The ECGs that will be read around the world—and save lives of sportspeople

Karim M Khan

‘Having a heart attack? There’s an iPhone App for that!’ is a true story. We congratulate its inventor Dr David Albert. This issue of BJSM contains advances that are equally exciting and which deserve as much media attention. Sports cardiology is one defining element of the sport and exercise medicine specialty (http://bjsm.bmj.com/content/46/Suppl_1/i2.full). Every one of us has seen an athlete drop dead on film—if not in real life. Whose heart has not stopped on each such occasion? “There but for the grace of God go I” as physician, athlete, or parent. “Houston, we have buy in.”

Sports cardiology is a BJSM ‘priority topic’. We cover priority topics in each of our three ‘pillars’—research, education and implementation. Another way of labelling the ‘pillars’ is innovation, education and knowledge translation. We use BJSM and partner ‘platforms’ (eg, see Partnering with BMJ Learning section) to deliver the content. Think of pages such as this one you are reading, the education tab on the BJSM home page with its popular podcasts such as those by Mathew Wilson, Sanjay Sharma, Jonathan Drezner, Jiri Dvorak with Jonathan Tobin, etc. Helping to alert the BJSM community to those riches is our Twitter feed (@BJSM_BMJ) and Facebook page. And our Senior Associate Editor, Evert Verhagen is tasked with developing BJSM’s Apps. He will not start with a sports cardiology App but BJSM will land a couple of Apps on this planet before the end of the calendar year.

PARTNERING WITH BMJ LEARNING

A worldwide team led by Jonathan Drezner (AMSSM) and listed in the ‘Seattle Criteria’ paper (bjsports-2012-092067) produced the four key papers in this issue—four papers that examine ECG findings in sports people with more rigour than has previously been managed. The experts assembled in Seattle in 2012, they had the compelling goal of clarifying the complexity of ECG interpretation in athletes among different races to distinguish physiological from pathological findings. The material is very logically dished out in four courses: (1) evidence of electrical disease (bjsports-2012-092070), (2) evidence of cardiomyopathy (bjsports-2012-092069), (3) the ‘Seattle Criteria’ and (4) the old chestnut now with new clarity—the ECG changes that are physiological in athletes (bjsports-2012-092068). A great innovation of this team was to embed ‘implementation’ into the plan from baseline. Thus, each of the papers provides the foundation for online education which will be available (in English initially) for the entire world to view. Because of the generosity of sponsors—such as AMSSM, and FIFA, this material will be free worldwide. The sports cardiology team is partnering with BMJ Learning—a branch of the BMJ Group dedicated to CME in the imaginative, broad sense of that term. Follow your favourite mobile sites such as AMSSM, FIFA or BJSM among others—to be apprised of this free content as soon as it is available.

PARTNERING WITH THE BMJ

The BMJ is deservedly an iconic brand and BJSM is delighted to reprint key sport and exercise medicine papers from recent BMJ issues. We launched this in 2008 but had to take a break to clear some backlog. Now with 18 BJSM issues a year, the best of BMJ is back for good! (bjsports-2012-e2672rep). And remember BJSM’s active bloggers—among them the evocative and provocative Domhnall MacAuley (@DMacA).

WHAT’S IN A NAME 50 YEARS LATER?

A year from now, 2014, would have marked the 50th anniversary of the ‘Bulletin of the British Association of Sport and Medicine’ had not some iconoclasts decided that a Journal was better than a Bulletin. The 1964 ‘Bulletin of the British Association of Sport and Medicine’ (http://bjsm.bmj.com/content/1/1/1.full.pdf+html) has had various iterations to reach its present title of BJSM. In today’s sport and exercise medicine setting, BJSM’s lack of ‘E’ seems anachronistic. Britain’s BASM readily become BASEM, Canada’s CASM equally CASEM. The title of BJSM mirrors neither the co-owner society name (BASEM) nor does it reflect the paradigm shift that has justified sport and exercise medicine specialty status in many countries. In 2013, Exercise is Medicine. Would ‘BJSEM’ make more sense and more accurately signal to patients, policy-makers and professional colleagues that our field of medicine embraces exercise (and physical activity)? Is it time to advance, walk on the spot, or really embrace our past and revert to a mighty ‘Bulletin’ again? If it’s time to advance, then innovators would ‘open the diamond’ and think bravely—consider options that would add member society value (ie, greater reach and brand value), before refining and making a new choice. The 2013 BJSM Management Meeting heard that BASEM considers BJSM’s ratio of ‘sports medicine’ content to ‘exercise medicine’ about right. The editors have heard that and are listening. But as for a journal title, the BASEM executive welcomes members’ comments on how our sport and exercise medicine community thinks we should ring in our 50th year, so let us know. You can Tweet (@BJSM_BMJ), post a message on Facebook, email, dial, or use Royal Mail.

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Electrocardiographic interpretation in athletes: the ‘Seattle Criteria’


This document was developed in collaboration between the American Medical Society for Sports Medicine (AMSSM), the Section on Sports Cardiology of the European Association for Cardiovascular Prevention and Rehabilitation (EACPR), a registered branch of the European Society of Cardiology (ESC), the FIFA Medical Assessment and Research Center (F-MARC), and the Pediatric & Congenital Electrophysiology Society (PACES).

ABSTRACT

Sudden cardiac death (SCD) is the leading cause of death in athletes during sport. Whether obtained for screening or diagnostic purposes, an ECG increases the ability to detect underlying cardiovascular conditions that may increase the risk for SCD. In most countries, there is a shortage of physician expertise in the interpretation of an athlete’s ECG. A critical need exists for physician education in modern ECG interpretation that distinguishes normal physiological adaptations in athletes from abnormal findings suggestive of pathology. On 13–14 February 2012, an international group of experts in sports cardiology and sports medicine convened in Seattle, Washington, to define contemporary standards for ECG interpretation in athletes. The objective of the meeting was to develop a comprehensive training resource to help physicians distinguish normal ECG alterations in athletes from abnormal ECG findings that require additional evaluation for conditions associated with SCD.

INTRODUCTION

Cardiovascular-related sudden death is the leading cause of mortality in athletes during sport. The majority of disorders associated with increased risk of sudden cardiac death (SCD), such as cardiomyopathies and primary electrical diseases, are suggested by abnormal findings present on a 12-lead ECG. ECG interpretation 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 diagnostic 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 important skill for physicians involved in the cardiovascular care of 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 lethal cardiovascular disorder from benign physiological adaptations occurring as the result of regular, intense training (ie, athlete’s heart). Several reports have outlined contemporary ECG criteria intended to distinguish normal ECG findings in athletes from ECG abnormalities requiring additional evaluation. Despite the publication of these consensus guidelines, most sports medicine and cardiology training programmes lack a standard educational curriculum on ECG interpretation in athletes.

