Special Report

Interpretation of the Electrocardiogram of Young Athletes

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Sudden cardiac death in a young athlete is a tragic and high-profile event. The best way to prevent such deaths is, however, highly debated. The Italian experience informed the European recommendation for the inclusion of a 12-lead ECG in screening tests for all athletes.[1,2] Although American authors have acknowledged the possible benefits of such an approach, many have expressed concern over the portability of such a model to the US healthcare system. Concern has focused in particular on the idea of mandatory testing, cost effectiveness, the availability of practitioners qualified to interpret ECGs, and the burden of false-positive results. With professional sports organizations such as the International Olympic Committee, the National Basketball Association, the National Football League, and the Union of European Football Associations endorsing or implementing screening programs for their athletes, with a recent analysis suggesting a degree of cost effectiveness in line with other accepted medical interventions,[3] and with the American Heart Association offering a cautious endorsement to the idea of local programs,[4] volunteer-led testing programs across the US have begun to emerge. Thus, although no detailed guidance for the interpretation of the athlete's ECG exists, many physicians will be called on to interpret an athlete's ECG.

Editorial see p 669

A principal obstacle to such interpretation is the difficulty in distinguishing abnormal patterns from physiological effects of training. Many clinical and ECG findings that may be a cause of concern in the general population are normal for athletes. In addition, the test characteristics of the ECG for different findings vary according to age, sex, ethnicity, sport, and level of training. In particular, different challenges exist for younger athletes because of the evolution of the ECG with age. This is further complicated by historical inconsistencies in the definition of ECG abnormalities and the uncertainty about criteria for final diagnosis of several diseases in secondary testing.[5–11] Finally, low disease prevalence limits the positive predictive value of many ECG criteria, even for those with otherwise favorable sensitivity and specificity.

Although this document focuses in large part on the diagnostic gray area presented by ECG screening, we have where possible included suggestions for secondary testing strategies. Current recommendations for patients diagnosed with cardiomyopathy or channelopathy are clearly against participation in high-level or competitive exercise.[11,12]

Revised European Guidelines Relating to the Athlete's ECG

An international group of experts under the auspices of the European Society of Cardiology (ESC) recently published new recommendations for the interpretation of the ECG in athletes.[13] As part of this report, they reanalyzed the 1005 highly trained athletes previously presented by Pelliccia and colleagues in a landmark study.[13,14] Originally, 40% (n=402) were considered to have findings possibly associated with cardiovascular disease. However, using the new ESC recommendations this percentage was lowered to 11%, which implies a meaningful increase in specificity. The age range of these athletes was 9 to 55 years with 25% female, 99% white, and most participating in Olympic sports. In a US context, we applied this reclassification scheme to a study of Stanford collegiate athletes (age range 18 to 22, 46% female, 10% black).[15] In our original analysis of 658 athletes, 62% of the men and 32% of the women had abnormal ECGs and 63 (10%) were considered to have ECG patterns possibly associated with cardiovascular diseases that warranted further testing. This latter classification category was similar to the

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group defined as "distinctly abnormal" by Pelliccia et al.[14] When these 63 "abnormal" ECGs were evaluated using our interpretation of the new ESC criteria, 34 (6%) were reclassified to the normal range and only 29 (4%) remained in the abnormal category. Thus, implied specificity rises to above 95%. These data seem to support the assertion by Corrado et al[13] that the new criteria improve the specificity of the ECG as part of the preparticipation examination, but this remains to be tested in a prospective study.

Training-Related ECG Changes

The principal change in the approach of the new ESC guidelines, which mirrors that of most practitioners in the US carrying out ECG screening in young people, is the recognition of the range of findings that are the direct result of training.

Increased QRS Voltage

A significant change in the most recent ESC guidelines document relates to the treatment of isolated QRS voltage. The largest proportion of athletes with ECGs classified as abnormal using previous criteria exhibited isolated increases in QRS voltage (prevalence of up to 80% in some series). Because of this and other evidence that such voltage correlates poorly with left ventricular mass in young athletes,[16] there is widespread acceptance that in the absence of other markers suggesting actual left ventricular hypertrophy (axis changes, changes in repolarization, atrial abnormalities, increased QRS width), high QRS voltage is not a sufficient reason in isolation to refer an athlete for further evaluation.[17,18]

Early Repolarization

The finding of ST elevation in V3–6 with an elevated J point and a peaked upright T wave (or more commonly in athletes of African descent, a domed ST segment followed by a biphasic or inverted T wave) is present in >50% of trained athletes. It is particularly prevalent in men. Of note, ECG changes of high voltage and abnormal repolarization can precede echocardiogram changes in hypertrophic cardiomyopathy. Although a normal echocardiogram in this setting (in the absence of other factors) may allow participation, such athletes should be followed up serially. In athletes, although the mechanism is uncertain, early repolarization seems to regress with age and when training declines and often changes or disappears during a bout of exercise or with increasing heart rate (suggesting potentially a vagally mediated or heart rate–sensitive mechanism). It is important to distinguish these findings from the Brugada-like ECG pattern that is recognized in V1–2.[19]

Recent interest in early repolarization focused on the finding of its increased prevalence in 206 patients with idiopathic ventricular tachycardia (VT)/ventricular fibrillation (VF).[20] Community epidemiological studies reported that J waves or terminal slurring of the QRS, particularly in the inferior leads, had an adjusted hazard ratio of 2 to 4 for cardiac death.[21,22] Although a hazard ratio of 2 is an important finding, it does not provide adequate differentiation for screening. Cappoto et al studied athletes with sudden cardiac death (SCD) and 365 healthy athletes.[19] J wave and/or QRS slurring was found more frequently among athletes with cardiac arrest/sudden death than in control athletes. Nevertheless, the presence of this ECG pattern did not confer a higher risk for recurrent malignant ventricular arrhythmias. Of additional note is that ST elevation >2 mm seems to be unusual even in athletes.

Manifestations of Increased Vagal Tone

Sinus bradycardia, prolonged PR interval, and Wenckebach phenomenon are common in athletes as a result of the high resting vagal tone, or significantly lower intrinsic heart rates.[23]

Authors' Recommendation

We do not recommend further evaluation for any degree of QRS voltage as long as it is isolated (ie, there are no other findings and it is associated with normal axis, acceptable repolarization, and normal atrial activation). Similarly, we do not recommend further evaluation for sinus bradycardia as low as 30 bpm (with sinus arrhythmia, some RR intervals could be prolonged to 3 seconds) or isolated early repolarization. A prolonged PR interval up to 300 ms should not prompt further workup, but longer intervals should be resolved with an exercise test (the PR interval should shorten as vagal tone is withdrawn). Similarly, Wenckebach phenomenon in isolation need not prompt further work up, but an exercise test could resolve any concern.

Comparison With European Society of Cardiology Document

These recommendations are in line with those of the ESC.

Q Waves

The pathophysiological basis of Q waves differs depending on the disease process (eg, ischemic or infiltrative myocardial disease versus classical asymmetrical hypertrophic cardiomyopathy (HCM). Notably, the diagnostic criteria also differ. Q-wave criteria for myocardial infarction range from the World Health Organization criteria (≥40 ms and amplitude >24% of the following R wave in 2 contiguous leads) to the computer-applied vectorial area criteria and Minnesota Code scores.[24,25] Q waves in HCM appear to be caused by ventricular asymmetry, as demonstrated by magnetic resonance imaging (MRI).[26] Distinct criteria for HCM have been tested that differ with respect to the definition of Q waves.[27] The best test characteristics for Q waves in HCM were found with >3 mm in depth and/or >40 ms duration in at least 2 leads. In this case, the "and/or" yields more positives than the "and" qualifier for Q wave criteria in coronary artery disease. Scores including[28] or excluding Q wave criteria[29] have been shown to have reasonable diagnostic yield in patients with HCM. The Q waves of HCM most often are seen in the inferior and/or lateral leads (Figure 1). The high QRS voltage seen in both athletes and HCM patients, however, means that these criteria could lead to the identification of many more athletes than those based on 25% of the proceeding R wave alone.

