, the proportion of abnormal ECGs declined from 26% to 8.1% to 5.7% using the ESC, Stanford, and Seattle criteria, respectively.[20]

Recent research has directed additional revisions to ECG interpretation standards that further lower the false-positive rate.[21,22] The 'refined' criteria were investigated in an instrumental study examining 4,297 white and 1,208 black elite athletes, and 103 athletes diagnosed with hypertrophic cardiomyopathy (HCM).[23] The total ECG abnormal rate declined dependent on the criteria applied – 21.5% (ESC), 9.6% (Seattle), and 6.6% (refined) – while all three criteria identified 98.1% of athletes with established HCM.[23] Of the 5,505 athletes screened, 15 (0.47%) were diagnosed with conditions associated with SCD, including 5 cases of HCM, 5 cases of

Wolff-Parkinson-White (WPW), 3 cases of long QT syndrome (LQTS), and 1 case each of Brugada syndrome (BrS) and an anomalous coronary artery.[23] Fourteen of 15 (93%) serious conditions were identified by ECG, and only one identified because of prior symptoms.[23] In another study of 2,491 athletes undergoing pre-participation screening, application of the ESC, Seattle, and 'refined' criteria led to abnormal ECG rates of 22.3%, 11.6%, and 5.3%, respectively, all with 100% sensitivity for the pathological conditions detected.[24]

Effective use of ECG in the cardiovascular care of athletes requires that abnormal findings receive appropriate secondary investigations to confirm or exclude conditions associated with SCD. However, the clinical response to abnormal ECG findings may vary based on physician training and experience. As a guide for clinicians, this document provides recommendations for the evaluation of specific ECG abnormalities.

**Limitations**

While ECG increases the ability to detect underlying cardiovascular conditions that place athletes at increased risk of SCD, ECG as a diagnostic tool has limitations in both sensitivity and specificity. Specifically, ECG can suggest or detect cardiomyopathies, ion channelopathies, myocarditis, and ventricular pre-excitation, yet other causes of SCD in young athletes such as anomalous coronary arteries, premature coronary atherosclerosis and aortopathies are not readily detected by ECG. Thus, even if properly interpreted, an ECG will not detect all conditions predisposing to SCD, and evaluation of cardiovascular symptoms, a concerning family history, or abnormal physical examination requires a more comprehensive investigation.

An objective of developing ECG standards is to improve the accuracy and reproducibility of interpretation. Indeed, studies have demonstrated that systematic evaluation of an athlete's ECG using standardized criteria improves interpretation accuracy among physicians across disciplines.[14,25] However, inter-observer variability and the reliability of ECG standards even among experienced physicians remains a major concern.[26,27] In one study, pediatric cardiologists, without use of a standardized criteria set, achieved a sensitivity of only 68% and a specificity of 70% for recognition of abnormal ECG patterns that occur infrequently but represent conditions causing SCD.[28] In the future, the accuracy of ECG interpretation in athletes may be assisted by ECG devices using athlete-specific interpretation algorithms.

**The Importance of Proper Electrode Placement**

Misplacement of the limb leads can cause errors in axis measurement and pseudo-Q waves. Misplacement of the precordial electrodes downward can cause pseudo-Q waves and failure to detect ST segment depression. Upward misplacement of the precordial leads can simulate myocardial injury, pericarditis or the Brugada type 2 pattern, due to pseudo-ST segment elevation. Right and left arm lead reversal is recognized by negative P waves, a negative QRS complex, and T wave inversion (TWI) in leads I and aVL but not in the lateral precordial leads (V5-V6).

Accurate placement of the precordial leads can be challenging. Leads V1, V2 and V4 have distinct bony landmarks for their placement that are critical for the placement of V3, V5 and V6.

V1 and V2 are close to the sternum in the 4th intercostal space (usually just above the nipple level). V4 is in the 5th intercostal space (usually just below the nipple line) and at the mid-clavicular line. V3 is placed on a line between V2 and V4. V5 and V6 are on a horizontal plane set by V4 and do not curve along the interspace. They intersect with vertical lines established by the anterior axillary fold (V5) and the mid-point of the axilla (V6).


## NORMAL ECG FINDINGS IN ATHLETES

### Overview of Physiological Cardiac Adaptations to Regular Exercise

Regular and long-term participation in intensive exercise (minimum of 4 hours per week) is associated with unique electrical manifestations 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).

Increased Cardiac Chamber Size
Voltage criterion for left ventricular hypertrophy (LVH) is observed commonly on an athlete's ECG and thought to reflect a physiological increase in cardiac mass from athletic cardiac remodeling (Figure 2). Likewise, voltage criteria for right ventricular hypertrophy (RVH) and incomplete right bundle branch block (RBBB) are also common ECG findings in athletes and considered a result of increased right ventricular (RV) size secondary to regular training.

Increased Vagal Tone
Common consequences of increased vagal tone include early repolarization, sinus bradycardia, and sinus arrhythmia (Figure 2). 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).

### QRS Voltage Criteria for Ventricular Hypertrophy

Left Ventricular Hypertrophy
QRS voltage can be influenced by a variety of factors, with male gender, athletic activity and younger age associated with higher QRS voltage, while obesity and pulmonary disease may cause lower voltage.[29] All existing ECG criteria for LVH correlate poorly with increased left ventricular (LV) wall thickness and LV mass on imaging studies.[30] Although there are several voltage criteria to define LVH, the Sokolow-Lyon criterion is used most commonly, defined as the sum of the S wave in V1 and the R wave in V5 or V6 (using the largest R wave) as > 3.5 mV (35 small squares with a standard amplification of the ECG at 10 mm/1 mV). Trained athletes often satisfy QRS voltage criterion for LVH, with up to 64% of athletes fulfilling the Sokolow-Lyon index (Figure 2).[31-36]

The presence of isolated QRS voltage criterion for LVH does not correlate with pathology in athletes and is present in isolation (without other associated ECG abnormalities) in less than 2% of patients with HCM.[30-32,37-40] Although clearance of athletes with isolated voltage criterion for LVH will fail to detect a very small minority of individuals with HCM, such individuals have a

milder phenotype with a low arrhythmogenic risk.[40] Conversely, pathological LVH is commonly associated with additional ECG features such as TWI in the inferior and lateral leads, ST segment depression, and pathological Q waves (Figure 3).[23,41] 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 the normal and training-related ECG changes in athletes related to physiological increases in cardiac chamber size and/or wall thickness and does not in itself require further evaluation. However, the additional presence of TWI, ST segment depression, or pathological Q waves should raise the possibility of pathological LVH and should prompt further evaluation.

<u>Right Ventricular Hypertrophy</u>
Voltage criterion for RVH is also fairly common in athletes with up to 13% of athletes fulfilling the Sokolow-Lyon index (R wave in V1 + largest S wave in V5 or V6 > 10.5 mV).[21,33] The presence of QRS voltage criterion for RVH correlates poorly with increased RV wall thickness on echocardiography.[21] Most importantly, QRS voltages for RVH, when present in isolation, do not correlate with underlying pathology in athletes, in particular arrhythmogenic right ventricular cardiomyopathy (ARVC) or pulmonary hypertension.[21] In a study comparing 627 athletes to patients with established ARVC or pulmonary hypertension, none of the athletes with isolated RVH by voltage criterion had any RV pathology on advanced cardiac imaging, and none of the patients with ARVC or pulmonary hypertension exhibited voltage criterion for RVH without additional ECG abnormalities.[21] Based on these considerations, it is reasonable to conclude that, similar to voltage criteria for LVH, isolated QRS voltage for RVH is part of the normal spectrum of ECG findings in athletes and in the absence of other ECG or clinical markers of pathology does not require further evaluation.

**Incomplete Right Bundle Branch Block**

Incomplete RBBB is defined by a QRS duration < 120 ms with a RBBB pattern: terminal R wave in lead V1 (commonly characterized as an rSR' pattern) and wide terminal S wave in leads I and V6 (Figure 4). Studies suggest that the mildly delayed RV conduction in athletes is caused by RV remodeling, with increased cavity size and resultant increased conduction time, rather than an intrinsic delay within the His-Purkinje system.[42] Therefore, incomplete RBBB represents a phenotype of cardiac adaptation to exercise and in the absence of other features suggestive of disease does not require further evaluation.

**Early Repolarization**

By convention, early repolarization is defined as elevation of the QRS-ST junction (J-point) by $\geq$ 0.1 mV often associated with a late QRS slurring or notching (J wave) affecting the inferior and/or lateral leads (Figure 2).[43-45] Early repolarization is a common finding in healthy populations (2-44%) and is more prevalent in athletes, young individuals, males, and black ethnicity.[43,46-50] Early repolarization consisting of J-point elevation with concave ST segment elevation and a peaked T wave 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).[32,33]

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.[44,51] A study of middle-aged individuals demonstrated the presence of an early repolarization pattern in the inferior leads accompanied by a horizontal or descending ST segment after the J-point, was associated with an increased risk of arrhythmic death, while a rapidly ascending ST segment, the dominant ST pattern in healthy athletes, was associated with a benign prognosis.[43,46] Early repolarization of some subtypes appears to be a dynamic process in athletes which is affected directly by exercise training, increasing in frequency at times of peak fitness.[48,49,52] 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.[53]

**Repolarization Findings in Black Athletes**

The electrocardiographic expression of the athlete's heart is influenced by several factors. Over the past decade, ethnicity has emerged as a major determinant of cardiac adaptation to exercise with black athletes exhibiting a higher prevalence of electrocardiographic anomalies, including repolarization changes. Notably, more than two-thirds of black athletes exhibit ST segment elevation and up to 25% demonstrate TWI on their ECG.[23,33,54,55]

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 5). A study of 904 black male athletes of African descent demonstrated that 13% exhibited isolated TWI in leads V1-V4 compared to only 4% of black sedentary controls.[33] The majority of anterior TWI were preceded by J-point elevation and convex (domed) ST segment elevation. None of the athletes with anterior TWI showed symptoms or signs of cardiomyopathy despite comprehensive evaluation and a 5-year follow-up period.[33] Similar findings have been described in female and adolescent black athletes.[54-56] Based on these considerations, TWI in leads V1-V4 when preceded by J-point elevation and convex ST segment elevation should be considered part of the "black athlete's heart" 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" ECG 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 (Figure 6). The juvenile pattern 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.[32,57,58] Anterior TWI that extends beyond lead V2 is rare (0.1%) in white athletes aged ≥ 16 years or younger athletes who have completed puberty when formal assessment by a pediatrician is feasible.[32,57] Based on current evidence, TWI in the anterior leads (V1-V3) in adolescent athletes < 16 years of age (or pre-pubertal athletes) should not prompt further evaluation in the absence of symptoms, signs or a family history of cardiac disease.

## Sinus Bradycardia

Sinus bradycardia is defined as a heart rate < 60 beats per minute (bpm) and is present in up to 80% of highly trained athletes.[32,35] In normal sinus rhythm, the heart rate is determined by the balance between the sympathetic and parasympathetic nervous systems. Resting sinus bradycardia is particularly prevalent in endurance athletes due to increased vagal tone and possible structural atrial remodelling.[59,60] In the absence of symptoms such as fatigue, dizziness, or syncope, heart rates ≥ 30 bpm are considered normal in highly trained athletes. Sinus bradycardia should resolve with the onset of physical activity.

## Sinus Arrhythmia

Sinus arrhythmia, the physiological fluctuation in heart rate with breathing, is considered a normal finding and should not be confused with sinus node dysfunction or sick sinus syndrome. Differentiating features that suggest sinus node dysfunction include lack of rhythmic changes in the heart rate, abrupt sustained rate increases and decreases, prolonged pauses or periods of sinus arrest, inappropriate rate response to exercise (including slowed acceleration and an inappropriately rapid deceleration), as well as any association with clinical symptoms such as exercise intolerance, pre-syncope and syncope. In sinus arrhythmia, the P wave axis remains normal in the frontal plane and the fluctuation in heart rate should resolve with the onset of exercise.