THE IMPACT OF STANDARDISED CRITERIA

Studies demonstrate that without further education the ability of many physicians to accurately interpret an athlete’s ECG is relatively poor and may lead to an unacceptable rate of false-negative interpretations and unnecessary secondary evaluations. However, providing physicians standardised criteria with which to evaluate an ECG considerably improves accuracy.

SUMMIT ON ECG INTERPRETATION IN ATHLETES

On 13–14 February 2012, the American Medical Society for Sports Medicine (AMSSM) co-sponsored by the FIFA Medical Assessment and Research Center (F-MARC) held a ‘Summit on Electrocardiogram Interpretation in Athletes’ in Seattle, Washington. Partnering medical societies included the European Society of Cardiology (ESC) Sports Cardiology Subsection and the Pediatric & Congenital Electrophysiology Society (PACES), as well as other leading cardiologists on ECG.
interpretation in athletes from the USA, Europe and around the world. The goals of the summit meeting were to:

1. define ECG interpretation standards to help physicians distinguish normal ECG alterations in athletes from abnormal ECG findings that require additional evaluation for conditions associated with SCD;
2. outline recommendations for the initial evaluation of ECG abnormalities suggestive of a pathological cardiovascular disorder; and
3. assemble this information into a comprehensive resource and online training course targeted for physicians around the world to gain expertise and competence in ECG interpretation.

The consensus recommendations developed are presented in three papers:

4. Normal Electrocardiographic Findings: Recognizing Physiologic Adaptations in Athletes
5. Abnormal Electrocardiographic Findings in Athletes: Recognizing Changes Suggestive of Cardiomyopathy
6. Abnormal Electrocardiographic Findings in Athletes: Recognizing Changes Suggestive of Primary Electrical Disease

Box 1 summarises a list of normal ECG findings in athletes that are considered physiological adaptations to regular exercise and do not require further evaluation. Table 1 summarises a list of abnormal ECG findings unrelated to athletic training that may suggest the presence of a pathological cardiac disorder and should trigger additional evaluation in an athlete.

### ONLINE E-LEARNING ECG TRAINING MODULE—FREE!

The Seattle Criteria will be used to develop a comprehensive online training module for physicians to acquire a common foundation in ECG interpretation in athletes. This state of the art E-learning resource provides additional ECG examples, figures and explanations, and is prepared in a user friendly educational format to optimise learning. This online training module is accessible at no cost to any physician world-wide at: http://learning.bmj.com/ECGathlete

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<table>
<thead>
<tr>
<th>Abnormal ECG finding</th>
<th>Definition</th>
</tr>
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<tbody>
<tr>
<td>T-wave inversion</td>
<td>&gt;1 mm in depth in two or more leads V2–V6, II and aVF, or I and AVL (excludes III, aVR and V1)</td>
</tr>
<tr>
<td>ST segment depression</td>
<td>≥0.5 mm in depth in two or more leads</td>
</tr>
<tr>
<td>Pathologic Q waves</td>
<td>&gt;3 mm in depth or &gt;40 ms in duration in two or more leads (except for III and aVR)</td>
</tr>
<tr>
<td>Complete left bundle</td>
<td>QRS ≥120 ms, predominantly negative QRS complex in lead V1 (Q5 or rS), and upright monophasic R wave in leads I and V6</td>
</tr>
<tr>
<td>branch block</td>
<td></td>
</tr>
<tr>
<td>Intraventricular</td>
<td>Any QRS duration ≥140 ms</td>
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<tr>
<td>conduction delay</td>
<td></td>
</tr>
<tr>
<td>Left axis deviation</td>
<td>–30° to +90°</td>
</tr>
<tr>
<td>Left atrial enlargement</td>
<td>P wave duration of &gt;120 ms in leads I or II with negative portion of the P wave ≥110 ms in depth and ≥40 ms in duration in lead V1</td>
</tr>
<tr>
<td>Right ventricular</td>
<td>R-V1+S–V5+ &gt;150 ms AND right axis deviation</td>
</tr>
<tr>
<td>hypertrophy pattern</td>
<td>&gt;120°</td>
</tr>
<tr>
<td>Ventricular pre-excitation</td>
<td>PR interval &lt;120 ms with a delta wave (steepest upstroke in the QRS complex) and wide QRS (&gt;120 ms)</td>
</tr>
<tr>
<td>Long QT interval*</td>
<td>QTc ≥470 ms (male)</td>
</tr>
<tr>
<td></td>
<td>QTc ≤480 ms (female)</td>
</tr>
<tr>
<td></td>
<td>QTc ≤500 ms (marked QT prolongation)</td>
</tr>
<tr>
<td>Short QT interval*</td>
<td>QTc ≤320 ms</td>
</tr>
<tr>
<td>Brugada-like ECG pattern</td>
<td>High take-off and downsloping ST segment elevation followed by a negative T wave in ≥2 leads in V1–V3</td>
</tr>
<tr>
<td>Profound sinus bradycardia</td>
<td>&lt;30 BPM or sinus pauses ≥3 s</td>
</tr>
<tr>
<td>Atrial tachyarrhythmias</td>
<td>Supraventricular tachycardia, atrial-fibrillation, atrial-flutter</td>
</tr>
<tr>
<td>Premature ventricular contractions</td>
<td>≥2 PVCs per 10 s tracing</td>
</tr>
<tr>
<td>Ventricular arrhythmias</td>
<td>Couplets, triplets and non-sustained ventricular tachycardia</td>
</tr>
</tbody>
</table>

Note: These ECG findings are unrelated to regular training or expected physiological adaptation to exercise, may suggest the presence of pathological cardiovascular disease, and require further diagnostic evaluation.

*The QT interval corrected for heart rate is ideally measured with heart rates of 60–90 bpm. Consider repeating the ECG after mild aerobic activity for borderline or abnormal QTc values with a heart rate <50 bpm.

### LIMITATIONS OF THE SEATTLE CRITERIA

While the ECG increases the ability to detect underlying cardiovascular conditions that place athletes at increased risk, ECG as a diagnostic tool has limitations in both sensitivity and specificity. Even if properly interpreted, an ECG will not detect all conditions predisposing to SCD. In addition, the true prevalence of specific ECG parameters in athletes and in diseases that predispose to SCD is often unknown and requires further study. The Seattle Criteria were developed with thoughtful attention to balance sensitivity (disease detection) and specificity (false-positives), while maintaining a clear and usable checklist of findings to guide ECG interpretation for physicians, including new learners.