Figure 1. A 5-mm Q wave in lead V5 in a patient with hypertrophic cardiomyopathy. Note this is considered abnormal by ESC criteria and by our recommendation, but not by the 25% of the R wave criterion.

Authors' Recommendation

Coronary artery disease is rare in individuals <40 years of age, whereas coronary anomalies tend not to be associated with myocardial infarction. Therefore, we recommend that HCM criteria for Q waves be used in young athletes (>3 mm in depth and/or >40 ms duration in any lead except AVR, III, and V1). We do not endorse the use of standard coronary disease criteria for Q waves in young athletes, but they should apply in athletes >40 years of age.

Comparison With the European Society of Cardiology Document

Specific criteria for Q waves are not discussed in the most recent ESC document, but prior publications from the group suggest an amplitude of 4 mm for Q-wave classification (adopted from the Pelliccia et al criteria for markedly abnormal). Athletes with Q waves should be referred for further evaluation.

Further Evaluation

Further evaluation for an athlete found to have Q waves should include a more detailed history, physical examination, and a full resting echocardiogram. This study should include standard measurements of chamber size, wall thickness, and valvular function. In addition, it is valuable to estimate left and right ventricular as well as left atrial volumes using the Simpson rule. Measures of diastolic function including the tissue Doppler of the lateral or medial mitral annulus can be particularly valuable for detecting subclinical cardiomyopathy.[30] Modern echocardiography probes can detect coronary ostia in most adults. Depending on availability, cardiac MRI can provide accurate estimations of all these parameters as well as providing the additional value of delayed gadolinium enhancement for the detection of myocardial fibrosis or infiltrative disease, clearer delineation of proximal coronary arteries, and characterization of the subvalvular apparatus of the mitral valve. Cardiopulmonary exercise testing, though not necessary, may provide additional value in the differentiation of cardiomyopathy from athlete's heart if this is still not clear from imaging studies and ECG.[31] In particularly borderline cases, a full 4-generation family pedigree and genetic testing may be able to rule in cardiomyopathy or channelopathy. Convincingly causal mutations are found in ≈50% of patients who clearly have the disease, implying that the yield for athletes with borderline findings would be considerably lower than this. However, more extensive genetic testing, including whole-genome sequencing, may change this in the near future. For patients with findings that remain truly in a gray zone (ie, not diagnostic for cardiomyopathy), we recommend full disclosure of the unknown nature of the risk with a personal decision by the athlete on whether to participate. Examples include isolated wall thickness <15 mm, isolated borderline low left ventricular ejection fraction (50% to 55%), and isolated hypertrabeculation. A period of deconditioning may be informative in certain cases.[32] Detailed discussion of secondary testing strategies is beyond the scope of this review; however, brief notes are made in each section below.

Conduction Delay

Although none would contest the need for further evaluation in athletes with a QRS duration longer than 120 ms, the excellent prognosis in asymptomatic air crewmen with this abnormality makes it likely that most will have normal imaging studies.[33] Left bundle branch block appears to be less common and more ominous than right bundle branch block (RBBB) in asymptomatic athletes. In countries where Chagas disease is prevalent, its association with conduction system disease should be considered. Most computer interpretive programs make RBBB the interpretive statement for QRS durations as short as 106 ms if a RBBB pattern is present. QRS duration is a documented risk marker in HCM[34] as well as in other populations.[35]

Authors' Recommendation

All athletes with a QRS duration >120 ms should be referred for further evaluation. This is one area where digital analysis can outperform standard visual measurement because the first onset and last offset in all of the leads can be considered. When an isolated RBBB pattern is present at <120 ms duration, most would not refer for further evaluation. However, the association of this pattern with atrial septal defect leads some practitioners to recommend an echocardiogram with contrast.

Comparison With the European Society of Cardiology Document

These recommendations are in line with the ESC document.

Further Evaluation

A standard secondary testing strategy (see Q-wave section) is indicated. A cardiac MRI may be particularly useful for ruling out infiltrative disease.

QRS Axis Deviation

The axis of the QRS complex is greatly dependent on age: it begins rightward at birth and shifts leftward with age. As most screened athletes are at an age when the axis is still in transition, right-axis deviation is a common finding (reported prevalence as high as 20%[36]). In older populations, right-axis deviation is rare and generally associated with pulmonary disease. Left-axis deviation occurs in 8% of healthy air crewmen and is the most common abnormal ECG finding in the 30 to 40s age group. Several large cohort studies have attempted to define normal axis for age. The largest of these studied 46 129 individuals with a low probability of cardiovascular disease and found that 95% of athletes <20 years of age had QRS axis 0 to 102° and 95% of athletes 20 to 29 years of age had QRS axis of −10 to 95°.[37] Sharma et al demonstrated similar findings in a cohort 18 years of age and under, with 95% of athletes exhibiting a QRS axis between 41 and 113°.[38]

Authors' Recommendation

In athletes, mild right-axis deviation or left-axis deviation should not trigger further evaluation unless there is a history of pulmonary disease or systemic hypertension, respectively. We recommend an acceptable range of between −30 and +115 degrees for isolated axis deviation.

Comparison With the European Society of Cardiology document

Specific criteria for axis deviation are not discussed in the ESC document. However, this recommendation is consistent with previous writings from that group.

Further Evaluation

A standard secondary testing strategy is appropriate (see Q-wave section).

Right Ventricular Hypertrophy

Various voltage criteria have been recommended for right ventricular hypertrophy including R wave >7 mm in V1, R/S ratio >1 in V1, and the sum of R wave in V1 and S wave in V5/6>10.5 mm (Sokolow-Lyon). Although the Sokolow-Lyon voltage criteria for right ventricular hypertrophy was seen in only 1 of 172 professional soccer players (0.6%),[39] Sharma et al[38] reported a prevalence of 10% to 12% among junior elite athletes and controls.

Authors' Recommendation

Until careful studies are made of the voltage measurements in the involved leads (R and S waves in V1/V2 and V5/6) of normals and athletes according to age, we do not recommend that traditional voltage criteria violations trigger further evaluations in athletes <30 years of age. We recommend that voltage-only criteria for right ventricular hypertrophy are, in general, not applicable to young athletes and that additional findings such as right atrial abnormalities (RAA), T-wave inversion in V2/3, and/or right-axis deviation are necessary to elicit further evaluation before participating in sports.

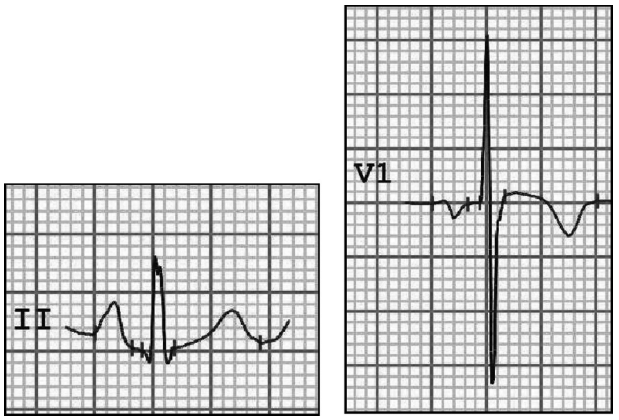


Figure 2. Example of atrial abnormalities in college athletes (left, RAA; right LAA).

Comparison With the European Society of Cardiology Document

The ESC document suggests further evaluation for athletes with right ventricular hypertrophy. Although specific criteria are not discussed, allusion is made to the Sokolow-Lyon voltage criteria.