## Junctional Escape Rhythm

A junctional escape (nodal) rhythm occurs when the QRS rate is faster than the resting P wave or sinus rate, which is typically slower in athletes due to increased vagal tone (Figure 7). The R-R interval is regular in a junctional escape rhythm, and the QRS complexes are narrow unless the baseline QRS has a bundle branch block. Sinus rhythm should resume with the onset of physical activity.

## Ectopic Atrial Rhythm

In an ectopic atrial rhythm, P waves are present but of different morphology compared to the sinus P wave, typically with a rate ≤ 100 bpm. Ectopic P waves are most easily seen when the P waves are negative in the inferior leads (Figure 8). Occasionally, two different P wave morphologies may be seen and this is known as a 'wandering atrial pacemaker'. A junctional escape rhythm or wandering atrial pacemaker is observed in up to 8% of all athletes under resting conditions.[32] Ectopic atrial rhythms occur due to a slowed resting sinus rate from increased vagal tone in athletes. Sinus rhythm should resume with the onset of physical activity.

## First Degree AV Block

First degree AV block is found in up to 7.5% of athletes on a resting ECG and is characterized by a prolonged (> 200 ms) PR interval.[32,35,61] This represents a delay in AV nodal conduction in

athletes, due to increased vagal activity or intrinsic AV node changes, and typically resolves with the onset of exercise.

**Mobitz Type I (Wenckebach) Second Degree AV Block**

In Mobitz Type I second degree AV block, the PR interval progressively lengthens from beat to beat, until there is a non-conducted P wave with no QRS complex (Figure 9). The first PR interval after the dropped beat is shorter than the last conducted PR interval before the dropped beat. This represents a greater disturbance of AV nodal conduction than first degree AV block, but is usually a normal finding in asymptomatic, well-trained athletes, and 1:1 conduction should return with the onset of exercise.

**Sports-specific ECG Considerations**

Adaptive changes in cardiac structure, function and electrophysiology occur in response to regular intense physical training. A relatively strong relationship exists between the amount of training performed, resulting fitness, and extent of adaptive cardiac remodeling. Using measured $VO_2$ max as a marker of athlete fitness, a robust linear association has been demonstrated between fitness and cardiac size.[62] In this context, it is not surprising that many ECG features associated with athletic training are more prevalent and more profound in athletes exposed to the greatest training stimulus. This concept has most frequently been addressed by comparing athletes involved in team, skill-based or strength sports with those competing in endurance exercise. In a study comparing the ECGs of 1,010 elite non-endurance and 251 endurance athletes, the latter group demonstrated more frequent bradycardia, voltage criterion for LVH, and early repolarization – all features considered common, benign and training-related.[63] However, uncommon features such as TWI extending to lead V3 was more common (4.0 vs. 1.1%, p<0.0001) in endurance athletes as compared with non-endurance athletes.[63] Another study also reported a higher prevalence of TWI in the anterior septal leads in elite rowers compared to non-endurance or mixed athletic cohorts.[64] Incomplete and complete RBBB are also commonly observed in athletes with a prevalence which appears to increase with cardiac size, thereby suggesting it is more frequently observed in endurance athletes.[42]


**BORDERLINE ECG FINDINGS IN ATHLETES**

**Overview of Borderline ECG Findings in Athletes**

Recent data suggests that some ECG findings previously categorized as abnormal may also represent normal variants or the result of physiological cardiac remodeling in athletes and do not usually represent pathological cardiac disease. These ECG findings, specifically axis deviation, voltage criteria for atrial enlargement, and complete RBBB, have been categorized as 'borderline' findings in athletes (Figure 1; Table 1).

**Axis Deviation and Voltage Criteria for Atrial Enlargement**

Several studies have shown that the Seattle criteria reduces the number of positive ECGs compared to the 2010 ESC criteria by at least 40% and improves specificity without compromising sensitivity.[18,23,24,65] Despite the improvements, more recent publications suggest that axis deviation and voltage criteria for atrial enlargement account for a large number of ECG patterns that are classified as abnormal in athletes but do not correlate with cardiac pathology. In a large study of 2,533 athletes aged 14-35 years old and 9,997 controls of similar age, isolated left or right axis deviation and isolated left or right atrial enlargement comprised 42.6% of all ECG findings.[22] The athletes revealed a slightly higher prevalence of these findings compared to controls (5.5% versus 4.4%; p=0.023). Specifically, athletes showed a higher prevalence of left axis deviation (LAD) and left atrial enlargement (LAE) versus controls (1.46% vs. 0.96%; p=0.028 and 2.13% vs. 1.37%; p=0.007, respectively), particularly in those who trained over 20 hours per week.[22] Whereas there were no significant differences in the prevalence of right axis deviation (RAD) and right atrial enlargement (RAE) between the groups (1.11% vs. 1.10%; p=0.983 and 0.83% vs. 0.92%; p=0.664, respectively).[22]

Athletes with LAD or LAE exhibited larger left atrial and ventricular dimensions compared with athletes with a normal ECG and those with other physiological ECG changes consistent with athlete's heart.[22] However, there were no appreciable differences in the number of athletes with cardiac dimensions exceeding predicted upper limits between the two groups. In contrast, there were no differences in cardiac dimensions between athletes with RAD or RAE compared to athletes with normal or usual physiological ECG changes.[22] 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, and the prevalence of minor congenital abnormalities was similar in these individuals to those with normal ECGs.[22]

Exclusion of axis deviation or atrial enlargement reduced the false positive rate from 13% to 7.5% and improved specificity from 90% to 94% with a minimal reduction in sensitivity (91% to 89.5%).[22] A U.S. study of 508 university athletes also revealed that of the 49 athletes considered to have abnormal ECGs at least 29 (59%) exhibited either voltage criteria for LAE alone or in combination with large QRS complexes, and subsequent echocardiography revealed a structurally normal heart or findings consistent with athletic training.[39]

These findings suggest that RAD and RAE occurring in isolation or in association with other electrical markers of 'athlete's heart' are probably normal variants, whereas LAD and LAE may reflect a relative increase in LV dimensions in some athletes.

**Complete Right Bundle Branch Block**

Although incomplete RBBB is common in young athletes, the significance of complete RBBB is less certain. This particular ECG pattern was placed in the abnormal category in the 2010 ESC recommendations and more recent 'refined' criteria, although considered a normal finding in the Seattle criteria if the QRS duration was < 140 ms.[5,10,23] 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%.[12,66-68] In a study of 510 U.S. collegiate athletes, RBBB was reported in 13 (2.5%) athletes and compared to athletes with normal QRS complexes and athletes with incomplete RBBB.[42] The 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 suggesting that that this particular ECG pattern may be a manifestation of more extreme right ventricular adaptation to exercise. These patterns among trained athletes could represent a spectrum of structural and physiological cardiac remodeling characterized by RV dilation with resultant QRS prolongation and a relative reduction in the RV systolic function at rest.[42]

Based on the aforementioned considerations, LAD, LAE, RAD, RAE 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 in combination places the athlete in the abnormal category warranting additional investigation (Figure 10).

## ABNORMAL ECG FINDINGS IN ATHLETES

### Overview of ECG Criteria for the Detection of Pathological Cardiac Disorders in Athletes

Many pathological cardiac disorders associated with SCD exhibit abnormalities on a resting ECG. TWI is the most consistent electrical abnormality in patients with cardiomyopathy. ST segment depression, pathological Q waves, and left bundle branch block (LBBB) are also recognized in cardiomyopathic disorders and ischaemic heart disease. Primary electrical disease such as ventricular pre-excitation, LQTS, and BrS are suggested and/or diagnosed by abnormal ECG findings. None of these abnormal findings as defined in this section are recognized features of athletic training and always require further assessment to exclude the presence of intrinsic cardiac disease (Figure 1; Table 1; Table 2). When ECG abnormalities are identified, an athlete's personal and family history should be thoroughly questioned as part of a comprehensive clinical investigation.

### Abnormal T Wave Inversion

T waves represent repolarization or recovery of the ventricular myocardium and constitute the final waveform of the cardiac cycle on ECG. T waves can be described by numerous attributes including duration, symmetry, skewness, slope of the ascending and descending waveform, and most importantly, directionality. Directionality (i.e. positivity or negativity) refers to the direction of T wave deflection with regards to the electrically neutral ECG baseline, conventionally defined as the PR segment. T wave directionality is typically concomitant with the net vector of the QRS complex and is thus deflected above the baseline (i.e. positive) in most leads. Normal exceptions, leads in which T waves are routinely negative, include aVR, III, and V1. T waves deflected negatively are referred to as TWI. When present in leads aside from aVR, III, and V1, TWI may have an association with underlying structural heart disease. TWI $\geq 1$ 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 (with the exception of TWI confined to

leads V1-V4 in black athletes and leads V1-V3 in all athletes aged < 16 years) and should prompt further evaluation for underlying structural heart disease (Table 1; Table 2).

Clinical Considerations

The relationship between abnormal TWI and several forms of structural heart disease including HCM, ARVC, dilated cardiomyopathy (DCM), left ventricular non-compaction, and myocarditis is well documented. In a cohort of young (age < 35 years) asymptomatic patients with confirmed HCM, 62% exhibited abnormal TWI.[69] In a similar study of asymptomatic or mildly symptomatic HCM patients, the prevalence of abnormal TWI was reported at 80%.[70] Most recently, the prevalence of abnormal TWI among athletes with newly diagnosed HCM was reported in excess of 90%.[71] In a study comparing 1,124 athletes with 255 patients with HCM, TWI in V4-V6 were present in 38% of patients with HCM versus 0.8% of athletes (p<0.001) (Figure 3).[72]

ARVC is similarly associated with abnormal TWI. Specifically, the presence of TWI in the right precordial leads (V1-V3) or beyond in the absence of a complete RBBB is common among ARVC patients and has been adopted as a major criterion for diagnosis (Figure 11).[73,74] Recently, the deleterious prognostic implications of TWI involving non-anterior lead territories among patients with ARVC, particularly inferior lead TWI, have been demonstrated.[75,76]

Abnormal TWI is relatively infrequent among healthy white competitive athletes with a reported prevalence ranging from < 1.0 to 4% based on factors including age, gender and type of sport but is reported in as many as 12% of black athletes.[18,32,33,39,56,64,77-79] Based on the overlap with the cardiomyopathies, abnormal TWI in asymptomatic athletes warrants a comprehensive clinical assessment to exclude underlying cardiomyopathy. There are no data relating to the significance of flat or biphasic T waves in athletes. 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 TWI*

There is mounting evidence that TWI in the lateral or inferolateral leads, in any athlete, are associated with the presence of quiescent cardiomyopathy.[33,79-81] Recommendations for the evaluation of abnormal TWI and other clinical considerations are presented in Table 2.

Although rarely seen in athletes without subsequently identified structural heart disease, TWI affecting the lateral leads (V5-V6, I and aVL) is considered abnormal and should prompt comprehensive investigation irrespective of ethnicity, including cardiac MRI, when echocardiography is non-diagnostic. Echocardiographic quality is variable and may have limited ability to detect hypertrophy of the anterolateral LV wall and apex.[82] Contrast-enhanced cardiac MRI provides superior assessment of myocardial hypertrophy and may demonstrate late gadolinium enhancement (LGE) which is a non-specific marker suggesting myocardial fibrosis. Exercise ECG testing and Holter monitoring also should be considered in the evaluation of lateral or inferolateral TWI, especially for 'grey zone' hypertrophy (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 a pathological diagnosis and is useful in risk stratification.[83]

Cardiac MRI should be a standard component of the assessment for markedly abnormal ECGs suggestive of apical HCM, specifically ECGs with deep (> -0.2 mV) TWI and ST segment depression in the lateral or inferolateral leads in which echocardiography may not provide adequate assessment of the LV apex and inferior septum. If cardiac MRI is not available, echocardiography with contrast should be considered as an alternative investigation for apical HCM. For athletes with lateral 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 (Figure 12).[80,81]

*Anterior TWI*
Anterior TWI is a normal variant in asymptomatic adolescent athletes age < 16 years and in black athletes when preceded by J-point elevation and convex ST segment elevation.[84] Anterior TWI involving lead V3 is also reported in white, adult, predominantly endurance athletes.[63] However, anterior TWI in leads V1-V2/V3 also is a recognized pattern in patients with ARVC and more rarely HCM (Figure 11). There are discrepancies among existing guidelines relating to the extent of anterior TWI inversion before considering further investigations.[5,6,16,23]

A large study of over 14,000 white adults aged 16-35 years old, including over 2,500 athletes showed that anterior TWI had a prevalence of 2.3%.[85] 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.[85] 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 would be justifiable to investigate only athletes with anterior TWI beyond V2 in the absence of other clinical or electrical features of ARVC.