The criteria define ECG findings that warrant further cardiovascular evaluation for disorders that predispose to SCD. The criteria were developed with consideration of ECG interpretation in the context of an asymptomatic athlete age 14–35. An athlete is defined as an individual who engages in regular exercise or training for sport or general physical fitness, typically with a goal of improving performance. In the presence of...
personal cardiac symptoms or a family history that is positive for genetic cardiovascular disease or premature SCD, the criteria may require modification. Physicians also may choose to deviate from consensus standards based on their experience or practice setting.

The evaluation of ECG abnormalities is performed ideally in consultation with a specialist with knowledge and experience in athlete’s heart and disorders associated with SCD in young athletes. As new scientific data become available, revision of the criteria may further improve the accuracy of ECG interpretation within the athletic population.

CONCLUSIONS
Prevention of SCD in athletes remains a highly visible topic in sports medicine and cardiology. Cardiac adaptation and remodelling from regular athletic training produces common ECG alterations that could be mistaken as abnormal. Whether performed for screening or diagnostic purposes as part of the cardiac evaluation in athletes, 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 ECG interpretation guidelines presented and the online training programme serve as an important foundation for improving the quality of ECG interpretations and the cardiovascular care of athletes.

Additional resources
For a free online training module on ECG interpretation in athletes, please visit: http://learning.bmj.com/ECGathlete. For the November 2012 BJSM supplement on “Advances in Sports Cardiology,” please visit: http://bsjm.bmj.com/content/46/Suppl_1.toc

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Normal electrocardiographic findings: recognising physiological adaptations in athletes

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This document was developed in collaboration between the American Medical Society for Sports Medicine (AMSSM), the Section on Sports Cardiology of the European Association for Cardiovascular Prevention and Rehabilitation (EACPR), a registered branch of the European Society of Cardiology (ESC), the FIFA Medical Assessment and Research Center (F-MARC), and the Pediatric & Congenital Electrophysiology Society (PACES).

ABSTRACT
Electrocardiographic changes in athletes are common and usually reflect benign structural and electrical remodelling of the heart as a physiological adaptation to regular and sustained physical training (athlete’s heart). The ability to identify an abnormality on the 12-lead ECG, suggestive of underlying cardiac disease associated with sudden cardiac death (SCD), is based on a sound working knowledge of the normal ECG characteristics within the athletic population. This document will assist physicians in identifying normal ECG patterns commonly found in athletes. The ECG findings presented as normal in athletes were established by an international consensus panel of experts in sports cardiology and sports medicine.

INTRODUCTION
Sudden death from intrinsic cardiac conditions remains the leading cause of mortality in athletes during sport. A resting 12-lead ECG is utilised as a diagnostic tool in the evaluation of both symptomatic and asymptomatic athletes for conditions associated with sudden cardiac death (SCD). The purpose of pre-participation cardiovascular screening is to provide medical clearance for participation in sport through routine systematic evaluations intended to identify pre-existing cardiovascular abnormalities, and thereby reduce the potential for adverse cardiac events and loss of life. Many pre-participation screening programmes include an ECG. Physicians responsible for the cardiovascular care of athletes should be knowledgeable of the physiological cardiac adaptations to regular exercise that are manifested on the ECG.

ECG changes in athletes are common and usually reflect the electrical and structural remodelling or autonomic nervous system adaptations that occur as a consequence of regular and sustained physical activity (ie, athlete’s heart). In fact, up to 60% of athletes demonstrate ECG changes (in isolation or in combination) such as sinus bradycardia, sinus arrhythmia, first-degree atrioventricular (AV) block, early repolarisation, incomplete right bundle branch block (IRBBB) and voltage criteria for left ventricular hypertrophy (LVH). The extent of these changes is also dependent on the athlete’s ethnicity, age, gender, sporting discipline and level of training and competition. Accordingly, the ability to identify an abnormal ECG suggestive of underlying cardiac disease is based on a sound understanding of ECG normality within a broad spectrum of athletic populations.

Concerns for the physician when interpreting an athlete’s ECG include both missing a dangerous cardiac condition and generating false-positive interpretations that cause needless further investigations, increased economic cost and potentially unnecessary activity restriction for the athlete. This paper focuses on the physiological ECG adaptations commonly found in athletes to help physicians distinguish normal ECG changes from abnormal ECG findings related to a pathological cardiac condition associated with SCD. Abnormal ECG findings in athletes suggestive of underlying cardiac disease are presented separately.

OVERVIEW OF ATHLETE’S HEART
Regular and long-term participation in intensive exercise (minimum of 4 h/week) is associated with unique electrical manifestations that reflect increased vagal tone and enlarged cardiac chamber size. These ECG findings in athletes are considered normal, physiological adaptations to regular exercise and do not require further evaluation (box 1).

Increased vagal tone
Common consequences of increased vagal tone include sinus bradycardia, sinus arrhythmia and early repolarisation (figure 1). Other, less common markers of increased vagal tone are first-degree AV block and Mobitz type I second-degree AV block. Sinus bradycardia is defined as a heart rate of <60 beats/min and is present in up to 80% of highly trained athletes. Heart rates ≥60 beats/min are considered normal in highly trained athletes. Sinus arrhythmia is also common, particularly in younger athletes.
Early repolarisation consists of concave ST segment elevation most commonly observed in the precordial leads and present in up to 45% of Caucasian athletes and 63–91% of black athletes of African-Caribbean descent (hereto referred to as ‘black/African’ athletes).\(^{11-13}\) Black/African athletes also commonly demonstrate a repolarisation variant consisting of convex ST segment elevation in the anterior leads (V1–V4) followed by T wave inversion. On the basis of current data, T wave inversions preceded by ST segment elevation are present in leads V1–V4 in up to 13% of black/African athletes and do not require further assessment in the absence of symptoms, positive family history or abnormal physical examination.\(^{12,13}\)

A junctional (nodal) rhythm or wandering atrial pacemaker may be observed in up to 8% of all athletes under resting conditions.\(^{11}\) First-degree AV block (4.5–7.5%) and less commonly Mobitz type I second-degree AV block are also seen in athletes and a result of increased vagal tone.\(^{6,11,14}\)

Increased cardiac chamber size

Voltage criterion for LVH is present in approximately 45% of male athletes and 10% female athletes.\(^{6,11-15}\) Increased QRS voltage is more common in black/African athletes.\(^{13}\) Although there are several voltage criteria to define LVH, the Sokolow-Lyon criterion is used most commonly. The Sokolow-Lyon voltage criterion for LVH is 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). The isolated presence of high QRS voltages fulfilling the Sokolow-Lyon voltage criterion for LVH is regarded as a normal finding in athletes related to physiological increases in cardiac chamber size and/or wall thickness and does not in itself require additional evaluation (figure 1). However, the additional presence of non-voltage criteria for LVH such as left atrial enlargement, left axis deviation, ST segment depression, T wave inversion or pathological Q waves should raise the possibility of pathological LVH and should prompt further evaluation.