Further Evaluation

A standard secondary testing strategy is appropriate (see Q-wave section). However, the echocardiogram is disadvantaged in characterizing the right ventricle. A cardiac MRI should be used if possible.

Atrial Abnormalities

Although the criterion for RAA is simple (P-wave amplitude >2.5 mm in any lead), the criteria for left atrial abnormality (LAA) are 2 fold: (1) a negative component of the P wave in V1 or V2 of 40 ms duration and 1 mm amplitude, and (2) total P-wave duration of >120 ms (Figure 2). Computer determination of this latter duration criteria on the basis of spatial mathematical constructs can result in "normal" values much greater than the visually determined 120 ms and accordingly are not applicable. The prevalence of P-wave voltage and duration consistent with LAA and RAA in athletes is variable and dependent on age. Sharma et al describe prevalence of 14% and 18% for LAA and RAA, respectively, in 1000 junior elite athletes with mean age of 16 years. However, in another population of 649 collegiate athletes with mean age of 20, LAA was seen in 0.7% and RAA in 1.8% of athletes. Pelliccia et al found the prevalence of LAA to be 4% and RAA to be 0.8% in 1005 trained athletes.[14]

Authors' Recommendation

Debate exists on the prevalence of LAA and RAA in athletes, and it appears to be more common in younger athletes.

Computer measurement of total P-wave duration is not standardized, and visual assessment is recommended. We recommend that in collegiate and adult athletes, atrial abnormalities should be regarded as abnormal and lead to secondary investigation. Isolated atrial abnormalities in younger athletes should lead to a careful physical examination by a qualified physician and repeated detailed medical and family history.

Comparison With the European Society of Cardiology Document

The ESC document recommends further workup for the finding of atrial enlargement in all athletes.

Further Evaluation

A standard secondary testing strategy is appropriate (see Q-Wave section).

T-Wave Inversion

T-wave inversion (TWI) has similar prevalence among athletes and sedentary controls,[38] suggesting that it is not a training-related phenomenon. Indeed, Pelliccia et al showed that TWI in the presence of normal imaging may be a harbinger of a future cardiomyopathic disease.[40] Overall, TWI in large populations of mostly white athletes seems to be present at around 2% to 3%. However, Sharma et al have emphasized the importance of considering the ethnicity of the athlete. In a recent study, 240 black female athletes exhibited a higher prevalence of T-wave inversion compared with matched nonblack athletes (14% versus 2%).[16]Notably, there were no significant differences in absolute values of maximal left ventricular wall thickness between athletes with T-wave inversions and those without. Black male athletes are also known to exhibit greater degrees of early repolarization, the voltage criterion for LVH and TWI. The last was found in 20% of 155 soccer players under 17 years of age in one African study.[41]

Detailed computer analyses including vectorial assessment of T waves of athletes would help clarify an ethnically specific range of normal for TWI. A further important consideration relates to the significance of the negative component of biphasic T waves.

Authors' Recommendation

In athletes not of African origin, TWI $\geq$1 mm in leads other than III, aVR, and V1/2 should lead to secondary evaluation. In athletes of African origin, TWI after ST elevation in V2-V4 does not need investigation whereas inferior or lateral lead TWI warrants follow-up. In athletes with biphasic T waves, we recommend considering the area contributed by the negative portion rather than considering depth below the isoelectric line until better classification is available.

Comparison With the European Society of Cardiology Document

The ESC document recommends that both minor (not defined) and significant ($\geq$2 mm in $\geq$2 adjacent leads) TWI should lead to a secondary evaluation. In athletes of African/Caribbean origin, TWI after ST elevation in V2-V4 does not need investigation whereas inferior or lateral lead TWI warrants follow-up.

Further Evaluation

A standard secondary testing strategy is appropriate (see Q-Wave section). Because TWI may be a harbinger of future disease, athletes with TWI whose imaging studies are negative (most likely >90%) should be followed annually with ECG and echocardiography. Cardiac MRI with gadolinium may be helpful in athletes with marked TWI in the inferior and lateral leads to rule out apical-variant HCM that may not be easily identified by echocardiography.

ST Depression

ST depression is rare in athletes and always deserves further workup. In assessing ST depression, careful attention must be paid to the choice of isoelectric line. Although the U-P wave level is theoretically appealing, the PR level is more practical. The PR level can be greatly affected by atrial repolarization and shortened PR intervals. Computer analysis further complicates this by detecting depression not easily seen visually. The ST level is also affected by whether the J-point, some later point, or the ST area is used. ST depression has been demonstrated to be prognostic in HCM,[42] as well as other cardiac conditions.

Authors' Recommendation

An athlete with any visually appreciated ST depression >0.5 mm below the PR isoelectric line occurring between the J-junction and beginning of the T wave >0.5 mm in any of the lateral leads (I, AVL, V5, V6) and >1 mm in any lead, should be sent for further evaluation.

Comparison With the European Society of Cardiology Document

The ESC document recommends that resting ST depression always be further evaluated but does not provide detail on to what extent and which leads.

Further Evaluation

A standard secondary testing strategy is appropriate (see Q-Wave section).

QT Abnormalities (Long and Short)

The QT interval reflects the time from initial depolarization of ventricular myocytes to the end of their repolarization. Repolarization abnormalities are caused by mutations in 1 of several membrane ion channels or exposure to medications that interfere with ion-channel function. A long-QT interval is indicative of prolonged repolarization that places athletes at higher risk of exercise-related SCD from the ventricular arrhythmia known as torsades de pointes. In a rare set of families with mutations causing augmented function of potassium ion channels responsible for repolarization, the QT shortens and is also associated with sudden death. The determination of an abnormal QT is challenging because of the often indistinct termination of the T wave and dynamic nature of the QT interval, which varies with heart rate, age, autonomic state, medications, and other factors. Numerous

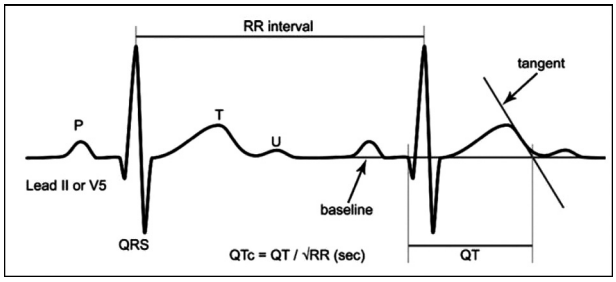


Figure 3. Illustration of a manual method of measuring QT interval. This also illustrates the isoelectric baseline used for measuring ST depression. Copyright © 2008, reprinted with permission from Elsevier.[45]

methods, each with different limitations, have been proposed for heart-rate correction of the QT interval (QTc)[43]; the most commonly used is the Bazett formula: QTc=QT/(square root of the preceding R-R interval). Of note, this is less reliable at lower heart rates. Computerized measurements of the QT interval can be misleading[44] and inconsistent between programs. Therefore, manual measurement and rate correction are necessary for accurate determination of the QTc. Methods such as using the tangent of the downslope of the T wave to calculate the T-wave intercept can be used to more accurately measure the QT interval[45] (Figure 3). There is no consensus on which lead to use for QTc measurement, but a reasonable approach is to measure the longest QT interval from a limb or precordial lead with a clear termination of the T wave. Others recommend focusing particularly on leads II and V5. Discrete, low-amplitude U waves should not be used in the calculation of the QT interval; however, U waves that merge with the T wave or give a "bifid" T-wave appearance should be taken into account. Other features of the T wave, such as morphology or variability of the QT across leads, may help distinguish between genotypes or reflect different features of repolarization but do not add as much prognostic value as the QTc interval itself.