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, patients with HCM, and patients with 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.[84] Conversely, anterior TWI associated with minimal or absent J-point elevation (< 1 mm) could reflect a cardiomyopathy.[84] 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.[34]

In most non-black athletes age ≥ 16 years, anterior TWI beyond lead V2 should prompt further evaluation given the potential overlap with ARVC. The extent of evaluation is dependent on the specific ECG findings suggestive of ARVC and will be more extensive in the presence of warning symptoms or significant family history. In athletes age ≥ 16 years with TWI beyond V2, 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.[84] Other ECG findings suggestive of ARVC in the presence of anterior TWI include low limb lead voltages, prolonged S wave upstroke, ventricular

ectopy with LBBB morphology, and epsilon waves.[74] A combination of tests is needed to effectively make the diagnosis of ARVC and may include echocardiography, cardiac MRI, Holter monitoring, exercise ECG test, and signal averaged ECG (SAECG).

Repolarization variants in the anterior precordial leads in black athletes must be distinguished from pathological repolarization changes found in ARVC (Figure 13). In ARVC, the J-point and/or ST segment is usually isoelectric or depressed prior to TWI, in contrast to the J-point elevation and 'domed' ST segment elevation which is the hallmark feature of the physiological repolarization variant in black athletes.

*Inferior TWI*
The significance of TWI confined to the inferior leads (II, III, and aVF) has not been studied in detail. TWI isolated only to the inferior leads is not a strong predictor of cardiomyopathy in the absence of other abnormal ECG or clinical features. However, this finding cannot be attributed to physiological remodeling and thus warrants further investigation with, at minimum, an echocardiogram until larger studies with longer follow up prove otherwise. Cardiac MRI should be considered based on the echocardiographic findings or clinical suspicion. Serial assessment, on an annual basis, is advised until data from longitudinal studies become available.

**ST Segment Depression**

While ST segment depression is common among patients with cardiomyopathy, it is not a feature of athletic training (Figure 3). Prevalence estimates of ST segment depression among patients with HCM range from 60-70%, and in one study the territorial distribution of ST segment depression among HCM patients was associated with the risk of sudden death or appropriate implantable cardioverter-defibrillator therapy.[41,86,87] In contrast, ST segment depressions are extremely rare in young athletes with structurally normal and physiologically remodeled hearts.[33,55,68,72] 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**

Q waves are defined as any initial negative deflection of the QRS complex and can be found with both physiological electrical activation of the ventricle and with certain pathological conditions, including cardiomyopathy, myocardial infarction, and conduction abnormalities.

Several pathological disorders can lead to the development of exaggerated (deep or wide) Q waves or unexpected Q waves in atypical leads. HCM commonly results in asymmetric septal hypertrophy, which can produce abnormal Q waves due to increased septal forces, septal fibrosis, and asymmetric electrical activation (Figure 14). Indeed, pathological Q waves are

among the most common abnormal ECG findings in HCM and present in 32-42% of patients.[41,69] Pathological Q waves can also result from prior transmural myocardial infarction. Any loss of myocardium and/or loss of myocardial electrical activity due to infarction, infiltration or fibrosis may lead to unopposed electrical activation of the opposite segments. Additionally, early activation from a bypass tract such as in WPW distorts the location of initial ventricular activation and may result in broad or atypical Q waves.

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. This finding is more common in women than men, and in one series 64% of subjects lost this QS pattern on repeat ECG.[88]

A physiological increase in myocardial mass, such as LVH from athletic remodeling, also can result in increased Q wave voltage, and the change in cardiac geometry and electrical axis may distort the location of Q waves slightly. Thus, although Q waves can be a marker of underlying heart conditions, differentiating physiological from pathological Q waves can be challenging.

Pathological Q waves have been reported in approximately 1-2% of all athletes, and may be higher in males and black athletes.[23,89] 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. Long narrow Q waves (> 3 mm in depth) as previously defined by the Seattle criteria have been shown to be an unreliable marker for pathological LVH in athletes with increased QRS voltage. In a report of 13,335 adolescents age 14-18 years screened with ECG, none of the 206 (1.5%) individuals with abnormal Q waves but no other ECG abnormality had pathological findings on limited echocardiogram consistent with cardiomyopathy.[90] Thus, modification of the criteria for pathological Q waves has the potential to greatly improve the specificity of ECG interpretation.

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.[23,24] Therefore, the consensus of this panel based on existing scientific data is to modify 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 a bypass tract, looking for a short PR interval or evidence of a delta wave. 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 a QS pattern should undergo additional evaluation.

Pathological Q waves in two or more contiguous leads warrants further investigation with echocardiography to exclude cardiomyopathy. A detailed family history and assessment of risk

factors for CAD also should occur, especially in athletes age ≥ 30 years as stress testing may be warranted in athletes with suspicion of prior myocardial infarction or multiple risk factors for CAD.

If the echocardiogram is normal and there are no other concerning clinical findings or ECG abnormalities, no additional testing is generally necessary. If pathological Q waves are present along with other ECG abnormalities, such as ST segment depression or TWI, or if suspicious clinical findings are present, additional evaluation with cardiac MRI should be considered.

**Complete Left Bundle Branch Block**

LBBB is found in less than 1 in 1,000 athletes but is common in patients with cardiomyopathy and ischaemic heart disease.[9,41,72,91,92] Thus, complete LBBB always should be considered an abnormal finding and requires a comprehensive evaluation to rule out a pathological cardiac disorder.

LBBB is recognized by a 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 (Figure 15). Q waves are often absent in leads I, V5, and V6, and the ST segment and T waves are usually in the opposite direction than the QRS.[93]

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

**Profound Nonspecific 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.[94,95] The significance of nonspecific IVCD with normal QRS morphology in healthy, asymptomatic athletes is uncertain.[96] The physiology underlying IVCD in athletes remains incompletely understood but likely includes some combination of neurally mediated conduction fiber slowing and increased myocardial mass. In patients with LVH, left ventricular mass seems to be closely related to QRS duration.[97]

While the exact cutoff 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. In asymptomatic athletes with isolated nonspecific IVCD < 140 ms, no further diagnostic evaluation is required. Athletes with cardiovascular symptoms or a concerning family history of sudden death or suspected cardiomyopathy with a nonspecific IVCD ≥ 140 ms or in tandem with other ECG abnormalities should be evaluated further.

Evaluation

In asymptomatic athletes with profound nonspecific 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 (Figure 16).[93] The WPW pattern occurs in approximately 1/1,000 to 4/1,000 athletes.[9,12,66,98] 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 associated with a delta wave is consistent with the WPW pattern and warrants further assessment of the refractory period of the accessory pathway. A short PR interval in isolation without a widened QRS or delta wave in an asymptomatic athlete should not be considered for further assessment.

Asymptomatic athletes with WPW pattern should be investigated for the presence of a low or high risk 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.[99,100] 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.[101] If non-invasive testing cannot confirm a low risk pathway or is inconclusive, electrophysiology testing should be considered to determine the shortest pre-excited RR interval during atrial fibrillation.[99] If the shortest pre-excited RR interval is ≤ 250 ms (240 bpm), then the accessory pathway is deemed high risk.[99,102] Young athletes with a shortest pre-excited RR interval ≤ 250 ms should proceed with transcatheter ablation.[99] An echocardiogram also should be considered due to the association of WPW with Ebstein's anomaly and cardiomyopathy.

**Prolonged QT Interval**

Congenital LQTS is a potentially lethal, genetically mediated ventricular arrhythmia syndrome with the hallmark electrocardiographic feature of QT prolongation. Symptoms if present include arrhythmic syncope, seizures, or aborted cardiac arrest/sudden death stemming from *torsades de pointes* and VF. The pathophysiology of LQTS involves delayed ventricular repolarization originating primarily from loss-of-function mutations in genes encoding voltage gated potassium channels that govern phase 3 repolarization. LQTS is estimated to affect 1 in 2,000 individuals, and this may be underestimated given the subpopulation of so-called "normal QT interval" or "concealed" LQTS.[103]

Autopsy negative sudden unexplained death represents 25-40% of sudden unexpected deaths in persons under age 40 years.[3,104-106] In cases that lack necropsy findings to establish a cause of

death, cardiac ion channelopathies have been implicated by post-mortem genetic testing as the probable cause in up to 25-35% of sudden unexplained death in selected cohorts.[107-109]

Calculating the QTc
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 and even heart rhythm specialists to accurately measure the QTc is suboptimal.[110] However, accurate assessment of the QTc can be achieved by adhering to the following six principles:[111]

1. Most ECG devices utilize the Bazett's heart rate correction formula ($QTc = QT/\sqrt{RR}$; note the RR interval is measured in seconds).[112] Although there are many heart rate correction formulas for the QTc, it is recommended to use Bazett's correction to confirm the computer's QTc as population-based QTc distributions most frequently have used Bazett-derived QTc values.
2. Bazett's formula loses accuracy at slow and fast heart rates; underestimating the inherent QTc at heart rates < 50 bpm, and overestimating the QTc at heart rates > 90 bpm. Accordingly, if an athlete has a heart rate < 50 bpm, a repeat ECG after mild aerobic activity is recommended to achieve a heart rate closer to 60 bpm where the formula is most accurate. Likewise, for heart rates > 90 bpm, repeating the ECG after additional resting time and ensuring the patient is not unduly cold, hot, or anxious may help achieve a lower heart rate where Bazett's formula will be more accurate.
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 to improve accuracy. Importantly, taking the maximum QT interval and dividing it by the square root of the shortest RR interval on the ECG will grossly overestimate the QTc.[113]
4. To properly perform a manual QT measurement, it is critical to identify the end of the T wave since the onset of the QRS is typically seen easily. Leads II and V5 usually provide the best delineation of the T wave, and these leads are often used for the rhythm strip at the bottom of the ECG.
5. Low amplitude U waves, common in the anterior precordial leads, should not be included in the QT calculation. Such U wave inclusion will greatly exaggerate the QTc. Instead, the "Teach-the-Tangent" or "Avoid-the-Tail" method to delineate the end of the T wave should be followed (Figure 17).[111]
6. The morphology of the T wave, not just the length of the QT interval, also can suggest the presence of LQTS.[114] 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.

With this framework, 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 of each other, 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.

QTc Cutoffs
Given the overlap between QTc distributions in population-derived cohorts of healthy individuals compared to patients with genetically confirmed LQTS, it must be acknowledged that no screening program will identify all persons with LQTS.[115-118] Instead, the QTc cutoff value, where the QTc measurement compels further evaluation, must be chosen carefully to balance the frequency of abnormal results and the positive predictive value that LQTS has been detected incidentally.