IRBBB (commonly characterised as an rSR’ pattern in V1 with QRS duration <120 ms) is commonly present in athletes (12–32%) and thought to reflect an increase in right ventricular (RV) size secondary to regular training.\(^{6,11-14}\)

**Box 1 Normal ECG findings in athletes**

1. Sinus bradycardia (≥30 bpm)
2. Sinus arrhythmia
3. Ectopic atrial rhythm
4. Junctional escape rhythm
5. First-degree AV block (PR interval>200 ms)
6. Mobitz type I (Wenckebach) second-degree AV block
7. Incomplete RBBB
8. Isolated QRS voltage criteria for LVH
   - Except:QRS voltage criteria for LVH occurring with any non-voltage criteria for LVH such as left atrial enlargement, left axis deviation, ST segment depression, T wave inversion or pathological Q waves
9. Early repolarisation (ST elevation, J-point elevation, J waves, or terminal QRS slurring)

These common training-related ECG alterations are physiological adaptations to regular exercise, considered normal variants in athletes, and do not require further evaluation in asymptomatic athletes.

AV, atrioventricular; bpm, beats per minute; LVH, left ventricular hypertrophy; RBBB, right bundle branch block.

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**Figure 1** ECG of a 29-year-old asymptomatic soccer player demonstrating sinus bradycardia, early repolarisation with ST elevation (arrows) and peaked T waves, and voltage criteria for left ventricular hypertrophy. These are common findings related to regular training. This figure is only reproduced in colour in the online version.

NORMAL ECG FINDINGS IN ATHLETES

Sinus bradycardia
The normal heartbeat is initiated by the sinus node which is located high in the right atrium near the junction of the superior vena cava and the right atrial appendage. To be classified as sinus rhythm, three criteria must be met: (1) there must be a P wave before every QRS complex, (2) there must be a QRS complex after every P wave and (3) the P wave must have a normal axis in the frontal plane (0°–90°). Assuming an intact sinus node, the heart rate is set by the balance between the sympathetic and parasympathetic nervous systems. In healthy adults, sinus rhythm < 60 beats/min is considered as ‘sinus bradycardia’ (figure 2). In well-trained athletes, resting sinus bradycardia is a common finding due to increased vagal tone. In endurance athletes, aerobic training also may induce intrinsic adaptations in the sinus node with decreased automaticity resulting in a high prevalence of sinus bradycardia. In the absence of symptoms such as fatigue, dizziness or syncope, a heart rate ≥ 30 beats/min should be considered normal in a well-trained athlete. Sinus bradycardia disappears with an increase in heart rate during physical activity.

Sinus arrhythmia
The heart rate usually increases slightly during inspiration and decreases slightly during expiration (figure 3). This response called sinus arrhythmia can be quite exaggerated in children and in well-trained athletes resulting in an irregular heart rhythm which originates from the sinus node. It has been estimated that up to 55% of well-trained athletes have sinus arrhythmia. This should not be confused with sinus node dysfunction (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 and an inappropriate rate response to exercise (both a slowed acceleration and an inappropriately rapid deceleration), as well as any association with clinical symptoms such as exercise intolerance, presyncope and syncope. While the heart rhythm is quite irregular in sinus arrhythmia, the P wave axis remains normal in the frontal plane. Accelerating the heart rate with physical activity normalises the heart rhythm. Sinus arrhythmia is considered as a normal finding in an athlete.

Junctional escape rhythm
A junctional or nodal rhythm occurs when the QRS rate is faster than the resting P wave or sinus rate which is slowed in athletes due to increased vagal tone (figure 4). The QRS rate for junctional rhythms is typically less than 100 beats/min, and the QRS complex usually narrow unless the baseline QRS has a bundle branch block. Sinus rhythm resumes with increased heart rates during exercise.

Ectopic atrial rhythm
In an ectopic atrial rhythm, P waves are present but are a different morphology compared to the sinus P wave. Ectopic P waves are most easily seen when the P waves are negative in the inferior leads (II, III and aVF; figure 5). The atrial rate is typically less than 100 beats/min. There also may be more than two different P wave morphologies known as a wandering atrial pacemaker. Ectopic atrial rhythms occur due to a slowed resting sinus rate from increased vagal tone in athletes, and sinus...

Figure 2  ECG demonstrates sinus bradycardia with a heart rate of 40 bpm. The three required features of sinus bradycardia include: (1) P wave before every QRS complex, (2) QRS after every P wave and (3) normal P wave axis (frontal plane 0°–90°). This figure is only reproduced in colour in the online version.
rhythm replaces the ectopic atrial rhythm when the heart rate is increased during exercise.

First-degree AV block
In first-degree AV block, the PR interval is prolonged (>200 ms) but is the same duration on every beat (figure 6). This represents a delay in AV nodal conduction in athletes, due to increased vagal activity or intrinsic AV node changes, and typically resolves with faster heart rates during 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 7). 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 with exercise there should be a return of 1:1 conduction.

Incomplete right bundle branch block
IRBBB is defined by a QRS duration <120 ms with an RBBB pattern: terminal R wave in lead V1 (rsR') and wide terminal S wave in leads I and V6 (figure 8). IRBBB is seen in less than 10% of the general population but is observed in up to 40% of highly trained athletes, particularly those engaged in endurance training and mixed sport disciplines that include both aerobic and anaerobic components. It has been suggested that the mildly delayed RV conduction is caused by RV remodelling, with increased cavity size and resultant increased conduction time, rather than an intrinsic delay within the His-Purkinje system itself.

The occurrence of IRBBB in an asymptomatic athlete with a negative family history and physical examination does not require further evaluation. During the physical examination,
particular care should be devoted to the auscultation of a fixed splitting of the second heart sound because IRBBB can be an associated ECG finding in patients with an atrial septal defect.