Debate exists about what should be considered a normal QTc interval.[46] Traditional cutoffs are up to 440 ms in men and 460 ms in women. It is clear that longer QTc intervals, particularly those >500 ms, are associated with higher risk of sudden death. However, traditional cutoffs also result in false-positive rates as high as 11%.[47] Meanwhile, although a normal QT interval is associated with a lower risk, it does not rule out a potentially lethal long-QT genotype. Although a very short QTc (<340ms) is seen in the context of a genetic diagnosis of short-QT syndromes caused by abnormalities in potassium or calcium channels, very short QTc intervals (even <320ms) seem to be rare in large adult populations and not associated with adverse outcomes.[48] However, there is significant uncertainty surrounding the risk of short QTc in athletes.

Authors' Recommendation

We recommend that all athletes with a QTc >470 ms in men or 480 ms in women should undergo further evaluation for long-QT syndrome. We believe these cutoffs provide the best balance between false-positive and negative findings. QTc intervals shorter than 340 ms should also lead to further evaluation.

Comparison With the European Society of Cardiology Document

The ESC does not give a definitive recommendation relative to QTc cutoffs for secondary evaluation of long QT. The authors note that some believe a QTc of >500 ms is indicative of unequivocal long-QT syndrome while defining a gray zone between 440 ms (men)/460 ms (women) to 500 ms where more careful history may be revealing. Some sports physicians use 440 ms (men) and 460 ms (women) as an absolute indication for secondary testing.

A short QTc is defined by the ESC authors as 330 ms (or 310 ms in children). Values <380 ms, however, should lead to ruling out secondary causes (hypercalcemia, hyperkalemia, hyperthermia, acidosis, and drugs such as digitalis), and if none is found, clinical evaluation is recommended.

Further Evaluation

Further evaluation for channelopathy includes exclusion of secondary causes of prolonged or shortened QT, a 4-generation family pedigree, exercise or medication stress, extended rhythm monitoring, and consideration of genetic testing. A lying and standing 12-lead ECG has recently been shown to uncover prolonged repolarization in a proportion of long-QT patients.[49] A long QT <500 ms in isolation without evidence to suggest arrhythmia and without a revealing family history represents a true gray area. In such cases, we recommend full disclosure of the unknown nature of risk to the athlete with a personal decision on competition.

Brugada-Like ECG Abnormalities and Arrythmogenic Right Ventricular Cardiomyopathy or Dysplasia

The Brugada ECG pattern is associated with ventricular arrhythmia and sudden death. In many cases, Brugada syndrome follows a familial pattern of inheritance and in ≈30% of cases, the pattern seems to be explained by a mutation in the sodium ion channel gene *SCN5A*. It is thought to be responsible for a significant proportion of SCD events, and although sudden death is generally not held to be precipitated by brief bouts of exercise, the increase in vagal tone associated with long-term intense training is thought to convey overall increased risk. The Brugada syndrome is defined by symptoms such as syncope in the presence of a type 1 Brugada pattern (RBBB morphology with a 2 mm ST-segment elevation). The second Brugada Syndrome Consensus Conference[50] established criteria for 3 different Brugada-like ECG patterns (Figure 4). The type 1 pattern is further characterized by a coved-type ST segment that gradually descends into an inverted T wave. Type-2 pattern has a saddleback ST T wave with an ST-segment elevation that is >1 mm with a positive or biphasic T wave. Type-3 pattern also has a saddleback ST T wave but with an ST-segment elevation that is <1 mm and a positive T wave. Although lead V2 is usually the most pronounced, these patterns can extend from lead V1 through V3. If the ECG pattern is equivocal, placement of the precordial leads 1 to 2 intercostal spaces

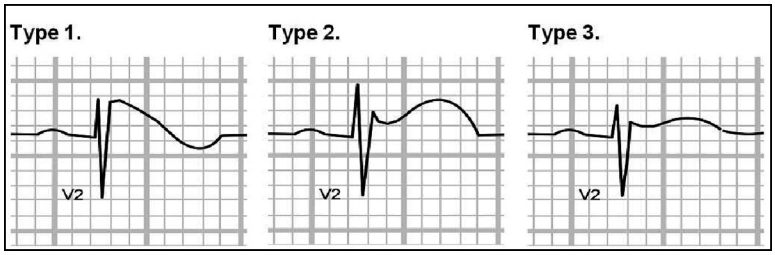


Figure 4. Illustration of the 3 types of Brugada pattern. Type 1 is the only one that is diagnostic for the condition.

higher can reveal an otherwise concealed type 1 pattern. Patients with inconclusive or type 2 and 3 ECG patterns can be challenged with sodium channel–blocking antiarrhythmic medications such as flecainide, procainamide, or ajmaline to reveal a type 1 pattern or induce ventricular arrhythmia.

Arrythmogenic right ventricular cardiomyopathy or dysplasia (ARVC/D) is a desmosomal disease resulting in fibro-fatty replacement of the right or left ventricle causing electric instability and cardiomyopathy.[51] Repolarization abnormalities are sensitive markers of disease expression in ARVC:TWI in V2–3, prolonged S-wave upstroke, and on occasion, epsilon waves. In the presence of RBBB, such repolarization abnormalities are harder to evaluate. However, extensive anterior TWI remains uncommon in the normal population. The modification of task force criteria for diagnosis of ARVC[51] now include Epsilon waves (reproducible low-amplitude signals between the end of the QRS complex and the onset of the T wave) in the right precordial leads (V1–V3) as a major criterion. Cause of death in exercise is usually due to acceleration of VT that degenerates to VF.[52] The mechanism behind exercise-induced SCD in ARVC/D, however, is not entirely clear. The finding of a slurred S wave is not felt to be sufficient to trigger further evaluation in athletes because its specificity is low.[53] Although the most common cause of sudden death in young people in Italy, and recognized at a significant percentage in Denmark[54] and the UK,[55] ARVC/D seems to be less recognized elsewhere. For example, it is associated with only 1% of deaths in military recruits in a US Army autopsy study.[56] However, in the US, autopsy methods and analysis, other than in the military, are not standardized, and the autopsy diagnosis of ARVC/D may be missed without careful examination of the right ventricle and right ventricular outflow tract.

Authors' Recommendation

Asymptomatic athletes noted on screening to have type 1 Brugada ECG pattern should be further evaluated. We do not recommend further evaluation of athletes with isolated findings consistent with Epsilon waves. However, when associated with TWI or a significant family history, secondary testing is mandatory.

Comparison With the European Society of Cardiology Document

The ESC document provides specific guidance for distinguishing the type 1 pattern from athlete's heart: Athletes exhibit an upsloping ST segment with a mean STJ/ST80 ratio ≤ 1 whereas Brugada patients show a downsloping ST segment with a STJ/ST80 ratio >1. Athletes with a suspected Brugada ECG should be referred for further evaluation. The ESC document does not make a specific recommendation in relation to ARVC, but it discusses the condition in relation to TWI and incomplete RBBB.

Further Evaluation

Secondary assessment for presence of the Brugada syndrome, including a search for associated clinical criteria, which include unexplained syncope, aborted sudden death, self-terminating VF or polymorphic VT, family history of sudden death, or nocturnal agonal respiration.[57] In long-term prospective population studies, asymptomatic subjects identified with spontaneous Brugada pattern had no adverse outcomes reported.[58] Brugada ECG patterns type 2 and 3 without the clinical features of Brugada syndrome do not warrant limitations in sport activity or further evaluation but may undergo serial follow-up for evidence of Brugada syndrome. Standard workup for ARVC/D includes cardiac MRI, signal-averaged ECG, Holter monitoring, and exercise testing.