Published definitions of a "prolonged QTc" requiring further evaluation have varied. Prior guidelines from the ESC for ECG interpretation in athletes define a QTc of > 440 ms in males and > 460 ms in females (but < 500 ms) as a 'grey zone' requiring further evaluation, and a QTc ≥ 500 ms, otherwise unexplained and regardless of family history and symptoms, as indicative of unequivocal LQTS.[5] In the U.S., the AHA/ACC/HRS guideline has dropped the term "borderline" QT prolongation and instead annotates a QTc ≥ 450 ms in men and ≥ 460 ms in women as "prolonged QTc".[119] Concern has been raised that these QTc cutoffs will produce a high number of false-positive test results if followed in a screening population of athletes.[115] More 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 cutoff values are around the 99th percentile and consistent with thresholds defined by the 36th Bethesda Conference.[120] 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*
While an abnormal cutoff for a short QT interval has been included in past ECG interpretation guidelines, the precise cutoff and clinical significance of a short QT interval in athletes is unknown. Data from over 18,000 asymptomatic young British individuals suggests that the prevalence of a QTc < 320 ms is 0.1%; suggesting an abnormal cutoff value of < 320 ms is pragmatic.[121] 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.[121] Based on the rarity of this finding and absence of data to suggest long-term morbidity in asymptomatic athletes, the consensus of this panel recommends that a short QT interval only be investigated in the context of concerning clinical markers such as syncope, premature atrial fibrillation, ventricular arrhythmias, or a relevant family history.

Evaluation
An athlete identified with a prolonged QTc (≥ 470 ms in males; ≥ 480 ms in females) should undergo further evaluation. It is critical that an athlete with a single QTc reading above these threshold values not be obligated a diagnosis of LQTS, but rather that these cutoff values have triggered the need for additional evaluation. The importance of additional evaluation but not a premature diagnosis of LQTS was demonstrated in a study of 2,000 elite athletes in which 7 (0.4%) had a prolonged QTc (range 460-570 ms).[122] 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.[122] As outlined below, personal symptoms, family history, scoring systems, electrocardiographic features, stress ECG and genetic testing may be needed to clarify the diagnosis.

A personal history of exercise/emotion/auditory-triggered syncope or seizures and a family history of exertional syncope, exercise/auditory-triggered "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 a heart rhythm specialist 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 cutoff values, then no additional evaluation is needed and the athlete should be reassured.

On the other hand, if the repeat ECG still exceeds the QTc cutoff 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 a heart rhythm specialist or cardiologist for the possibility of newly discovered LQTS. Reversible, extrinsic factors, such as electrolyte abnormalities (hypokalemia) 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 a heart rhythm specialist or cardiologist as the probability of LQTS and future adverse events has increased.[123] Further testing, such as provocative treadmill stress testing, epinephrine QT stress testing, and genetic testing need to be considered carefully and should be performed and interpreted by a cardiologist familiar with the disease.

LQTS is diagnosed based on a combination of symptoms, family history, electrocardiographic findings, and genetic testing, and the Schwartz-Moss score used to invoke low, intermediate, and high probability.[124-126] Genetic testing for LQTS is recommended for any athlete where a cardiologist has an index of suspicion for LQTS (intermediate or high probability score), or for an asymptomatic patient with no family history but an incidental ECG finding with a QTc ≥ 480 ms pre-puberty and ≥ 500 ms post-puberty that is confirmed on repeat ECG testing.[127]

**Brugada Type 1 Pattern**

Brugada syndrome (BrS) is a primary electrical disease characterized by the distinctive Brugada ECG pattern of "high take-off" ST segment elevation in the right precordial leads (Figure 6). Although three types were described, only the type 1 Brugada pattern is now considered diagnostic.[128-130] BrS predisposes to ventricular tachyarrhythmias in the absence of clinically demonstrable structural heart disease, and sudden death in patients with BrS occurs more often during states of enhanced vagal tone.[131] Loss of function mutations in the sodium channel gene SCN5A accounts for up to 20% of cases.[128,132] BrS is estimated to cause up to 4% of all sudden deaths in the general population and 5-20% of sudden unexplained deaths with a structurally normal heart at autopsy.[128,133]

The type 1 Brugada pattern 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. Classic type 2 and 3 Brugada patterns have a "saddleback" appearance with J-point elevation ≥ 2 mm, ST segment elevation > 1 mm in type 2 and ≤ 1 mm in type 3, and either a positive or biphasic T wave. A recent consensus document grouped the classic types 2 and 3 as type 2.[129]

Distinguishing between true type 1 or 2 patterns and a host of Brugada-like ECG patterns can be challenging. Brugada-like patterns may be caused by both physiological (normal variant, early repolarization, incomplete RBBB) and pathological (ARVC, pulmonary hypertension, hyperkalemia) conditions.[129,134] Pharmacologic and electrocardiographic maneuvers may clarify the diagnosis. Confirmation of proper precordial lead placement is paramount, as high placement of the V1 and V2 electrodes in the 2nd and 3rd intercostal space (rather than the 4th intercostal space) can accentuate a type 1 Brugada ECG pattern in known Brugada patients but also produce type 2 like patterns in athletes.[135]

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).[136] 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 18).

Evaluation
The type I Brugada ECG pattern is not a recognized variant of athlete's heart and should raise the possibility of a sodium ion channelopathy. Patients with a type 1 ECG pattern should be referred to a cardiac electrophysiologist for further evaluation, 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 high precordial lead ECG, then referral to a heart rhythm specialist is indicated. Consideration should be given to potential accentuating factors for a Brugada-like ECG pattern, such as hyperkalemia, fever, medications with sodium ion channel blocking properties, and lead placement.

The type 2 Brugada ECG pattern may overlap with repolarization changes in the anterior leads in black athletes and endurance athletes. Multiple criteria for evaluating type 2 patterns have been proposed; however, in the absence of symptoms or a concerning family history, athletes with type 2 patterns do not require further testing.

**Profound Sinus Bradycardia**

Sinus bradycardia is a hallmark feature of athletic conditioning with heart rates commonly between 40-60 bpm or even slower. A resting heart rate ≤ 30 bpm or a sinus pause ≥ 3 seconds may be normal in a well-trained athlete but nevertheless should prompt further evaluation.

Evaluation

Evaluation of profound sinus bradycardia should include assessing the chronotropic response to mild aerobic activity, such as running on the spot or climbing stairs. If the heart rate increases appropriately and the athlete is asymptomatic, no further testing is necessary. If there is no change in heart rate or in athletes with a history of symptoms such as pre-syncope or syncope, further testing should be performed to exclude primary sinus node disease. Exercise testing is useful in this situation to provide an objective measure of the heart rate response to aerobic activity.

**Profound First Degree Heart Block**

Mild to moderate first degree heart block with a PR interval of 200 to 399 ms may be present in athletes due to increased vagal tone. A PR interval ≥ 400 ms is significantly prolonged and requires further evaluation.

Evaluation
A small amount of aerobic activity should be performed to assess if the PR interval shortens appropriately. If the PR interval normalizes with exercise, the PR prolongation is due to vagal mechanisms and is benign. If the PR interval does not shorten, or there are symptoms of syncope or a family history of cardiac disease or sudden death, further evaluation should be performed.[137] An exercise test can demonstrate more clearly if the PR interval changes at higher heart rates. Depending on the clinical scenario, an echocardiogram or ambulatory ECG monitor may be indicated.

**High Grade AV Block**

Mobitz type II second degree AV block and third degree (complete) AV block are pathological disruptions in AV conduction and abnormal findings in athletes. In Mobitz type II AV block, there are intermittently non-conducted P waves with a fixed PR interval. In complete AV block, there are more P waves than QRS complexes, and the ventricular rhythm is regular due to an undisturbed junctional or ventricular pacemaker. 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. Complete heart block requires further evaluation for underlying cardiac disease.

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 a heart rhythm specialist is essential.

**Multiple Premature Ventricular Contractions**

Multiple ($\geq$ 2) premature ventricular contractions (PVCs) are uncommon and present in < 1% of athlete ECGs (Figure 19).[9,12] When 2 or more PVCs are recorded on a standard (10 second) ECG, it is possible the athlete has a high 24 hour PVC burden. While multiple PVCs are most likely benign in a highly trained athlete, their presence may be the hallmark of underlying heart disease.[138] In athletes with $\geq$ 2,000 PVCs per 24 hours, up to 30% were found to have underlying structural heart disease, in contrast to 3% and 0% in those with < 2,000 and < 100 PVCs per day, respectively.[139] Over half of the athletes with $\geq$ 2,000 PVCs also had bursts of non-sustained ventricular tachycardia.[139] Therefore, the finding of $\geq$ 2 PVCs on an ECG should prompt more extensive evaluation to exclude underlying structural heart disease.

Evaluation

The extent of evaluation for $\geq$ 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. However, in athletes with $\geq$ 2,000 PVCs per 24 hours 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 EP study.[140] Therefore, many such cases require referral to a specialist center.[141] Although some studies have suggested that regression of the PVC burden with detraining indicates a good prognosis, other studies have not confirmed this.[142-144] Thus, detraining as a diagnostic or therapeutic measure is not recommended.

*Considerations in High-Dynamic Athletes*

In high-level endurance athletes, such as cyclists, triathlon athletes, and rowers, concern has been raised about the high volume and pressure loads on the right ventricle that could promote or induce ARVC. Competitive sport definitively accelerates and enhances the phenotypic expression of desmosomal mutations causing ARVC.[145,146] There also is evolving evidence that high-dynamic exercise sustained over a long period of time may result in 'exercise-induced' ARVC, even in the absence of desmosomal mutations or a familial history.[141,147-149] PVCs originating from the right ventricular outflow tract conduct with LBBB and an inferior axis and are usually regarded as benign. In contrast, PVCs originating from the main body of the right ventricle typically show a LBBB pattern and superior axis (predominantly negative QRS vector in V1 and the inferior leads) and may be associated with right ventricular pathology (Figure 19). Therefore, a lower threshold for extensive evaluation of a single PVC may be warranted in high-dynamic athletes, especially when $\geq$ 25 years of age and with a LBBB morphology and superior axis.

**Atrial Tachyarrhythmias**

Atrial tachyarrhythmias, comprised of supraventricular tachycardia (SVT), atrial fibrillation, and atrial flutter, are heart rhythms > 100 bpm which originate in the sinus node, atrial tissue, or AV node. Sinus tachycardia is the most common atrial tachyarrhythmia but is very rarely due to intrinsic cardiac disease.

SVT, atrial fibrillation, and atrial flutter are rarely seen on a resting ECG in athletes and require investigation. One study demonstrated just 6 of 32,561 young athletes during ECG screening with an atrial tachyarrhythmia, four (0.01%) with ectopic atrial tachycardia and two (<0.01%) with atrial fibrillation.[9] An examination of ECGs from the South Korean Army also found zero soldiers with an atrial tachyarrhythmia.[150] Atrial tachyarrhythmias are rarely life-threatening and usually lead to symptoms like palpitations, shortness of breath, chest pressure, dizziness, neck pounding, or syncope from rapid heart rates. AV dyssynchrony also may limit peak physical activity. More importantly, they can be associated with other conditions that can lead to SCD, including LQTS, WPW, BrS, myocarditis, congenital heart disease, and any form of cardiomyopathy.

Evaluation

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 to assess whether the SVT terminates with a P wave or QRS complex, which can help elucidate the mechanism of the SVT. An echocardiogram, ambulatory ECG monitor, and exercise treadmill test should be completed, and referral to a heart rhythm specialist 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.[151] 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. An exercise treadmill test also should be completed to assess the maximum heart rate in atrial fibrillation. 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.

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 etiologies may be sought, including fever, infection, dehydration, stimulant use, anemia, hyperthyroidism, or, rarely, underlying cardiac or pulmonary disease.

**Ventricular Arrhythmias**

Ventricular couplets, triplets, and non-sustained ventricular tachycardia always require investigation. Alone, these are not life-threatening arrhythmias, but can be a marker for underlying cardiac pathology or lead to sustained ventricular tachycardia which may cause SCD. These ventricular arrhythmias may be idiopathic or secondary to the cardiomyopathies, ion channelopathies, or other diseases such as myocarditis, myocardial infarction, or sarcoidosis.