IRBBB may be seen in patients with arrhythmogenic RV cardiomyopathy (ARVC). However, in ARVC, the IRBBB pattern is usually associated with other ECG abnormalities, such as T wave inversion involving the mid-precordial leads beyond V2, low limb-lead voltages, prolonged S wave upstroke and/or premature ventricular beats with a left bundle branch block (LBBB) morphology (figure 9).

In some cases, IRBBB may be confused with a Brugada-ECG pattern, which is characterised by a high take-off and downsloping ST segment elevation followed by a negative T wave in ≥2 leads in V1–V3. Unlike the R’ wave in IRBBB, the ‘J wave’

Figure 7  ECG shows Mobitz type I (Wenckebach) second-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. This figure is only reproduced in colour in the online version.

Figure 8  ECG demonstrates incomplete right bundle branch block (IRBBB) with rSR’ pattern in V1 and QRS duration of <120 ms. IRBBB is a common and normal finding in athletes and does not require additional evaluation. This figure is only reproduced in colour in the online version.
seen in a Brugada-ECG pattern does not indicate delayed RV activation, but reflects early repolarisation with J point elevation and a high take-off with downsloping ST segment followed by a negative T wave (figure 10).

**Early repolarisation**

Early repolarisation is an ECG pattern consisting of ST elevation and/or a J wave (distinct notch) or slur on the downslope of the R wave (figure 11). Traditional examples of early repolarisation referred to ST elevation, but newer definitions also include J waves or terminal QRS slurring (figure 12).

Early repolarisation is a common finding in trained athletes and considered a benign ECG pattern in apparently healthy, asymptomatic individuals. Depending on how it is defined, early repolarisation is reported in up to 35–91% of trained athletes and is more prevalent in young males and black/
Early repolarisation is most common in the precordial leads but can be present in any lead. Commercially available computer diagnostic ECG programmes commonly misreport early repolarisation patterns in athletes as acute ischaemia/myocardial infarction or pericarditis.

The early repolarisation pattern in athletes typically involves a concave and ascending/upward ST segment elevation. Late QRS slurring or notching with horizontal ST segment elevation in the inferolateral leads has been associated with an increased risk of arrhythmic death in one study of middle-aged, non-athletic Finnish citizens. However, a significant percentage of young competitive athletes (25–30%) show early repolarisation with similar morphological features in either the inferior or lateral leads. These findings are more common in athletes at times of peak fitness, suggesting early repolarisation is a dynamic process and is at least in part a direct result of exercise training. To date, no data support the presence of an association between early repolarisation and SCD in athletes.

![Figure 11](image1.png)

**Figure 11** ECG from a 29-year-old asymptomatic soccer player demonstrating early repolarisation (J-point and ST elevation) in I, II, aVF, V2–V6 (arrows) and tall, peaked T waves (circles). These are common, training-related findings in athletes and do not require more evaluation. This figure is only reproduced in colour in the online version.

![Figure 12](image2.png)

**Figure 12** (A and B) Classic definition of early repolarisation based on ST elevation at QRS end (J-point). Examples without (A) and with (B) a J wave. (C and D) New definitions of early repolarisation showing slurred QRS downstroke (C) and J-wave (D) without ST elevation.
Although further investigation is warranted to fully characterise the prognostic implications of early repolarisation in competitive athletes, all patterns of early repolarisation, including inferolateral subtypes, should be considered normal variants in athletes.24

QRS voltage criteria for LVH

The most commonly used voltage criterion for LVH is the Sokolow-Lyon index. However, ECG QRS voltage may not be a reliable predictor of LVH. The limitation of the ECG in identifying ventricular hypertrophy is due to the reliance of

Figure 13  ECG from a 19-year-old asymptomatic soccer player demonstrating voltage criteria for left ventricular hypertrophy (S-V1+R-V5>35 mm). Note the absence of left atrial enlargement, left axis deviation, ST depression, T wave inversion, or pathological Q waves. Increased QRS amplitude without other ECG abnormalities is a common finding in trained athletes and does not require additional testing. This figure is only reproduced in colour in the online version.

Figure 14  Abnormal ECG from a patient with hypertrophic cardiomyopathy. In addition to voltage criteria for left ventricular hypertrophy, note the deep T wave inversions extending to the lateral leads (I and aVL, V5–V6). These findings are abnormal, not related to regular training and require additional evaluation. This figure is only reproduced in colour in the online version.
measuring the electrical activity of the heart by electrodes on the surface of the body. Consequently, anything between the left ventricular myocardium and the surface electrodes will affect the voltage. ECG QRS voltage, therefore, can be influenced by a variety of factors other than LV size or mass. Males, athletes and black/African individuals have higher QRS voltage, while obesity, older age and pulmonary disease may cause lower voltage.\textsuperscript{31} The goal of identifying clinically relevant LVH by voltage criteria alone is particularly problematic in children. The standards for QRS voltage have been derived from studies of populations of clinically normal children. Furthermore, the limited studies do not consistently include referencing to body size, gender or ethnicity. Lastly, correlation with echocardiography is limited, and reference standards from autopsy or MRI are not available.\textsuperscript{31}

Figure 15  ECG from a 24-year-old asymptomatic black/African soccer player demonstrating ‘domed’ ST elevation followed by T wave inversion in leads V1–V4 (circles). This is a normal repolarisation pattern in black/African athletes. This figure is only reproduced in colour in the online version.

Figure 16  (A) Normal variant repolarisation changes in a black/African athlete characterised by domed ST segment elevation and T wave inversion in V1–V4. (B) Pathological T wave inversion and ST depression in the lateral leads. T wave inversion in V5–V6 is always an abnormal finding and requires additional testing to rule out cardiomyopathy. This figure is only reproduced in colour in the online version.
In athletes, intensive conditioning is also associated with morphological cardiac changes of increased cavity dimensions and wall thickness that are reflected on the ECG. These changes constitute physiological LVH in trained athletes and usually manifests as an isolated increase in QRS amplitude (figure 13). ECGs with increased QRS amplitudes meeting ECG voltage criteria for LVH are prevalent and present in up to 45% of athletes and 25% of sedentary young adults. As a result, the accuracy of increased QRS voltage as an indicator of pathological LVH is poor.

Increased QRS voltage and HCM
Several studies have evaluated athletes and young adults with isolated increased QRS voltage using echocardiography or cardiac MRI and none had hypertrophic cardiomyopathy.