Ventricular Preexcitation

The presence of an accessory pathway of conduction between the atrium and ventricle outside the atrioventricular node can predispose patients to atrioventricular reentry tachycardia, as well as very rapid ventricular conduction during other supraventricular tachycardias such as atrial fibrillation. Conduction via the accessory pathway leading to preexcitation of the ventricle can be seen on the ECG as the Wolf-Parkinson-White (WPW) pattern: a delta wave, which is slurring of the initial QRS and a short PR interval (<120 ms). Wolf-Parkinson-White syndrome refers to preexcitation seen on ECG associated with the clinical tachyarrhythmia atrioventricular reentry tachycardia. However, not all patients with the WPW pattern will develop clinical tachycardias, and not all patients with atrioventricular reentry tachycardia will have preexcitation apparent on the surface ECG.

The WPW pattern occurs in ≈ 1 in 1000 persons and follow-up studies have demonstrated a prognosis similar to the general population.[59,60] Most studies using the ECG as part of the PPE have demonstrated a prevalence of ventricular preexcitation pattern in athletes of $\approx 0.1\%$ to 0.3%.[2] The major risk associated with WPW is development of rapidly conducting atrial fibrillation that then degenerates to VF and sudden death. Because athletes are at higher risk for AF than the general population,[61,62] there is greater concern for associated sudden death. In an extensive systematic review of the literature to identify causes of SCD in athletes, Bille et al[63] reported that of 1101 SCDs in athletes <35 years of age, only 1 case of SCD was definitively related to WPW syndrome. To

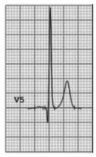
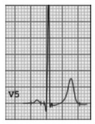
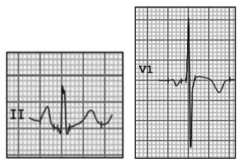
ECG Abnormality	Criteria for further evaluation	Example
Q waves	>3 mm in depth or >40 ms duration in any lead except III, aVR, aVL and V1	
ST depression	>0.5 mm below PR isoelectric line between J-junction and beginning of T waves in V4, V5, V6, I, aVL >1 mm in any lead	
T wave inversion	>1 mm in leads other than III, aVR and V1 (except V2 and V3 in women <25 years)	
Atrial abnormalities	Right: P wave amplitude >2.5 mm Left: i) Negative portion of P wave in V1, V2 of >40 ms duration and 1 mm in depth; or ii) total P wave duration >120 ms	
Right ventricular hypertrophy	>30 years: i) R wave >7 mm in V1; or ii) R/S ratio >1 in V1; or iii) sum of R wave in V1 and S wave in V5 or V6 >10.5 mm <30 years: above plus right atrial enlargement, T wave inversion in V2, V3, or right axis deviation >115°	

Figure 5. Summary of recommendations for screening PPE ECG. PPE indicates preparticipation examination; RAA, right atrial abnormality; LAA, left atrial abnormality; RVH, right ventricular hypertrophy; RAD, right axis deviation; RBBB, right bundle branch block; TWI, T-wave inversion; and QTc, heart-rate correction of the QT interval.

help determine the appropriate treatment strategy in patients with the WPW pattern, several studies have evaluated the clinical outcomes in patients with WPW. Although supraventricular tachycardia is not uncommon in patients with WPW (20%), SCD was rare.[59,60,64–66]

Authors' Recommendation

We recommend further evaluation for athletes with evidence of the WPW pattern, including an echocardiogram.

Comparison With the European Society of Cardiology Document

The ESC authors recommend referral for electrophysiology study in all athletes with evidence of preexcitation. Inducibility of atrioventricular reentry tachycardia and the refractoriness of the accessory pathway are proposed to influence eligibility for athletic competition. Echocardiography is also recommended.

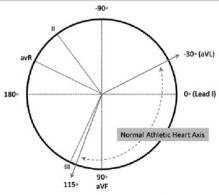
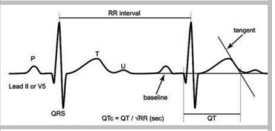
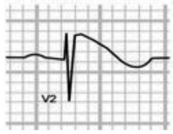
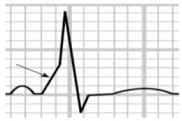
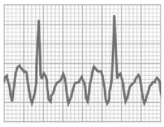
Further Evaluation

The appropriate diagnostic workup of asymptomatic athletes identified with the WPW pattern remains controversial.[67] We recommend an echocardiogram because of its association with cardiomyopathy[68] and rhythm monitoring. Athletes who develop symptoms on close follow-up or have documentation of supraventricular tachycardia should undergo EP study. Given the low rate of sudden death, we do not believe routine EPS should be required to allow continued sports participation. Exercise testing to estimate the refractory period of manifest accessory pathways may be considered, with the assumption that a short refractory period places the patient at higher risk of atrial fibrillation–induced sudden death. However, in light of the low likelihood of sudden death observed in population studies, there is insufficient data to recommend this routinely.

Some experts have a lower threshold for EPS if athletes are involved in activities in which a sudden onset of an arrhythmia might put the athlete or others at risk (for example, swimming, biking, and auto racing).[12]

Ventricular Extrasystoles and Supraventricular Arrhythmia

A common question relates to athletes whose random ECG shows ≥1 premature ventricular contraction (PVC). In contrast with clinical populations where 5% of cardiology clinic patients aged 60 manifest a PVC on a random ECG[69] (10% in heart failure patients[70] and associated with risk) in college athletes, <1% exhibit a PVC on a random ECG. The significance of such a finding is unknown.

LBBB RBBB IVCD	Any QRS >120 ms	
QRS axis deviation	More leftward than -30° More rightward than 115°	
QTc interval	>470 ms in males >480 ms in females <340 ms in any athlete	
Brugada pattern	Presence of Type 1 pattern: coved ST segment in V1 and V2 gradually descending into inverted T wave	
Pre-Excitation	Delta wave and PR interval <120 ms	
Ventricular extrasystoles, heart block, and supraventricular arrhythmia	Atrial fibrillation/flutter, supraventricular tachycardia, complete heart block or ≥2 PVCs in one 12 lead ECG	

RBBB – right bundle branch block; LBBB – left bundle branch block, IVCD – intra- ventricular conduction delay, PVC – premature ventricular contraction. Measurements are by visual analysis.

Figure 5 (Continued).

Authors' Recommendation

Any athlete with documented atrial fibrillation/flutter, supraventricular tachycardia, complete heart block, or ≥2 PVCs on a screening ECG should be referred for further evaluation. Some practitioners believe that PVCs with left bundle branch block configuration coexisting with left-axis deviation are of more concern and should always require further evaluation.

Comparison With the European Society of Cardiology Document

The ESC document does not specifically comment on ventricular extrasystoles.

Further Evaluation

A standard secondary evaluation is indicated (see Q-Wave section) with the inclusion of extended rhythm monitoring. In the absence of structural heart disease or symptomatic syncopal events, patients with ventricular extrasystoles or supraventricular arrhythmias including lone atrial fibrillation should not be limited from competitive activity.

Contiguous Leads

A requirement for findings in 2 contiguous leads formed part of the guidelines for thrombolysis for acute myocardial infarction and has been included in ECG criteria in other areas. As far as we can tell, this is a remnant from the early days of electrocardiography when each lead was only viewed for several seconds and respiratory variation and noise were problematic. Requiring 2 leads lessened false positives though unappreciated effects were to widen the area of involvement and lessen sensitivity. Today, most physicians use computerized systems that average 10 seconds on all leads, making a finding in 1 lead sufficient representation of that area.

Conclusion

The appropriate interpretation of the ECG in young athletes is challenging. False-positive findings created by the application of standard ECG criteria to the interpretation of athletes' ECGs have led to a highly variable rate of secondary testing (8% to 15%).[3,4,13] To reduce the financial and psychological burden of false-positive ECGs, interpretation should be car-

ried out using athlete-specific criteria. We provide in Figure 5 a summary of our approach to interpreting athletic ECGs with examples.