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.[105,106] The number of older, masters athletes at all levels of competition is increasing worldwide. In addition, older athletes may be less fit compared to 20-30 years ago, increasing the possibility of underlying CAD.[152] In a cohort study of Swedish competitive cross-country runners with a mean age of 60 years, 2% were shown to have severe underlying cardiovascular disease, mainly CAD.[153] While the overall health benefits of regular exercise and physical activity are unequivocal, exercise may be a trigger for acute cardiac events in individuals with underlying and silent CAD. The risk of an acute cardiovascular event during exercise seems to be U-shaped, with the greatest risk conferred in those who are physically inactive but also in those exercising at the highest intensity. Therefore, it is important to consider relevant changes on a resting ECG that may represent occult CAD in asymptomatic athletes ≥ 30 years of age.

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 right bundle branch block, abnormal R wave progression, left anterior hemiblock, and atrial fibrillation.[154-156] Thus, the major role of a resting ECG in screening for CAD may be in older athletes generally considered to be at an intermediate risk, where an abnormal ECG would place them into a high risk group warranting further testing.[155,157,158]

<u>Evaluation</u>
Additional evaluation for underlying CAD should be considered in asymptomatic older athletes with TWI, pathological Q waves, ST segment depression, left or right bundle branch block, abnormal R wave progression, left anterior hemiblock, and atrial fibrillation. Initial testing should normally 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.

**ECG Patterns Requiring Serial Follow-up**

All abnormal ECG findings discussed in this document should prompt a comprehensive evaluation for underlying structural and/or electrical heart disease. The specific components of this evaluation should be individualized to the athlete based on the presence or absence of symptoms and relevant family history and on the specific attributes of the abnormal ECG. While the abnormal findings delineated in this document were chosen to maximize specificity for true underlying disease and therefore minimize the likelihood of false positive ECG testing, no single ECG abnormality has perfect accuracy. Further, ECG abnormalities may precede the development of overt structural heart disease in athletes with a genetic predisposition to cardiomyopathy.[41,80,81] Athletes who demonstrate one or more overtly abnormal ECG findings may therefore undergo comprehensive clinical evaluations that reveal no definitive evidence of true pathology. In these cases, the clinical significance of the presenting abnormal ECG remains uncertain.

Several common heritable cardiomyopathies including HCM, ARVC, and familial DCM may present with ECG abnormalities prior to the onset of overt heart muscle pathology. The natural history of athletes with markedly abnormal ECGs but structurally normal hearts on initial evaluation was first documented in a study of 81 athletes in which 6% (5/81) developed overt cardiomyopathy (HCM, n=3; ARVC, n=1; DCM, n=1) during an average follow-up period of 9 years.[80] In a more recent series of athletes with abnormal ECG findings, 6% (5/85) with initially unrevealing clinical evaluations developed features consistent with cardiomyopathy (HCM, n=2; ARVC, n=1; arrhythmic event, n=2) during a 12 month follow-up period.[81]

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. Specifically, asymptomatic athletes with abnormal TWI, ST segment depression, pathological Q waves, LBBB, and profound IVCD (QRS duration $\geq$ 140 ms) require serial cardiac imaging to rule out the development of a cardiomyopathic disease (Figure 12). These athletes may be permitted to participate in competitive athletics without restriction contingent on longitudinal follow-up. Follow-up assessments should include a medical history to ascertain new cardiovascular symptoms or otherwise unexplained decrements in exercise tolerance, a physical examination, and non-invasive cardiac imaging as deemed suitable for the appropriate suspected form of disease. These evaluations should be conducted on an annual basis while participating in competitive athletics, or more frequently as determined on an individual basis. Athletes should be educated about the importance of longitudinal surveillance to promote ownership of the process if they change teams, medical staff, or locations.

**Temporary Restriction during Secondary Evaluation of ECG Abnormalities**

Temporary restriction from athletic activity should be considered for athletes with abnormal ECGs of uncertain clinical significance until secondary investigations are completed. While the absolute risk of suffering an adverse cardiac event attributable to underlying structural or electrical heart disease is low while awaiting definitive evaluation, the risk is not negligible. Although this conservative approach is accepted commonly for an athlete with symptoms, the asymptomatic athlete found to have an abnormal ECG during pre-participation screening presents other challenges. Athletes in this situation often have limited time to participate in team based tryouts, and sport restriction may jeopardize their athletic eligibility or generate pressure from coaches, managers, parents or the athletes themselves. To circumvent some of these challenges, proper advanced planning prior to pre-participation screening is required to establish avenues for prompt secondary testing and consultation for ECG abnormalities. If ECG is included in the cardiovascular screening of athletes, it must be conducted with adequate cardiology oversight and resources to assist with the secondary investigation of ECG abnormalities. Ideally, pre-participation screening should be performed at a time point that permits athletes to undergo additional cardiovascular testing prior to the onset of organized competitive athletics. The optimal duration of time between pre-participation screening and the beginning of organized team based training will vary as a function of local resources.

**Considerations in the Symptomatic Athlete and in the Athlete with a Family History of Sudden Death or Hereditary Cardiovascular Disease**

Symptoms suggestive of underlying cardiovascular disease including exertional chest discomfort, inappropriate exertional dyspnea, unexplained/unheralded syncope, unexplained seizure-like activity, and exertional palpitations, particularly when coupled with abnormal ECG findings, are strongly suggestive of a pathological process. In the symptomatic athlete either with or without an abnormal ECG, comprehensive testing should be tailored to the presenting symptoms and performed in a timely fashion. Asymptomatic athletes with a family history of sudden death or a heritable cardiomyopathy or ion channelopathy may similarly carry an increased risk of true occult disease.[159,160] Great care must be taken to confirm the nature of the family history as athletes may have incomplete or erroneous information about their relatives. In the evaluation of an athlete with a compelling family history, genetic testing permits a more definitive assessment of individual and familial risk for specific disease processes such as HCM, ARVC, and congenital LQTS. The use of genetic testing and its interpretation should be performed by a cardiovascular specialist with expertise in clinical genetics.

**Psychological Considerations of Caring for the Athlete with Potentially Lethal Cardiac Disease**

Athletes diagnosed with serious cardiac diseases, regardless of the method used for disease detection, are at risk for psychological morbidity and represent an emotionally vulnerable population.[161] Risk factors for increased psychological morbidity include a higher level of competition, complete disqualification from athletic competition, daily reminders of disease (i.e. medication or implantable cardioverter defibrillator), and unanticipated outcomes (i.e. failed cardiac procedure).

Informing an athlete that he/she has an underlying cardiac disorder that may affect sports participation is difficult. In fact, many athletes may not completely comprehend the nature of their disease, and several follow-up discussions are likely necessary. Assessment of emotional well-being should be a standard part of all follow-up evaluations, and psychological support for the athlete may be enhanced by a multi-disciplinary approach. In cases where competitive sport is not recommended, additional measures should consider helping the athlete refocus away from athletics, such as excelling in school or becoming involved in other team-related activities (i.e. coaching). Athletes disqualified from competitive athletics should be offered exercise recommendations thought to be safer to foster long-term physical and mental health. While exercise guidelines for individuals with inherited cardiac disorders exist, more research is needed to fully understand safe, disease-specific physical activity recommendations.[162,163]

**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. Cardiac adaptation and remodeling from regular athletic training produce common ECG alterations that could be mistaken as abnormal. Whether performed for screening or diagnostic purposes, it is critical that physicians responsible for the cardiovascular care of athletes be guided by ECG interpretation standards that improve disease detection and limit false-positive results. 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.

**FIGURE LEGENDS**

Figure 1.  International Consensus Standards for Electrocardiographic Interpretation in Athletes

Figure 2.  ECG of a 29 year old male asymptomatic soccer player showing sinus bradycardia (44 bpm), early repolarization in I, II, aVF, V2-V6 (arrows), voltage criterion for LVH (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. ECG from a patient with HCM demonstrating QRS voltage criterion for LVH in association with deep TWI and ST segment depression predominantly in the lateral leads (I, aVL, V4-V6), voltage criterion for left atrial and right atrial enlargement, and LAD.

Figure 4.  ECG demonstrates incomplete RBBB with rSR' pattern in V1 and QRS duration of < 120 ms. Incomplete RBBB is a common and normal finding in athletes and does not require additional evaluation.

Figure 5.  ECG from a black athlete demonstrating voltage criterion for LVH, and convex ('domed') ST segment elevation followed by TWI in V1-V4 (circles). This is a normal repolarization pattern in black athletes.

Figure 6.  Normal and abnormal patterns of TWI. (A) Anterior TWI in V1-V3 in a 12 year old asymptomatic athlete without a family history of SCD considered a normal "juvenile" pattern. (B) TWI in V1-V4 in a 17 year old asymptomatic mixed race (Middle-Eastern/black) athlete without a family history of SCD. This is a normal repolarization pattern in black athletes. (C) Biphasic TWI in V3 in a 31 year old asymptomatic black athlete without a family history of SCD. 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 TWI in V1-V6 in an adult symptomatic former soccer player with genetically confirmed ARVC and a positive family history of SCD (brother died at 26 years of age). (E) An ECG 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. (F) Inferolateral TWI 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.

Figure 7.  A 28 year old asymptomatic Caucasian handball player demonstrating a junctional escape rhythm (red arrows). Note the constant RR interval between beats.

Figure 8.  ECG shows an ectopic atrial rhythm. The atrial rate is 63 beats per minute and the P wave morphology is negative in leads II, III, and aVF (arrows), signifying an ectopic atrial rhythm.

Figure 9.  ECG shows Mobitz Type I (Wenckebach) 2nd degree AV block demonstrated by progressively longer PR intervals until there is a non-conducted P-wave (arrows) and no QRS

complex. Note the first PR interval after the dropped beat is shorter than the last conducted PR interval prior to the dropped beat.

Figure 10.  ECG from an asymptomatic 22 year old black male athlete demonstrating complete RBBB (QRS ≥ 120 ms), LAD (-57°), and RAE (P wave ≥ 2.5 mm in II and aVF). The presence of two or more borderline ECG findings warrants additional investigation to exclude pathologic cardiac disease.

Figure 11.  ECG from a 30 year old patient with ARVC showing anterior TWI in V1-V3 preceded by a flat or downsloping ST segment without J-point elevation. PVCs are also present.

Figure 12.  Panel A: ECG from an 18 year old black basketball player demonstrating abnormal TWI extending into V5. Initial cardiac imaging was non-diagnostic. Panel B: ECG from the same athlete at age 20 showing abnormal TWI in the inferolateral leads with the development of deep TWI and ST segment depression in V4-V6. Follow-up cardiac MRI demonstrated distinct findings of apical HCM with a maximum left ventricular wall thickness of 21 mm with small foci of LGE. Athletes with TWI in V5 and/or V6 require serial evaluation for the development of cardiomyopathy.

Figure 13.  Examples of physiological (A) and pathological TWI (B). Panel A demonstrates TWI in V1-V4 preceded by convex 'domed' ST segment elevation (green circles). This should not be confused with pathological TWI (Panel B) which demonstrates TWI in V1-V6 with absent J-point elevation and a downsloping ST segment (red circles).

Figure 14.  ECG from an 18 year old female swimmer demonstrating deep and wide pathological Q waves in V4-V6, I and aVL. Diagnostic testing revealed HCM.

Figure 15.  ECG with complete LBBB demonstrating a QRS ≥ 120 ms, predominantly negative QRS complex in lead V1, upright R wave in leads I and V6, and ST segments and T waves in the opposite direction of the QRS. LBBB is always an abnormal finding in athletes and warrants a comprehensive evaluation to exclude myocardial disease.

Figure 16.  ECG demonstrating the classic findings of WPW pattern with a short PR interval (< 120 ms), delta wave (slurred QRS upstroke), and prolonged QRS (> 120 ms).

Figure 17.  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.

Figure 18.  Brugada type 1 ECG (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.

Figure 19.  ECG from a patient with ARVC.  Note multiple PVCs with a LBBB pattern and superior axis (negative QRS vector in inferior leads).