Figure 17  (A) Normal variant repolarisation changes in a black/African athlete characterised by domed ST segment elevation and T wave inversion in V1–V4. (B) Pathological T wave inversion in V1–V3. Note the isoelectric ST segment. The absence of ST segment elevation prior to T wave inversion makes this ECG abnormal. Additional testing is required to rule out arrhythmogenic right ventricular cardiomyopathy. This figure is only reproduced in colour in the online version.

Figure 18  (A) Normal variant repolarisation changes in a black/African athlete characterised by domed ST segment elevation and T wave inversion in V1–V4. (B) A downsloping ST segment elevation followed by T wave inversion in V1–V2 suggestive of a Brugada-pattern ECG. Note the high-take off and absence of upward convexity ('dome' shape) of the ST segment distinguishing this from the repolarisation variant in black/African athletes. This figure is only reproduced in colour in the online version.
(HCM). Furthermore, increased QRS voltage in the absence of other ECG abnormalities is uncommon in subjects with HCM being present in less than 2% of individuals with the disease. However, when other ECG abnormalities such as ST depression, T wave inversion, pathological Q waves, left axis deviation or left atrial abnormalities are present, the possibility of HCM should be investigated by additional testing (figure 14). Therefore, isolated increased QRS voltage on the ECG in the absence of other abnormalities in an asymptomatic athlete with a negative family history is not a reliable indicator of LVH or HCM and does not require further evaluation.

Repolarisation findings in black/African athletes

Growing attention has been paid to ethnic-related differences in morphological and ECG features of the athlete’s heart. Notably, there are specific repolarisation patterns in black/African athletes that are normal variants and should be distinguished from abnormal findings suggestive of a pathological cardiac disorder.

As aforementioned, early repolarisation is common in athletes and usually characterised by an elevated ST segment with upward concavity, ending in a positive (upright ‘peaked’) T wave (figure 11). There is also a normal variant early repolarisation pattern found in some black/African athletes, characterised by an elevated ST segment with upward convexity (‘dome’ shaped), followed by a negative T wave confined to leads V1–V4 (figure 15). The presence of either repolarisation pattern in an asymptomatic black/African athlete does not require additional testing.

Differentiating normal repolarisation variants from pathological findings

The presence of early repolarisation and T wave inversion in the anterior leads in black/African athletes probably represents a specific, ethnically dependent adaption to regular exercise. More than two-thirds of black athletes exhibit ST segment elevation and up to 25% show T wave inversions. However, normal repolarisation changes in black/African athletes do not extend beyond V4. Thus, T wave inversion in the lateral leads (V5–V6) is always considered as an abnormal finding and requires additional testing to rule out HCM or other cardiomyopathies (figure 16).

Repolarisation variants in black/African athletes also must be distinguished from pathological repolarisation changes in the anterior precordial leads found in ARVC and Brugada-pattern ECGs. In ARVC, the ST segment is usually isoelectric prior to T wave inversion, in contrast to the ‘domed’ ST segment elevation which is the hallmark feature of the normal repolarisation variant in black/African athletes (figure 17). In Brugada-pattern ECGs, the high take-off and downsloping ST segment prior to T wave inversion distinguishes this from the ‘domed’ ST segment elevation preceding the negative T wave in black/African athletes (figure 18). Pathological repolarisation changes in the anterior precordial leads suggesting either ARVC or Brugada-pattern require additional testing.

CONCLUSIONS

Fundamental to the cardiovascular care of athletes is an understanding of the physiological cardiac adaptations in athletes that are manifested on the ECG. The ECG can provide valuable information when interpreted properly, accounting for the electrical and structural changes that are a common result of regular training. Distinguishing ECG findings related to athlete’s heart from changes suggestive of an underlying pathological disorder is critical to the identification of athletes at risk of SCD.


Normal electrocardiographic findings: recognising physiological adaptations in athletes

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Abnormal electrocardiographic findings in athletes: recognising changes suggestive of cardiomyopathy

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ABSTRACT
Cardiomyopathies are a heterogeneous group of heart muscle diseases and collectively are the leading cause of sudden cardiac death (SCD) in young athletes. The 12-lead ECG is utilised as both a screening and diagnostic tool for detecting conditions associated with SCD. Fundamental to the appropriate evaluation of athletes undergoing ECG is an understanding of the ECG findings that may indicate the presence of an underlying pathological cardiac disorder. This article describes ECG findings present in cardiomyopathies afflicting young athletes and outlines appropriate steps for further evaluation of these ECG abnormalities. The ECG findings defined as abnormal in athletes were established by an international consensus panel of experts in sports cardiology and sports medicine.

INTRODUCTION
The cardiomyopathies are a diverse group of heart muscle diseases that are defined and subdivided in clinical practice by different structural and functional characteristics. As a family of related diseases, the cardiomyopathies are the leading cause of sudden cardiac death (SCD) in young competitive athletes.1–3 Athletes with an underlying cardiomyopathy may present with disease-related symptoms or may be asymptomatic and thus only identified by abnormal testing during pre-participation screening. Although a definitive diagnosis may require extensive evaluation by a cardiovascular specialist, the 12-lead ECG is commonly abnormal among athletes with an underlying cardiomyopathy. Therefore, it is of paramount importance that clinicians responsible for ECG interpretation in athletes be familiar with key findings associated with underlying diseases of the heart muscle. This paper will review the principal ECG findings associated with the most common forms of cardiomyopathy relevant to the care of the young athlete. Initial testing for further evaluation of abnormal ECG findings is also presented.

DISTINGUISHING NORMAL FROM ABNORMAL
A challenge in the use of ECG for screening or diagnostic evaluations in athletes is the ability to accurately differentiate findings suggestive of a potentially lethal cardiovascular disorder from benign physiological adaptations occurring as the result of regular and sustained intensive training (ie, athlete’s heart). Several reports have outlined ECG criteria intended to distinguish normal ECG findings in athletes from ECG abnormalities requiring additional evaluation.4–9

On 13–14 February 2012, an international group of experts in sports cardiology and sports medicine convened in Seattle, Washington, to define contemporary standards for ECG interpretation in athletes. The objective of the meeting was to help physicians distinguish normal ECG alterations in athletes from abnormal ECG findings that require additional evaluation for conditions that predispose to SCD.10 A review of normal ECG findings in athletes is presented separately.11

In this paper, abnormal ECG findings are presented relative to the most common cardiomyopathies associated with SCD in athletes: hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), dilated cardiomyopathy (DCM) and left-ventricular non-compaction (LVNC). Table 1 summarises a list of abnormal ECG findings unrelated to athletic training that may suggest the presence of an underlying cardiomyopathy and should trigger additional evaluation in an athlete.