An unappreciated benefit of adding the ECG to the PPE is that ECG abnormalities often lead to intensified questioning of athletes and their family members. A more thorough history can further stratify an athlete's risk before he or she participates in sports. The subsequent clinical, ECG, and echocardiographic evaluation of family members provides further objective data.

In an attempt to standardize the interpretation of ECGs, minimize human error, and reduce the requirement for immediate interpreter expertise, some recommend computer-based algorithms for analysis of ECGs. It should be acknowledged that the available ECG interpretive programs appear to make more exact and reproducible measurements than visual assessment but that their diagnostic statements are often inappropriate. At times, statements from commercial computerized ECG devices cause unnecessary alarm to families, coaches, and primary care physicians. Accordingly, ECG tracings from these systems should not be given to athletes for their own records without considerable editing by an experienced qualified physician.

One of the major limitations in this field is the absence of good data on the validity of specific ECG criteria. For example, although many studies describe ECGs in large populations of athletes, few have directly compared ECG findings in normal athletes to those with inherited cardiovascular diseases such as those known to cause sudden death. These issues along with the need for long-term studies have been discussed previously.[71] One area that has begun to be addressed is cost effectiveness.[3] However, many other areas such as sport-specific differences in sudden death as well as sex- and ethnicity-specific differences in the ECG and in sudden death rates remain poorly understood. Finally, we note that the recommendations detailed in this article represent the consensus opinions of experts from many countries with decades of experience in dealing with athletes and ECGs. Of note, they do not represent an official guideline from any 1 national body.

Disclosures

None.