**REFERENCES**

1.      Harmon KG, Asif IM, Klossner D, Drezner JA. Incidence of sudden cardiac death in national collegiate athletic association athletes. *Circulation.* 2011;123(15):1594-1600.
2.      Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. *Circulation.* 2009;119(8):1085-1092.
3.      Harmon KG, Asif IM, Maleszewski JJ, et al. Incidence, Etiology, and Comparative Frequency of Sudden Cardiac Death in NCAA Athletes: A Decade in Review. *Circulation.* 2015.
4.      Corrado D, Biffi A, Basso C, Pelliccia A, Thiene G. 12-lead ECG in the athlete: physiological versus pathological abnormalities. *Br J Sports Med.* 2009;43(9):669-676.
5.      Corrado D, Pelliccia A, Heidbuchel H, et al. Recommendations for interpretation of 12-lead electrocardiogram in the athlete. *Eur Heart J.* 2010;31(2):243-259.
6.      Uberoi A, Stein R, Perez MV, et al. Interpretation of the electrocardiogram of young athletes. *Circulation.* 2011;124(6):746-757.
7.      Williams ES, Owens DS, Drezner JA, Prutkin JM. Electrocardiogram interpretation in the athlete. *Herzschrittmacherther Elektrophysiol.* 2012;23(2):65-71.
8.      Drezner J. Standardised criteria for ECG interpretation in athletes: a practical tool. *Br J Sports Med.* 2012;46(Suppl I): i6-i8.
9.      Marek J, Bufalino V, Davis J, et al. Feasibility and findings of large-scale electrocardiographic screening in young adults: data from 32,561 subjects. *Heart Rhythm.* 2011;8(10):1555-1559.
10.     Drezner JA, Ackerman MJ, Anderson J, et al. Electrocardiographic interpretation in athletes: the 'Seattle criteria'. *Br J Sports Med.* 2013;47(3):122-124.
11.     Corrado D, Pelliccia A, Bjornstad HH, et al. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J.* 2005;26(5):516-524.
12.     Pelliccia A, Culasso F, Di Paolo FM, et al. Prevalence of abnormal electrocardiograms in a large, unselected population undergoing pre-participation cardiovascular screening. *Eur Heart J.* 2007;28(16):2006-2010.
13.     Corrado D, Basso C, Pavei A, Michieli P, Schiavon M, Thiene G. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. *JAMA.* 2006;296(13):1593-1601.
14.     Drezner JA, Asif IM, Owens DS, et al. Accuracy of ECG interpretation in competitive athletes: the impact of using standised ECG criteria. *Br J Sports Med.* 2012;46(5):335-340.
15.     Drezner JA, Fischbach P, Froelicher V, et al. Normal electrocardiographic findings: recognising physiological adaptations in athletes. *Br J Sports Med.* 2013;47(3):125-136.
16.     Drezner JA, Ashley E, Baggish AL, et al. Abnormal electrocardiographic findings in athletes: recognising changes suggestive of cardiomyopathy. *Br J Sports Med.* 2013;47(3):137-152.

17. Drezner JA, Ackerman MJ, Cannon BC, et al. Abnormal electrocardiographic findings in athletes: recognising changes suggestive of primary electrical disease. *Br J Sports Med.* 2013;47(3):153-167.

18. Brosnan M, La Gerche A, Kalman J, et al. The Seattle Criteria increase the specificity of preparticipation ECG screening among elite athletes. *Br J Sports Med.* 2013.

19. Price DE, McWilliams A, Asif IM, et al. Electrocardiography-inclusive screening strategies for detection of cardiovascular abnormalities in high school athletes. *Heart Rhythm.* 2014;11(3):442-449.

20. Pickham D, Zarafshar S, Sani D, Kumar N, Froelicher V. Comparison of three ECG criteria for athlete pre-participation screening. *J Electrocardiol.* 2014;47(6):769-774.

21. Zaidi A, Ghani S, Sheikh N, et al. Clinical significance of electrocardiographic right ventricular hypertrophy in athletes: comparison with arrhythmogenic right ventricular cardiomyopathy and pulmonary hypertension. *Eur Heart J.* 2013;34(47):3649-3656.

22. Gati S, Sheikh N, Ghani S, et al. Should axis deviation or atrial enlargement be categorised as abnormal in young athletes? The athlete's electrocardiogram: time for re-appraisal of markers of pathology. *Eur Heart J.* 2013;34(47):3641-3648.

23. Sheikh N, Papadakis M, Ghani S, et al. Comparison of electrocardiographic criteria for the detection of cardiac abnormalities in elite black and white athletes. *Circulation.* 2014;129(16):1637-1649.

24. Riding NR, Sheikh N, Adamuz C, et al. Comparison of three current sets of electrocardiographic interpretation criteria for use in screening athletes. *Heart.* 2014;101(5):384-390.

25. Exeter DJ, Elley CR, Fulcher ML, Lee AC, Drezner JA, Asif IM. Standardised criteria improve accuracy of ECG interpretation in competitive athletes: a randomised controlled trial. *Br J Sports Med.* 2014;48(15):1167-1171.

26. Brosnan M, La Gerche A, Kumar S, Lo W, Kalman J, Prior D. Modest agreement in ECG interpretation limits the application of ECG screening in young athletes. *Heart Rhythm.* 2015;12(1):130-136.

27. Magee C, Kazman J, Haigney M, et al. Reliability and validity of clinician ECG interpretation for athletes. *Ann Noninvasive Electrocardiol.* 2014;19(4):319-329.

28. Hill AC, Miyake CY, Grady S, Dubin AM. Accuracy of Interpretation of Preparticipation Screening Electrocardiograms. *J Pediatr.* 2011;159(5):783-788.

29. Hancock EW, Deal BJ, Mirvis DM, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part V: electrocardiogram changes associated with cardiac chamber hypertrophy: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society: endorsed by the International Society for Computerized Electrocardiology. *Circulation.* 2009;119(10):e251-261.

30. Sohaib SM, Payne JR, Shukla R, World M, Pennell DJ, Montgomery HE. Electrocardiographic (ECG) criteria for determining left ventricular mass in young healthy men; data from the LARGE Heart study. *J Cardiovasc Magn Reson.* 2009;11:2.

31. Pelliccia A, Maron BJ, Culasso F, et al. Clinical significance of abnormal electrocardiographic patterns in trained athletes. *Circulation.* 2000;102(3):278-284.

32. Papadakis M, Basavarajaiah S, Rawlins J, et al. Prevalence and significance of T-wave inversions in predominantly Caucasian adolescent athletes. *Eur Heart J.* 2009;30(14):1728-1735.

33. Papadakis M, Carre F, Kervio G, et al. The prevalence, distribution, and clinical outcomes of electrocardiographic repolarization patterns in male athletes of African/Afro-Caribbean origin. *Eur Heart J.* 2011;32(18):2304-2313.

34. Riding NR, Salah O, Sharma S, et al. ECG and morphologic adaptations in Arabic athletes: are the European Society of Cardiology's recommendations for the interpretation of the 12-lead ECG appropriate for this ethnicity? *Br J Sports Med.* 2013.

35. Sharma S, Whyte G, Elliott P, et al. Electrocardiographic changes in 1000 highly trained junior elite athletes. *Br J Sports Med.* 1999;33(5):319-324.

36. Huston TP, Puffer JC, Rodney WM. The athletic heart syndrome. *N Engl J Med.* 1985;313(1):24-32.

37. Ryan MP, Cleland JG, French JA, et al. The standard electrocardiogram as a screening test for hypertrophic cardiomyopathy. *Am J Cardiol.* 1995;76(10):689-694.

38. Sathanandam S, Zimmerman F, Davis J, Marek J. Abstract 2484: ECG Screening Criteria for LVH Does Not Correlate With Diagnosis of Hypertrophic Cardiomyopathy. *Circulation.* 2009(120):S647.

39. Weiner RB, Hutter AM, Wang F, et al. Performance of the 2010 European Society of Cardiology criteria for ECG interpretation in the athlete. *Heart.* 2011;97:1573-1577.

40. Calore C, Melacini P, Pelliccia A, et al. Prevalence and clinical meaning of isolated increase of QRS voltages in hypertrophic cardiomyopathy versus athlete's heart: relevance to athletic screening. *Int J Cardiol.* 2013;168(4):4494-4497.

41. Lakdawala NK, Thune JJ, Maron BJ, et al. Electrocardiographic features of sarcomere mutation carriers with and without clinically overt hypertrophic cardiomyopathy. *Am J Cardiol.* 2011;108(11):1606-1613.

42. Kim JH, Noseworthy PA, McCarty D, et al. Significance of electrocardiographic right bundle branch block in trained athletes. *Am J Cardiol.* 2011;107(7):1083-1089.

43. Tikkanen JT, Anttonen O, Junttila MJ, et al. Long-term outcome associated with early repolarization on electrocardiography. *N Engl J Med.* 2009;361(26):2529-2537.

44. Haissaguerre M, Derval N, Sacher F, et al. Sudden cardiac arrest associated with early repolarization. *N Engl J Med.* 2008;358(19):2016-2023.

45. Macfarlane PW, Antzelevitch C, Haissaguerre M, et al. The Early Repolarization Pattern: A Consensus Paper. *J Am Coll Cardiol.* 2015;66(4):470-477.

46. Tikkanen JT, Junttila MJ, Anttonen O, et al. Early repolarization: electrocardiographic phenotypes associated with favorable long-term outcome. *Circulation.* 2011;123(23):2666-2673.

47. Uberoi A, Jain NA, Perez M, et al. Early repolarization in an ambulatory clinical population. *Circulation.* 2011;124(20):2208-2214.

48. Junttila MJ, Sager SJ, Freiser M, McGonagle S, Castellanos A, Myerburg RJ. Inferolateral early repolarization in athletes. *J Interv Card Electrophysiol.* 2011;31(1):33-38.

49. Noseworthy PA, Weiner R, Kim J, et al. Early repolarization pattern in competitive athletes: clinical correlates and the effects of exercise training. *Circ Arrhythm Electrophysiol.* 2011;4(4):432-440.

50.   Noseworthy PA, Tikkanen JT, Porthan K, et al. The early repolarization pattern in the general population: clinical correlates and heritability. *J Am Coll Cardiol.* 2011;57(22):2284-2289.

51.   Rosso R, Kogan E, Belhassen B, et al. J-point elevation in survivors of primary ventricular fibrillation and matched control subjects: incidence and clinical significance. *J Am Coll Cardiol.* 2008;52(15):1231-1238.

52.   Brosnan MJ, Kumar S, LaGerche A, et al. Early repolarization patterns associated with increased arrhythmic risk are common in young non-Caucasian Australian males and not influenced by athletic status. *Heart Rhythm.* 2015;12(7):1576-1583.

53.   Quattrini FM, Pelliccia A, Assorgi R, et al. Benign clinical significance of J-wave pattern (early repolarization) in highly trained athletes. *Heart Rhythm.* 2014;11(11):1974-1982.

54.   Sheikh N, Papadakis M, Carre F, et al. Cardiac adaptation to exercise in adolescent athletes of African ethnicity: an emergent elite athletic population. *Br J Sports Med.* 2013;47(9):585-592.

55.   Di Paolo FM, Schmied C, Zerguini YA, et al. The athlete's heart in adolescent Africans: an electrocardiographic and echocardiographic study. *J Am Coll Cardiol.* 2012;59(11):1029-1036.

56.   Rawlins J, Carre F, Kervio G, et al. Ethnic differences in physiological cardiac adaptation to intense physical exercise in highly trained female athletes. *Circulation.* 2010;121(9):1078-1085.

57.   Migliore F, Zorzi A, Michieli P, et al. Prevalence of cardiomyopathy in Italian asymptomatic children with electrocardiographic T-wave inversion at preparticipation screening. *Circulation.* 2012;125(3):529-538.

58.   Calo L, Sperandii F, Martino A, et al. Echocardiographic findings in 2261 peri-pubertal athletes with or without inverted T waves at electrocardiogram. *Heart.* 2015;101(3):193-200.