HYPERTROPHIC CARDIOMYOPATHY
HCM is a genetic disease of the heart muscle. It is characterised by ventricular hypertrophy in the absence of a recognisable cause such as aortic valve disease or hypertension. A common pattern of hypertrophy in HCM is an asymmetric septal hypertrophy where the interventricular septum is thicker than the rest of the left ventricle. However, many other patterns of pathological hypertrophy are consistent with HCM such as apical...
hypertrophy, concentric hypertrophy and proximal septal hypertrophy. Poor ventricular compliance (diastolic dysfunction) is characteristic, along with microvascular dysfunction which contribute to ischaemia during exercise. Some patients have dynamic left ventricular (LV) outflow tract obstruction caused by the combination of hypertrophy and abnormalities of the mitral valve which leads to systolic anterior motion of the anterior leaflet. However, only about 25% of patients with HCM have a murmur from LV outflow tract obstruction during resting examination. Symptoms of HCM include chest pain, syncope and exercise intolerance, but for many persons the disease can be asymptomatic and SCD may be the clinical presentation of the disease. TWI in the lateral or inferior leads is reported in 2% of black/African athletes. TWI beyond V2 is a rare abnormality found in 10% of black/African athletes. TWI in the lateral or inferolateral leads is seen commonly in black patients with HCM, TWI occurs more commonly in the lateral leads (77%) and less frequently in the inferolateral leads (2%). Abnormal TWI is defined as >1 mm in depth in two or more leads V2–V6, II and aVF, or I and aVL (excludes leads III, aVR and V1). Deep TWI in the mid-precordial to lateral precordial leads (V4–V6) should raise the possibility of apical HCM. In healthy athletes, TWI in the lateral or inferior leads is uncommon. TWI beyond V2 is a rare abnormality found in only 0.1% of Caucasian adolescent athletes older than 16 years. In a college athletic population of mixed ethnicity, TWI in the lateral or inferolateral leads is reported in 2% of athletes. In Caucasian elite athletes, the prevalence of TWI in the lateral or inferior leads is also about 2%. However, TWI is more common in black athletes of African-Caribbean descent (hereto referred to as ‘black/African’ athletes). TWI in the lateral or inferior leads is reported in 8–10% of black/African athletes.

**Prevalence**

HCM is among the most common inherited cardiovascular disorders and may occur in 1:500 adults and at equal prevalence in men and women. However, the reported prevalence of HCM in competitive athletes is apparently lower, approximately 1 in 1000 to 1 in 1500 athletes. HCM is inherited primarily as autosomal dominant with variable penetrance, and morphological expression of HCM may appear in childhood but typically develops in early adolescence through young adulthood. This may contribute to the lower prevalence of HCM found in younger athletes.

**Contribution as a cause of SCD**

In most case series, HCM is among the most common causes of SCD in young athletes. In the USA, HCM accounts for approximately one-third of identified causes of SCD in athletes, and in the UK HCM represents 11% of cases. In a large common cause of sudden death in other populations. In US military personnel, HCM accounted for only 6% of SCD, and in the US general population (less than 35 years old) only 5% of cases of sudden cardiac arrest were attributed to HCM.

**Diagnostic criteria**

HCM can be diagnosed by ECG in combination with echocardiography or cardiac MRI. An LV wall thickness of 1.5 cm or greater is normally required to make the diagnosis, but marked asymmetry with lower absolute wall thickness measurement is also compatible with HCM. The upper limit of normal wall thickness in most echocardiography laboratories is 1.2 cm. A ‘grey area’ is defined between 1.2 and 1.5 cm. In borderline cases, other features favouring a diagnosis of HCM include impaired diastolic function, small LV cavity size, LV wall thickness asymmetry, mitral valve pathology (leak or redundancy and systolic anterior motion) and the presence of myocardial fibrosis (late gadolinium enhancement) on cardiac MRI.

**Abnormal ECG findings in HCM**

Over 90% of patients with HCM will have an abnormal ECG. ECG abnormalities include T wave inversion (TWI), ST segment depression, pathological Q waves, conduction delay, left-axis deviation (LAD) and left atrial enlargement (LAE).

| T wave inversion | >1 mm in depth in two or more leads V2–V6, II and aVF or I and aVL (excludes III, aVR and V1) |
| ST segment depression | ≥0.5 mm in depth in two or more leads |
| Pathological Q waves | ≥3 mm in depth or >40 ms in duration in two or more leads (except III and aVF) |
| Complete left bundle branch block | QRS>120 ms, predominantly negative QRS complex in lead V1 (Q or R), and upright monophasic R wave in leads I and V6 |
| Intraventricular conduction delay | Any QRS duration ≥140 ms |
| Left axis deviation | −30° to −90° |
| Left atrial enlargement | Prolonged P wave duration of >120 ms in leads I or II with negative portion of the P wave≥1 mm in depth and ≥40 ms in duration in lead V1 |
| Right ventricular hypertrophy pattern | Rv1+Sv5>10.5 mm AND right axis deviation >120° |
| Premature ventricular contractions | ≥2 PVCs per 10 s tracing |
| Ventricular arrhythmias | Couplets, triplets and non-sustained ventricular tachycardia |

Note: These ECG findings are unrelated to regular training or expected physiological adaptation to exercise, may suggest the presence of pathological cardiovascular disease, and require further diagnostic evaluation.

**Repolarisation variant in black/African athletes**

TWI in the anterior precordial leads should be distinguished from TWI in the lateral or inferior leads in black/African athletes. TWI in the anterior precordial leads may be part of a normal variant pattern of repolarisation in black/African athletes consisting of convex (‘dome’ shaped) ST segment elevation followed by TWI in V1–V4 (figure 4). On the basis of current data, TWI preceded by ST segment elevation are present in the anterior precordial leads in up to 13% of black/African athletes and do not require further assessment in the absence of symptoms, positive family history or abnormal physical examination. However, TWI in the lateral or inferolateral leads (V5–V6, I and aVL, II and aVF), regardless of ethnicity, is considered abnormal and requires additional testing to rule out HCM (figures 1–3).
Juvenile pattern of TWI
TWI in the anterior precordial leads in younger, prepubertal athletes often reflects a persistent juvenile pattern and requires careful interpretation. In Caucasian adolescent athletes, anterior precordial TWI extending beyond V2 was present in 1.2% of athletes <16 years but only 0.1% of athletes ≥16 years.24 In a study of Italian adolescent athletes, incomplete pubertal development was an independent predictor for right precordial TWI.28 The prevalence of right precordial TWI decreased significantly with increasing age, 8.4% in children <14 years of age versus 1.7% in those ≥14 years.28

Biphasic T waves
Biphasic T waves create a challenge and currently there is no consensus regarding the definition of TWI when a large positive deflection precedes a negative portion below the isoelectric line. If...
the negative portion of the T wave is >1 mm in depth in two or more leads (excluding leads III, aVR, and V1), it is reasonable to consider this pattern as abnormal until more data are obtained.