References

1. Pelliccia A, Fagard R, Bjornstad HH, Anastassakis A, Arbustini E, Assanelli D, Biffi A, Borjesson M, Carre F, Corrado D, Delise P, Dorwarth U, Hirth A, Heidbuchel H, Hoffmann E, Mellwig KP, Panhuyzen-Goedkoop N, Pisani A, Solberg EE, van-Buuren F, Vanhees L, Blomstrom-Lundqvist C, Deligiannis A, Dugmore D, Glikson M, Hoff PI, Hoffmann A, Hoffmann E, Horstkotte D, Nordrehaug JE, Oudhof J, McKenna WJ, Penco M, Priori S, Reybrouck T, Senden J, Spataro A, Thiene G. Recommendations for competitive sports participation in athletes with cardiovascular disease: a consensus document from the Study Group of Sports 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;26:1422–1445.
2. Pelliccia A, Culasso F, Di Paolo FM, Accettura D, Cantore R, Castagna W, Ciacciarelli A, Costini G, Cuffari B, Drago E, Federici V, Gribaudo CG, Iacovelli G, Landolfi L, Menichetti G, Atzeni UO, Parisi A, Pizzi AR, Rosa M, Santelli F, Santilio F, Vagnini A, Casasco M, Di Luigi L. Prevalence of abnormal electrocardiograms in a large, unselected population undergoing pre-participation cardiovascular screening. *Eur Heart J*. 2007;28:2006–2010.
3. Wheeler MT, Heidenreich PA, Froelicher VF, Hlatky MA, Ashley EA. Cost-effectiveness of preparticipation screening for prevention of sudden cardiac death in young athletes. *Ann Intern Med*. 2010;152:276–286.
4. Maron BJ, Thompson PD, Ackerman MJ, Balady G, Berger S, Cohen D, Dimeff R, Douglas PS, Glover DW, Hutter AM Jr, Krauss MD, Maron MS, Mitten MJ, Roberts WO, Puffer JC. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: A Scientific Statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: Endorsed by the American College of Cardiology Foundation. *Circulation*. 2007;115:1643–1455.
5. Kligfield P, Gettes LS, Bailey JJ, Childers R, Deal BJ, Hancock EW, van Herpen G, Kors JA, Macfarlane P, Mirvis DM, Pahlm O, Rautaharju P, Wagner GS, Josephson M, Mason JW, Okin P, Surawicz B, Wellens H. Recommendations for the standardization and interpretation of the electrocardiogram: part I: the electrocardiogram and its technology: 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. *J Am Coll Cardiol*. 2007;49:1109–1127.
6. Wagner GS, Macfarlane P, Wellens H, Josephson M, Gorgels A, Mirvis DM, Pahlm O, Surawicz B, Kligfield P, Childers R, Gettes LS, Bailey JJ, Deal BJ, Gorgels A, Hancock EW, Kors JA, Mason JW, Okin P, Rautaharju PM, van Herpen G. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part VI: acute ischemia/infarction: 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. *J Am Coll Cardiol*. 2009;53:1003–1011.
7. Hancock EW, Deal BJ, Mirvis DM, Okin P, Kligfield P, Gettes LS, Bailey JJ, Childers R, Gorgels A, Josephson M, Kors JA, Macfarlane P, Mason JW, Pahlm O, Rautaharju PM, Surawicz B, van Herpen G, Wagner GS, Wellens H. 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. *J Am Coll Cardiol*. 2009;53:992–1002.
8. Surawicz B, Childers R, Deal BJ, Gettes LS, Bailey JJ, Gorgels A, Hancock EW, Josephson M, Kligfield P, Kors JA, Macfarlane P, Mason JW, Mirvis DM, Okin P, Pahlm O, Rautaharju PM, van Herpen G, Wagner GS, Wellens H. 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. *J Am Coll Cardiol*. 2009;53:976–981.
9. Rautaharju PM, Surawicz B, Gettes LS, Bailey JJ, Childers R, Deal BJ, Gorgels A, Hancock EW, Josephson M, Kligfield P, Kors JA, Macfarlane P, Mason JW, Mirvis DM, Okin P, Pahlm O, van Herpen G, Wagner GS, Wellens H. 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:e241–e250.
10. Hancock EW, Deal BJ, Mirvis DM, Okin P, Kligfield P, Gettes LS, Bailey JJ, Childers R, Gorgels A, Josephson M, Kors JA, Macfarlane P, Mason JW, Pahlm O, Rautaharju PM, Surawicz B, van Herpen G, Wagner GS, Wellens H. 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:e251–e261.
11. Zipes DP, Ackerman MJ, Estes NA III, Grant AO, Myerburg RJ, Van Hare G. Task force 7: Arrhythmias. *J Am Coll Cardiol*. 2005;45: 1354–1363.
12. Pelliccia A, Zipes DP, Maron BJ. Bethesda Conference 36 and the European Society of Cardiology consensus recommendations revisited: a comparison of US and European criteria for eligibility and disqualification of competitive athletes with cardiovascular abnormalities. *J Am Coll Cardiol*. 2008;52:1990–1996.
13. Corrado D, Pelliccia A, Heidbuchel H, Sharma S, Link M, Basso C, Biffi A, Buja G, Delise P, Gussac I, Anastasakis A, Borjesson M, Bjornstad HH, Carre F, Deligiannis A, Dugmore D, Fagard R, Hoogsteen J, Mellwig KP, Panhuyzen-Goedkoop N, Solberg E, Vanhees L, Drezner J, Estes NA, III, Iliceto S, Maron BJ, Peidro R, Schwartz PJ, Stein R, Thiene G, Zeppilli P, McKenna WJ. Recommendations for interpretation of 12-lead electrocardiogram in the athlete. *Eur Heart J*. 2010;31:243–259.
14. Pelliccia A, Maron BJ, Culasso F, Di Paolo FM, Spataro A, Biffi A, Caselli G, Piovano P. Clinical significance of abnormal electrocardiographic patterns in trained athletes. *Circulation*. 2000;102:278–284.
15. Le VV, Wheeler MT, Mandic S, Dewey F, Fonda H, Perez M, Sungar G, Garza D, Ashley EA, Matheson G, Froelicher V. Addition of the electrocardiogram to the preparticipation examination of college athletes. *Clin J Sport Med*. 2010;20:98–105.
16. Rawlins J, Carre F, Kervio G, Papadakis M, Chandra N, Edwards C, Whyte GP, Sharma S. Ethnic differences in physiological cardiac adaptation to intense physical exercise in highly trained female athletes. *Circulation*. 2010;121:1078–1085.
17. Ashley EA, Raxwal VK, Froelicher VF. The prevalence and prognostic significance of electrocardiographic abnormalities. *Curr Probl Cardiol*. 2000;25:1–72.
18. Hsieh BP, Pham MX, Froelicher VF. Prognostic value of electrocardiographic criteria for left ventricular hypertrophy. *Am Heart J*. 2005;150: 161–167.
19. Cappato R, Furlanello F, Giovinazzo V, Infusino T, Lupo P, Pittalis M, Foresti S, De Ambroggi G, Ali H, Bianco E, Riccamboni R, Butera G, Ricci C, Ranucci M, Pelliccia A, De Ambroggi L. J wave, QRS slurring, and ST elevation in athletes with cardiac arrest in the absence of heart disease: marker of risk or innocent bystander? *Circ Arrhythm Electrophysiol*. 2010;3:305–311.
20. Haissaguerre M, Derval N, Sacher F, Jesel L, Deisenhofer I, de Roy L, Pasquie JL, Nogami A, Babuty D, Yli-Mayry S, De Chillou C, Scanu P, Mabo P, Matsuo S, Probst V, Le Scouarnec S, Defaye P, Schlaepfer J, Rostock T, Lacroix D, Lamaison D, Lavergne T, Aizawa Y, Englund A, Anselme F, O'Neill M, Hocini M, Lim KT, Knecht S, Veenhuyzen GD, Bordachar P, Chauvin M, Jais P, Coureau G, Chene G, Klein GJ, Clementy J. Sudden cardiac arrest associated with early repolarization. *N Engl J Med*. 2008;358:2016–2023.
21. Tikkanen JT, Anttonen O, Junttila MJ, Aro AL, Kerola T, Rissanen HA, Reunanen A, Huikuri HV. Long-term outcome associated with early repolarization on electrocardiography. *N Engl J Med*. 2009;361: 2529–2537.
22. Sinner MF, Reinhard W, Muller M, Beckmann BM, Martens E, Perz S, Pfeufer A, Winogradow J, Stark K, Meisinger C, Wichmann HE, Peters A, Riegger GA, Steinbeck G, Hengstenberg C, Kaab S. Association of early repolarization pattern on ECG with risk of cardiac and all-cause mortality: a population-based prospective cohort study (MONICA/KORA). *PLoS Medicine.* 2010;7:e1000314.
23. 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;39:1033–1038.
24. Ammar KA, Kors JA, Yawn BP, Rodeheffer RJ. Defining unrecognized myocardial infarction: a call for standardized electrocardiographic diagnostic criteria. *Am Heart J*. 2004;148:277–284.
25. Ammar KA, Yawn BP, Urban L, Mahoney DW, Kors JA, Jacobsen S, Rodeheffer RJ. Identification of optimal electrocardiographic criteria for the diagnosis of unrecognized myocardial infarction: a population-based study. *Ann Noninvasive Electrocardiol*. 2005;10:197–205.
26. Papavassiliu T, Fluchter S, Haghi D, Suselbeck T, Wolpert C, Dinter D, Kuhl H, Borggrefe M. Extent of myocardial hyperenhancement on late gadolinium-enhanced cardiovascular magnetic resonance correlates with Q waves in hypertrophic cardiomyopathy. *J Cardiovasc Magn Reson*. 2007;9:595–603.
27. Konno T, Shimizu M, Ino H, Yamaguchi M, Terai H, Uchiyama K, Oe K, Mabuchi T, Kaneda T, Mabuchi H. Diagnostic value of abnormal Q waves for identification of preclinical carriers of hypertrophic cardiomyopathy based on a molecular genetic diagnosis. *Eur Heart J*. 2004;25: 246–251.
28. Charron P, Forissier JF, Amara ME, Dubourg O, Desnos M, Bouhour JB, Isnard R, Hagege A, Benaiche A, Richard P, Schwartz K, Komajda M. Accuracy of European diagnostic criteria for familial hypertrophic cardiomyopathy in a genotyped population. *Int J Cardiol.* 2003;90:33–38; discussion 38–40.
29. Ostman-Smith I, Wisten A, Nylander E, Bratt EL, Granelli AW, Oulhaj A, Ljungstrom E. Electrocardiographic amplitudes: a new risk factor for sudden death in hypertrophic cardiomyopathy. *Eur Heart J*. 2010;31: 439–449.
30. Ho CY, Sweitzer NK, McDonough B, Maron BJ, Casey SA, Seidman JG, Seidman CE, Solomon SD. Assessment of diastolic function with Doppler tissue imaging to predict genotype in preclinical hypertrophic cardiomyopathy. *Circulation*. 2002;105:2992–2997.
31. Sharma S, Elliott P, Whyte G, Jones S, Mahon N, Whipp B, McKenna WJ. Utility of cardiopulmonary exercise in the assessment of clinical determinants of functional capacity in hypertrophic cardiomyopathy. *Am J Cardiol*. 2000;86:162–168.
32. Maron BJ, Pelliccia A, Spataro A, Granata M. Reduction in left ventricular wall thickness after deconditioning in highly trained Olympic athletes. *Br Heart J*. 1993;69:125–128.
33. Rotman M, Triebwasser JH. A clinical and follow-up study of right and left bundle branch block. *Circulation*. 1975;51:477–484.
34. Bongioanni S, Bianchi F, Migliardi A, Gnavi R, Pron PG, Casetta M, Conte MR. Relation of QRS duration to mortality in a community-based cohort with hypertrophic cardiomyopathy. *Am J Cardiol*. 2007;100: 503–506.
35. Desai AD, Yaw TS, Yamazaki T, Kaykha A, Chun S, Froelicher VF. Prognostic significance of quantitative QRS duration. *Am J Med*. 2006; 119:600–606.
36. Crouse SF, Meade T, Hansen BE, Green JS, Martin SE. Electrocardiograms of collegiate football athletes. *Clin Cardiol*. 2009;32:37–42.
37. Mason JW, Ramseth DJ, Chanter DO, Moon TE, Goodman DB, Mendzelevski B. Electrocardiographic reference ranges derived from 79 743 ambulatory subjects. *J Electrocardiol*. 2007;40:228–234.
38. Sharma S, Whyte G, Elliott P, Padula M, Kaushal R, Mahon N, McKenna WJ. Electrocardiographic changes in 1000 highly trained junior elite athletes. *Br J Sports Med*. 1999;33:319–324.
39. Somauroo JD, Pyatt JR, Jackson M, Perry RA, Ramsdale DR. An echocardiographic assessment of cardiac morphology and common ECG findings in teenage professional soccer players: reference ranges for use in screening. *Heart*. 2001;85:649–654.
40. Pelliccia A, Di Paolo FM, Quattrini FM, Basso C, Culasso F, Popoli G, De Luca R, Spataro A, Biffi A, Thiene G, Maron BJ. Outcomes in athletes with marked ECG repolarization abnormalities. *N Engl J Med*. 2008;358: 152–161.
41. Schmied C, Zerguini Y, Junge A, Tscholl P, Pelliccia A, Mayosi BM, Dvorak J. Cardiac findings in the precompetition medical assessment of football players participating in the 2009 African under-17 championships in Algeria. *Br J Sports Med*. 2009;43:716–721.
42. Haghjoo M, Mohammadzadeh S, Taherpour M, Faghfurian B, Fazelifar AF, Alizadeh A, Rad MA, Sadr-Ameli MA. ST-segment depression as a risk factor in hypertrophic cardiomyopathy. *Europace*. 2009;11:643–649.
43. Funck-Brentano C, Jaillon P. Rate-corrected QT interval: techniques and limitations. *Am J Cardiol.* 1993;72:17B–22B.
44. Kligfield P, Hancock EW, Helfenbein ED, Dawson EJ, Cook MA, Lindauer JM, Zhou SH, Xue J. Relation of QT interval measurements to evolving automated algorithms from different manufacturers of electrocardiographs. *Am J Cardiol*. 2006;98:88–92.
45. Postema PG, De Jong JS, Van der Bilt IA, Wilde AA. Accurate electrocardiographic assessment of the QT interval: teach the tangent. *Heart Rhythm*. 2008;5:1015–1018.
46. Johnson JN, Ackerman MJ. QTc: how long is too long? *Br J Sports Med*. 2009;43:657–662.
47. 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;327:846–852.
48. Anttonen O, Junttila MJ, Rissanen H, Reunanen A, Viitasalo M, Huikuri HV. Prevalence and prognostic significance of short QT interval in a middle-aged Finnish population. *Circulation*. 2007;116:714–720.