59.   Stein R, Medeiros CM, Rosito GA, Zimerman LI, Ribeiro JP. Intrinsic sinus and atrioventricular node electrophysiologic adaptations in endurance athletes. *J Am Coll Cardiol.* 2002;39(6):1033-1038.

60.   Northcote RJ, Canning GP, Ballantyne D. Electrocardiographic findings in male veteran endurance athletes. *Br Heart J.* 1989;61(2):155-160.

61.   Meytes I, Kaplinsky E, Yahini JH, Hanne-Paparo N, Neufeld HN. Wenckebach A-V block: a frequent feature following heavy physical training. *Am Heart J.* 1975;90(4):426-430.

62.   Prior DL, La Gerche A. The athlete's heart. *Heart.* 2012;98(12):947-955.

63.   Brosnan M, La Gerche A, Kalman J, et al. Comparison of frequency of significant electrocardiographic abnormalities in endurance versus nonendurance athletes. *Am J Cardiol.* 2014;113(9):1567-1573.

64.   Wasfy MM, DeLuca J, Wang F, et al. ECG findings in competitive rowers: normative data and the prevalence of abnormalities using contemporary screening recommendations. *Br J Sports Med.* 2015;49(3):200-206.

65.   Bessem B, de Bruijn MC, Nieuwland W. The ECG of high-level junior soccer players: comparing the ESC vs. the Seattle criteria. *Br J Sports Med.* 2014;49(15):1000-1006.

66.   Fudge J, Harmon KG, Owens DS, et al. Cardiovascular screening in adolescents and young adults: a prospective study comparing the Pre-participation Physical Evaluation Monograph 4th Edition and ECG. *Br J Sports Med.* 2014;48(15):1172-1178.

67.     Magalski A, McCoy M, Zabel M, et al. Cardiovascular screening with electrocardiography and echocardiography in collegiate athletes. *Am J Med.* 2011;124(6):511-518.

68.     Baggish AL, Hutter AM, Jr., Wang F, et al. Cardiovascular screening in college athletes with and without electrocardiography: A cross-sectional study. *Ann Intern Med.* 2010;152(5):269-275.

69.     Rowin EJ, Maron BJ, Appelbaum E, et al. Significance of false negative electrocardiograms in preparticipation screening of athletes for hypertrophic cardiomyopathy. *Am J Cardiol.* 2012;110(7):1027-1032.

70.     Chen X, Zhao T, Lu M, et al. The relationship between electrocardiographic changes and CMR features in asymptomatic or mildly symptomatic patients with hypertrophic cardiomyopathy. *Int J Cardiovasc Imaging.* 2014;30 Suppl 1:55-63.

71.     Sheikh N, Papadakis M, Schnell F, et al. Clinical Profile of Athletes With Hypertrophic Cardiomyopathy. *Circ Cardiovasc Imaging.* 2015;8(7):e003454.

72.     Bent RE, Wheeler MT, Hadley D, et al. Systematic Comparison of Digital Electrocardiograms From Healthy Athletes and Patients With Hypertrophic Cardiomyopathy. *J Am Coll Cardiol.* 2015;65(22):2462-2463.

73.     Nasir K, Bomma C, Tandri H, et al. Electrocardiographic features of arrhythmogenic right ventricular dysplasia/cardiomyopathy according to disease severity: a need to broaden diagnostic criteria. *Circulation.* 2004;110(12):1527-1534.

74.     Marcus FI, McKenna WJ, Sherrill D, et al. Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia: proposed modification of the task force criteria. *Circulation.* 2010;121(13):1533-1541.

75.     Link MS, Laidlaw D, Polonsky B, et al. Ventricular arrhythmias in the North American multidisciplinary study of ARVC: predictors, characteristics, and treatment. *J Am Coll Cardiol.* 2014;64(2):119-125.

76.     Saguner AM, Ganahl S, Kraus A, et al. Electrocardiographic features of disease progression in arrhythmogenic right ventricular cardiomyopathy/dysplasia. *BMC Cardiovasc Disord.* 2015;15:4.

77.     Bjornstad H, Storstein L, Meen HD, Hals O. Electrocardiographic findings of repolarization in athletic students and control subjects. *Cardiology.* 1994;84(1):51-60.

78.     Wilson MG, Chatard JC, Carre F, et al. Prevalence of electrocardiographic abnormalities in West-Asian and African male athletes. *Br J Sports Med.* 2012;46(5):341-347.

79.     Chandra N, Bastiaenen R, Papadakis M, et al. Prevalence of electrocardiographic anomalies in young individuals: relevance to a nationwide cardiac screening program. *J Am Coll Cardiol.* 2014;63(19):2028-2034.

80.     Pelliccia A, Di Paolo FM, Quattrini FM, et al. Outcomes in athletes with marked ECG repolarization abnormalities. *N Engl J Med.* 2008;358(2):152-161.

81.     Schnell F, Riding N, O'Hanlon R, et al. Recognition and significance of pathological T-wave inversions in athletes. *Circulation.* 2015;131(2):165-173.

82.     Maron MS, Maron BJ, Harrigan C, et al. Hypertrophic cardiomyopathy phenotype revisited after 50 years with cardiovascular magnetic resonance. *J Am Coll Cardiol.* 2009;54(3):220-228.

83.     Authors/Task Force m, Elliott PM, Anastasakis A, et al. 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy: the Task Force for the

Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). *Eur Heart J.* 2014;35(39):2733-2779.

84. Calore C, Zorzi A, Sheikh N, et al. Electrocardiographic anterior T-wave inversion in athletes of different ethnicities: differential diagnosis between athlete's heart and cardiomyopathy. *Eur Heart J.* 2015.

85. Malhotra A, Walker M, Dhutia H, et al. ECG interpretation in the athlete: A comparison of ethnic groups when three different criteria are applied. *Eur J Prev Cardiol.* 2015;22(1):s84-85.

86. Maron BJ, Wolfson JK, Ciro E, Spirito P. Relation of electrocardiographic abnormalities and patterns of left ventricular hypertrophy identified by 2-dimensional echocardiography in patients with hypertrophic cardiomyopathy. *Am J Cardiol.* 1983;51(1):189-194.

87. Haghjoo M, Mohammadzadeh S, Taherpour M, et al. ST-segment depression as a risk factor in hypertrophic cardiomyopathy. *Europace.* 2009;11(5):643-649.

88. MacAlpin RN. Clinical significance of QS complexes in V1 and V2 without other electrocardiographic abnormality. *Ann Noninvasive Electrocardiol.* 2004;9(1):39-47.

89. Bent RE, Wheeler MT, Hadley D, Froelicher V, Ashley E, Perez MV. Computerized Q wave dimensions in athletes and hypertrophic cardiomyopathy patients. *J Electrocardiol.* 2015;48(3):362-367.

90. Marek J, Toresdahl B, Zimmerman F, Drezner J. The Seattle ECG criteria for abnormal Q waves is not associated with findings of cardiomyopathy on limited echocardiography. Paper presented at: Heart Rhythm Society; May, 2014; San Francisco, CA.

91. Kim JH, Baggish AL. Electrocardiographic right and left bundle branch block patterns in athletes: prevalence, pathology, and clinical significance. *J Electrocardiol.* 2015;48(3):380-384.

92. Le VV, Wheeler MT, Mandic S, et al. Addition of the electrocardiogram to the preparticipation examination of college athletes. *Clin J Sport Med.* 2010;20(2):98-105.

93. Surawicz B, Childers R, Deal BJ, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society: endorsed by the International Society for Computerized Electrocardiology. *Circulation.* 2009;119(10):e235-240.

94. Aro AL, Anttonen O, Tikkanen JT, et al. Intraventricular conduction delay in a standard 12-lead electrocardiogram as a predictor of mortality in the general population. *Circ Arrhythm Electrophysiol.* 2011;4(5):704-710.

95. Desai AD, Yaw TS, Yamazaki T, Kaykha A, Chun S, Froelicher VF. Prognostic Significance of Quantitative QRS Duration. *Am J Med.* 2006;119(7):600-606.

96. Dunn T, Abdelfattah R, Aggarwal S, Pickham D, Hadley D, Froelicher V. Are the QRS duration and ST depression cut-points from the Seattle criteria too conservative? *J Electrocardiol.* 2015;48(3):395-398.

97. Xiao HB, Brecker SJ, Gibson DG. Relative effects of left ventricular mass and conduction disturbance on activation in patients with pathological left ventricular hypertrophy. *Br Heart J.* 1994;71(6):548-553.

98. Drezner JA, Prutkin JM, Harmon KG, et al. Cardiovascular screening in college athletes. *J Am Coll Cardiol.* 2015;65(21):2353-2355.

99. Cohen MI, Triedman JK, Cannon BC, et al. PACES/HRS expert consensus statement on the management of the asymptomatic young patient with a Wolff-Parkinson-White (WPW, ventricular preexcitation) electrocardiographic pattern: developed in partnership between the Pediatric and Congenital Electrophysiology Society (PACES) and the Heart Rhythm Society (HRS). Endorsed by the governing bodies of PACES, HRS, the American College of Cardiology Foundation (ACCF), the American Heart Association (AHA), the American Academy of Pediatrics (AAP), and the Canadian Heart Rhythm Society (CHRS). *Heart Rhythm.* 2012;9(6):1006-1024.

100. Daubert C, Ollitrault J, Descaves C, Mabo P, Ritter P, Gouffault J. Failure of the exercise test to predict the anterograde refractory period of the accessory pathway in Wolff Parkinson White syndrome. *Pacing Clin Electrophysiol.* 1988;11(8):1130-1138.

101. Klein GJ, Gulamhusein SS. Intermittent preexcitation in the Wolff-Parkinson-White syndrome. *Am J Cardiol.* 1983;52(3):292-296.

102. Klein GJ, Bashore TM, Sellers TD, Pritchett EL, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. *N Engl J Med.* 1979;301(20):1080-1085.

103. Schwartz PJ, Stramba-Badiale M, Crotti L, et al. Prevalence of the congenital long-QT syndrome. *Circulation.* 2009;120(18):1761-1767.

104. Tester DJ, Ackerman MJ. Cardiomyopathic and channelopathic causes of sudden unexplained death in infants and children. *Annu Rev Med.* 2009;60:69-84.

105. Eckart RE, Shry EA, Burke AP, et al. Sudden death in young adults an autopsy-based series of a population undergoing active surveillance. *J Am Coll Cardiol.* 2011;58(12):1254-1261.

106. Meyer L, Stubbs B, Fahrenbruch C, et al. Incidence, causes, and survival trends from cardiovascular-related sudden cardiac arrest in children and young adults 0 to 35 years of age: a 30-year review. *Circulation.* 2012;126(11):1363-1372.

107. Behr E, Wood DA, Wright M, et al. Cardiological assessment of first-degree relatives in sudden arrhythmic death syndrome. *Lancet.* 2003;362(9394):1457-1459.

108. Tester DJ, Spoon DB, Valdivia HH, Makielski JC, Ackerman MJ. Targeted mutational analysis of the RyR2-encoded cardiac ryanodine receptor in sudden unexplained death: a molecular autopsy of 49 medical examiner/coroner's cases. *Mayo Clin Proc.* 2004;79(11):1380-1384.

109. Tan HL, Hofman N, van Langen IM, van der Wal AC, Wilde AA. Sudden unexplained death: heritability and diagnostic yield of cardiological and genetic examination in surviving relatives. *Circulation.* 2005;112(2):207-213.

110. Viskin S, Rosovski U, Sands AJ, et al. Inaccurate electrocardiographic interpretation of long QT: the majority of physicians cannot recognize a long QT when they see one. *Heart Rhythm.* 2005;2(6):569-574.

111. Postema PG, De Jong JS, Van der Bilt IA, Wilde AA. Accurate electrocardiographic assessment of the QT interval: teach the tangent. *Heart Rhythm.* 2008;5(7):1015-1018.