49. Viskin S, Postema PG, Bhuiyan ZA, Rosso R, Kalman JM, Vohra JK, Guevara-Valdivia ME, Marquez MF, Kogan E, Belhassen B, Glikson M, Strasberg B, Antzelevitch C, Wilde AA. The response of the QT interval to the brief tachycardia provoked by standing: a bedside test for diagnosing long QT syndrome. *J Am Coll Cardiol*. 2010;55:1955–1961.
50. Antzelevitch C, Brugada P, Borggrefe M, Brugada J, Brugada R, Corrado D, Gussak I, LeMarec H, Nademanee K, Perez Riera AR, Shimizu W, Schulze-Bahr E, Tan H, Wilde A. Brugada syndrome: report of the Second Consensus Conference. Endorsed by the Heart Rhythm Society and the European Heart Rhythm Association. *Circulation*. 2005;111: 659–670.
51. Marcus FI, McKenna WJ, Sherrill D, Basso C, Bauce B, Bluemke DA, Calkins H, Corrado D, Cox MG, Daubert JP, Fontaine G, Gear K, Hauer R, Nava A, Picard MH, Protonotarios N, Saffitz JE, Sanborn DM, Steinberg JS, Tandri H, Thiene G, Towbin JA, Tsatsopoulou A, Wichter T, Zareba W. Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia: proposed modification of the task force criteria. *Circulation*. 2010;121:1533–1541.
52. Gemayel C, Pelliccia A, Thompson PD. Arrhythmogenic right ventricular cardiomyopathy. *J Am Coll Cardiol*. 2001;38:1773–1781.
53. Nasir K, Bomma C, Tandri H, Roguin A, Dalal D, Prakasa K, Tichnell C, James C, Spevak PJ, Marcus F, Calkins H. Electrocardiographic features of arrhythmogenic right ventricular dysplasia/cardiomyopathy according to disease severity: a need to broaden diagnostic criteria. *Circulation*. 2004;110:1527–1534.
54. Holst AG, Winkel BG, Theilade J, Kristensen IB, Thomsen JL, Ottesen GL, Svendsen JH, Haunso S, Prescott E, Tfelt-Hansen J. Incidence and etiology of sports-related sudden cardiac death in Denmark: implications for preparticipation screening. *Heart Rhythm*. 2010;7:1365–1371.
55. de Noronha SV, Sharma S, Papadakis M, Desai S, Whyte G, Sheppard MN. Aetiology of sudden cardiac death in athletes in the United Kingdom: a pathological study. *Heart (British Cardiac Society)*. 2009;95: 1409–1414.
56. Eckart RE, Scoville SL, Campbell CL, Shry EA, Stajduhar KC, Potter RN, Pearse LA, Virmani R. Sudden death in young adults: a 25-year review of autopsies in military recruits. *Ann Intern Med*. 2004;141: 829–834.
57. Wilde AA, Antzelevitch C, Borggrefe M, Brugada J, Brugada R, Brugada P, Corrado D, Hauer RN, Kass RS, Nademanee K, Priori SG, Towbin JA. Proposed diagnostic criteria for the Brugada syndrome. *Eur Heart J*. 2002;23:1648–1654.
58. Lee C, Soni A, Tate RB, Cuddy TE. The incidence and prognosis of Brugada electrocardiographic pattern in the Manitoba Follow-Up Study. *Can J Cardiol*. 2005;21:1286–1290.
59. Munger TM, Packer DL, Hammill SC, Feldman BJ, Bailey KR, Ballard DJ, Holmes DR Jr, Gersh BJ. A population study of the natural history of Wolff-Parkinson-White syndrome in Olmsted County, Minnesota, 1953–1989. *Circulation*. 1993;87:866–873.
60. Fitzsimmons PJ, McWhirter PD, Peterson DW, Kruyer WB. The natural history of Wolff-Parkinson-White syndrome in 228 military aviators: a long-term follow-up of 22 years. *Am Heart J*. 2001;142:530–536.
61. Grimsmo J, Grundvold I, Maehlum S, Arnesen H. High prevalence of atrial fibrillation in long-term endurance cross-country skiers: Echocardiographic findings and possible predictors: a 28–30 years follow-up study. *Eur J Cardiovasc Prev Rehabil*. 2011;17:100–105.
62. Molina L, Mont L, Marrugat J, Berruezo A, Brugada J, Bruguera J, Rebato C, Elosua R. Long-term endurance sport practice increases the incidence of lone atrial fibrillation in men: a follow-up study. *Europace*. 2008;10:618–623.
63. Bille K, Figueiras D, Schamasch P, Kappenberger L, Brenner JI, Meijboom FJ, Meijboom EJ. Sudden cardiac death in athletes: the Lausanne Recommendations. *Eur J Cardiovasc Prev Rehabil*. 2006;13: 859–875.
64. Goudevenos JA, Katsouras CS, Graekas G, Argiri O, Giogiakas V, Sideris DA. Ventricular pre-excitation in the general population: a study on the mode of presentation and clinical course. *Heart*. 2000;83:29–34.
65. Timmermans C, Smeets JL, Rodriguez LM, Vrouchos G, van den Dool A, Wellens HJ. Aborted sudden death in the Wolff-Parkinson-White syndrome. *Am J Cardiol*. 1995;76:492–494.
66. Klein GJ, Prystowsky EN, Yee R, Sharma AD, Laupacis A. Asymptomatic Wolff-Parkinson-White: should we intervene? *Circulation*. 1989;80: 1902–1905.
67. Sarubbi B. The Wolff-Parkinson-White electrocardiogram pattern in athletes: how and when to evaluate the risk for dangerous arrhythmias: the opinion of the paediatric cardiologist. *J Cardiovasc Med (Hagerstown)*. 2006;7:271–278.
68. Arad M, Maron BJ, Gorham JM, Johnson WH Jr, Saul JP, Perez-Atayde AR, Spirito P, Wright GB, Kanter RJ, Seidman CE, Seidman JG. Glycogen storage diseases presenting as hypertrophic cardiomyopathy. *N Engl J Med*. 2005;352:362–372.
69. Engel G, Cho S, Ghayoumi A, Yamazaki T, Chun S, Fearon WF, Froelicher VF. Prognostic significance of PVCs and resting heart rate. *Ann Noninvasive Electrocardiol*. 2007;12:121–129.
70. Le VV, Mitiku T, Hadley D, Myers J, Froelicher VF. Rest premature ventricular contractions on routine ECG and prognosis in heart failure patients. *Ann Noninvasive Electrocardiol*. 2010;15:56–62.
71. Myerburg RJ, Vetter VL. Electrocardiograms should be included in preparticipation screening of athletes. *Circulation*. 2007;116:2616–2626; discussion 2626.