112. Bazett HC. An analysis of the time-relations of electrocardiograms. *Heart.* 1920(7):353-370.

113. Johnson JN, Ackerman MJ. The prevalence and diagnostic/prognostic utility of sinus arrhythmia in the evaluation of congenital long QT syndrome. *Heart Rhythm.* 2010;7(12):1785-1789.

114. Malfatto G, Beria G, Sala S, Bonazzi O, Schwartz PJ. Quantitative analysis of T wave abnormalities and their prognostic implications in the idiopathic long QT syndrome. *J Am Coll Cardiol.* 1994;23(2):296-301.
115. Johnson JN, Ackerman MJ. QTc: how long is too long? *Br J Sports Med.* 2009;43(9):657-662.
116. Taggart NW, Haglund CM, Tester DJ, Ackerman MJ. Diagnostic miscues in congenital long-QT syndrome. *Circulation.* 2007;115(20):2613-2620.
117. Mason JW, Ramseth DJ, Chanter DO, Moon TE, Goodman DB, Mendzelevski B. Electrocardiographic reference ranges derived from 79,743 ambulatory subjects. *J Electrocardiol.* 2007;40(3):228-234.
118. Vincent GM, Timothy KW, Leppert M, Keating M. The spectrum of symptoms and QT intervals in carriers of the gene for the long-QT syndrome. *N Engl J Med.* 1992;327(12):846-852.
119. Rautaharju PM, Surawicz B, Gettes LS, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part IV: the ST segment, T and U waves, and the QT interval: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society: endorsed by the International Society for Computerized Electrocardiology. *Circulation.* 2009;119(10):e241-250.
120. Maron BJ, Zipes DP. 36th Bethesda Conference: Eligibility recommendations for competitive athletes with cardiovascular abnormalities. *J Am Coll Cardiol.* 2005;45(8):1312-1377.
121. Dhutia H, Malhotra A, Parpia S, et al. The prevalence and significance of a short QT interval in 18 825 low-risk individuals including athletes. *Br J Sports Med.* 2016;50(2):124-129.
122. Basavarajaiah S, Wilson M, Whyte G, Shah A, Behr E, Sharma S. Prevalence and significance of an isolated long QT interval in elite athletes. *Eur Heart J.* 2007;28(23):2944-2949.
123. Goldenberg I, Moss AJ, Peterson DR, et al. Risk factors for aborted cardiac arrest and sudden cardiac death in children with the congenital long-QT syndrome. *Circulation.* 2008;117(17):2184-2191.
124. Gollob MH, Redpath CJ, Roberts JD. The short QT syndrome: proposed diagnostic criteria. *J Am Coll Cardiol.* 2011;57(7):802-812.
125. Schwartz PJ, Moss AJ, Vincent GM, Crampton RS. Diagnostic criteria for the long QT syndrome. An update. *Circulation.* 1993;88(2):782-784.
126. Schwartz PJ, Crotti L. QTc behavior during exercise and genetic testing for the long-QT syndrome. *Circulation.* 2011;124(20):2181-2184.
127. Ackerman MJ, Priori SG, Willems S, et al. HRS/EHRA expert consensus statement on the state of genetic testing for the channelopathies and cardiomyopathies this document was developed as a partnership between the Heart Rhythm Society (HRS) and the European Heart Rhythm Association (EHRA). *Heart Rhythm.* 2011;8(8):1308-1339.
128. Wilde AA, Antzelevitch C, Borggrefe M, et al. Proposed diagnostic criteria for the Brugada syndrome: consensus report. *Circulation.* 2002;106(19):2514-2519.
129. Bayes de Luna A, Brugada J, Baranchuk A, et al. Current electrocardiographic criteria for diagnosis of Brugada pattern: a consensus report. *J Electrocardiol.* 2012;45(5):433-442.

130. Priori SG, Wilde AA, Horie M, et al. HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes: document endorsed by HRS, EHRA, and APHRS in May 2013 and by ACCF, AHA, PACES, and AEPC in June 2013. *Heart Rhythm.* 2013;10(12):1932-1963.

131. Antzelevitch C, Brugada P, Borggrefe M, et al. Brugada syndrome: report of the second consensus conference: endorsed by the Heart Rhythm Society and the European Heart Rhythm Association. *Circulation.* 2005;111(5):659-670.

132. Kapplinger JD, Tester DJ, Alders M, et al. An international compendium of mutations in the SCN5A-encoded cardiac sodium channel in patients referred for Brugada syndrome genetic testing. *Heart Rhythm.* 2010;7(1):33-46.

133. Tester DJ, Medeiros-Domingo A, Will ML, Haglund CM, Ackerman MJ. Cardiac channel molecular autopsy: insights from 173 consecutive cases of autopsy-negative sudden unexplained death referred for postmortem genetic testing. *Mayo Clin Proc.* 2012;87(6):524-539.

134. Chung EH. Brugada ECG patterns in athletes. *J Electrocardiol.* 2015;48(4):539-543.

135. Chung EH, McNeely DE, 3rd, Gehi AK, et al. Brugada-type patterns are easily observed in high precordial lead ECGs in collegiate athletes. *J Electrocardiol.* 2014;47(1):1-6.

136. Zorzi A, Leoni L, Di Paolo FM, et al. Differential diagnosis between early repolarization of athlete's heart and coved-type Brugada electrocardiogram. *Am J Cardiol.* 2015;115(4):529-532.

137. Groh WJ. Arrhythmias in the muscular dystrophies. *Heart Rhythm.* 2012;9(11):1890-1895.

138. Verdile L, Maron BJ, Pelliccia A, Spataro A, Santini M, Biffi A. Clinical significance of exercise-induced ventricular tachyarrhythmias in trained athletes without cardiovascular abnormalities. *Heart Rhythm.* 2015;12(1):78-85.

139. Biffi A, Pelliccia A, Verdile L, et al. Long-term clinical significance of frequent and complex ventricular tachyarrhythmias in trained athletes. *J Am Coll Cardiol.* 2002;40(3):446-452.

140. Corrado D, Basso C, Leoni L, et al. Three-dimensional electroanatomical voltage mapping and histologic evaluation of myocardial substrate in right ventricular outflow tract tachycardia. *J Am Coll Cardiol.* 2008;51(7):731-739.

141. Heidbuchel H, Hoogsteen J, Fagard R, et al. High prevalence of right ventricular involvement in endurance athletes with ventricular arrhythmias. Role of an electrophysiologic study in risk stratification. *Eur Heart J.* 2003;24(16):1473-1480.

142. Biffi A, Maron BJ, Verdile L, et al. Impact of physical deconditioning on ventricular tachyarrhythmias in trained athletes. *J Am Coll Cardiol.* 2004;44(5):1053-1058.

143. Delise P, Lanari E, Sitta N, Centa M, Allocca G, Biffi A. Influence of training on the number and complexity of frequent VPBs in healthy athletes. *J Cardiovasc Med (Hagerstown).* 2011;12(3):157-161.

144. Delise P, Sitta N, Lanari E, et al. Long-term effect of continuing sports activity in competitive athletes with frequent ventricular premature complexes and apparently normal heart. *Am J Cardiol.* 2013;112(9):1396-1402.

145. James CA, Bhonsale A, Tichnell C, et al. Exercise increases age-related penetrance and arrhythmic risk in arrhythmogenic right ventricular dysplasia/cardiomyopathy-associated desmosomal mutation carriers. *J Am Coll Cardiol.* 2013;62(14):1290-1297.

146. Ruwald AC, Marcus F, Estes NA, 3rd, et al. Association of competitive and recreational sport participation with cardiac events in patients with arrhythmogenic right ventricular cardiomyopathy: results from the North American multidisciplinary study of arrhythmogenic right ventricular cardiomyopathy. *Eur Heart J.* 2015;36(27):1735-1743.

147. La Gerche A, Robberecht C, Kuiperi C, et al. Lower than expected desmosomal gene mutation prevalence in endurance athletes with complex ventricular arrhythmias of right ventricular origin. *Heart.* 2010;96(16):1268-1274.

148. Sawant AC, Bhonsale A, te Riele AS, et al. Exercise has a disproportionate role in the pathogenesis of arrhythmogenic right ventricular dysplasia/cardiomyopathy in patients without desmosomal mutations. *J Am Heart Assoc.* 2014;3(6):e001471.

149. Heidbuchel H, Prior DL, Gerche AL. Ventricular arrhythmias associated with long-term endurance sports: what is the evidence? *Br J Sports Med.* 2012;46 Suppl 1:i44-i50.

150. Uhm JS, Hwang IU, Oh YS, et al. Prevalence of electrocardiographic findings suggestive of sudden cardiac death risk in 10,867 apparently healthy young Korean men. *Pacing Clin Electrophysiol.* 2011;34(6):717-723.

151. Anderson JL, Halperin JL, Albert NM, et al. Management of patients with atrial fibrillation (compilation of 2006 ACCF/AHA/ESC and 2011 ACCF/AHA/HRS recommendations): a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2013;61(18):1935-1944.

152. Aagaard P, Sahlen A, Braunschweig F. Performance trends and cardiac biomarkers in a 30-km cross-country race, 1993-2007. *Med Sci Sports Exerc.* 2012;44(5):894-899.

153. Sahlen A, Gustafsson TP, Svensson JE, et al. Predisposing factors and consequences of elevated biomarker levels in long-distance runners aged >or=55 years. *Am J Cardiol.* 2009;104(10):1434-1440.

154. Rose G, Baxter PJ, Reid DD, McCartney P. Prevalence and prognosis of electrocardiographic findings in middle-aged men. *Br Heart J.* 1978;40(6):636-643.

155. Chou R, Arora B, Dana T, Fu R, Walker M, Humphrey L. Screening asymptomatic adults with resting or exercise electrocardiography: a review of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med.* 2011;155(6):375-385.

156. Daviglus ML, Liao Y, Greenland P, et al. Association of nonspecific minor ST-T abnormalities with cardiovascular mortality: the Chicago Western Electric Study. *JAMA.* 1999;281(6):530-536.

157. Borjesson M, Urhausen A, Kouidi E, et al. Cardiovascular evaluation of middle-aged/senior individuals engaged in leisure-time sport activities: position stand from the sections of exercise physiology and sports cardiology of the European Association of Cardiovascular Prevention and Rehabilitation. *Eur J Cardiovasc Prev Rehabil.* 2011;18(3):446-458.

158. Maron BJ, Araujo CG, Thompson PD, et al. Recommendations for preparticipation screening and the assessment of cardiovascular disease in masters athletes: an advisory for healthcare professionals from the working groups of the World Heart Federation, the International Federation of Sports Medicine, and the American Heart Association Committee on Exercise, Cardiac Rehabilitation, and Prevention. *Circulation.* 2001;103(2):327-334.

159. Ranthe MF, Carstensen L, Oyen N, et al. Family history of premature death and risk of early onset cardiovascular disease. *J Am Coll Cardiol.* 2013;60(9):814-821.

160.    Ranthe MF, Carstensen L, Oyen N, et al. Risk of Cardiomyopathy in Younger Persons With a Family History of Death from Cardiomyopathy: A Nationwide Family Study in a Cohort of 3.9 Million Persons. *Circulation.* 2015;132(11):1013-1019.

161.    Asif IM, Price D, Fisher LA, et al. Stages of psychological impact after diagnosis with serious or potentially lethal cardiac disease in young competitive athletes: a new model. *J Electrocardiol.* 2015;48(3):298-310.

162.    Maron BJ, Chaitman BR, Ackerman MJ, et al. Recommendations for physical activity and recreational sports participation for young patients with genetic cardiovascular diseases. *Circulation.* 2004;109(22):2807-2816.

163.    Takken T, Giardini A, Reybrouck T, et al. Recommendations for physical activity, recreation sport, and exercise training in paediatric patients with congenital heart disease: a report from the Exercise, Basic & Translational Research Section of the European Association of Cardiovascular Prevention and Rehabilitation, the European Congenital Heart and Lung Exercise Group, and the Association for European Paediatric Cardiology. *Eur J Prev Cardiol.* 2012;19(5):1034-1065.