**ST segment depression**

ST segment depression is a common abnormality in HCM but extremely rare in otherwise healthy athletes, making it a concerning indicator of disease if identified on an athlete’s ECG. ST segment depression is reported in 46–50% of patients with HCM, but in <1% of apparently healthy athletes or adolescents undergoing ECG screening. Any degree of ST depression beyond 0.5 mm in two or more leads is significant and requires further investigation for cardiomyopathy (figures 1 and 2).

**Pathological Q waves**

Q waves have been defined in different ways in different populations. In patients with overt HCM, pathological Q waves are
In one series of asymptomatic patients with HCM, 42% demonstrated pathological Q waves. The consensus of this group is to define Q waves for HCM as >3 mm in depth or >40 ms in duration in at least two leads (excluding leads III and aVR; figure 5). This detects HCM with a sensitivity of 35% and a specificity of 95% in patients with preclinical HCM based on molecular genetic diagnosis.

Intraventricular conduction delay

Left bundle branch block (LBBB) is an abnormal finding detected in 2% of patients with HCM but not reported in screening populations of athletes or adolescents. LBBB pattern with a QRS duration of 120 ms or greater should prompt further evaluation (figure 6). Right bundle branch block (RBBB) is found more commonly in HCM than in athletes but the frequency of incomplete and complete RBBB in athletes is felt to limit its differentiating value. The significance of a non-specific intraventricular conduction delay (IVCD) with normal QRS morphology is uncertain. However, marked non-specific IVCD >140 ms is considered abnormal and should prompt further evaluation.

Left axis deviation

LAD, defined as –30° to –90°, is present in almost 12% of HCM patients but less than 1% of athletes (figure 7). LAD can be a secondary marker for pathological LV hypertrophy (LVH) and if present warrants additional evaluation.

Left atrial enlargement

ECG findings suggestive of LAE have been defined in different ways. Overall, LAE on ECG is present in approximately 10–21% of HCM patients but has been reported in up to 44% of black patients with HCM (figure 8). LAE is defined as a prolonged P wave duration of >120 ms in leads I or II with negative portion of the P wave ≥1 mm in depth and ≥40 ms in duration in lead V1. LAE on ECG is an uncommon finding in athletes and should prompt additional investigation.

Increased QRS voltage in athletes and HCM

The isolated presence of high QRS voltages fulfilling voltage criterion for LVH is regarded as a normal finding in athletes related to physiological increases in cardiac chamber size and/or wall thickness and does not in itself require additional evaluation. As expected, voltage criteria for LVH also are commonly identified in individuals with HCM. However, the presence of isolated increased QRS voltage in the absence of other ECG abnormalities is uncommon and present in <2% of individuals with the disease. Several studies have evaluated athletes and young adults with isolated increased QRS voltage using echocardiography or cardiac MRI and none had HCM. Therefore, isolated increased QRS voltage on the ECG in the absence of other abnormalities in an asymptomatic athlete with a negative family history is not a reliable indicator of HCM and does not require further evaluation. Co-existing ECG abnormalities such as TWI, ST segment depression, pathological Q waves, IVCD, LAD or LAE should be investigated by additional testing.

Evaluation of suspected HCM

If HCM is suspected based on ECG abnormalities, evaluation of LV morphology and function is required. Echocardiography provides assessment of LV cavity size and wall thickness, systolic and diastolic function and valvular structure and function, and is the first test of choice under most circumstances. HCM can be diagnosed when wall thickness is ≥1.5 cm with normal or small LV cavity size in the absence of other causes capable of causing myocardial hypertrophy. Diastolic dysfunction, mitral valve pathology and LV outflow tract obstruction are other findings that support the diagnosis of HCM.

However, echocardiographic quality is variable based on numerous factors, including operator proficiency and patient acoustic windows, and may have limited ability to detect hypertrophy of the anterolateral LV wall and apex (figure 9). Cardiac MRI provides superior assessment of myocardial hypertrophy and may demonstrate late gadolinium enhancement which is a non-specific marker suggesting myocardial fibrosis. Cardiac MRI should be considered when echocardiography is insufficient to assess all myocardial segments or when

Figure 5 Abnormal ECG in a patient with hypertrophic cardiomyopathy. Note the abnormal Q waves (>3 mm in depth) in V5–V6, II and aVF. This figure is only reproduced in colour in the online version.

myocardial hypertrophy falls into the ‘grey zone’ between 1.2 and 1.5 cm. Cardiac MRI is recommended for markedly abnormal ECGs suggestive of apical HCM, specifically ECGs with deep TWI and/or ST depression in the inferolateral leads (V4–V6, I, aVL, II and aVF), in which echocardiography often does not provide an adequate assessment of the LV apex or inferior septum (figures 1–3).

A common clinical dilemma is the detection of myocardial hypertrophy in an athlete in which the hypertrophy may be due to physiological adaptation to exercise. Differentiating athlete’s heart from HCM requires careful clinical evaluation by an experienced provider. Cardiac MRI, cardiopulmonary exercise testing and Holter monitoring should be considered. Findings suggestive of HCM include the presence of unusual patterns of hypertrophy with substantial differences in wall thickness of the LV segments, normal or reduced LV cavity size, extreme LAE, diastolic dysfunction, family history of HCM or SCD, below normal peak oxygen consumption (peak VO$_2$), and the presence of ventricular arrhythmias. When diagnostic uncertainty remains, genetic testing and/or a period of deconditioning followed by reassessment to document regression (or lack thereof) of exercise-induced LVH may be considered.

Long-term follow-up of markedly abnormal ECGs

Highly trained athletes occasionally present markedly abnormal ECG patterns instinctively suggesting the presence of a cardiomyopathy. Such abnormal ECGs raise the question of differentiating between the initial, subtle expression of cardiac disease or the extreme but innocent ECG expression of the ‘athlete’s heart’.

Investigators researched the clinical outcomes of 81 athletes presenting initially with markedly abnormal repolarization patterns in the absence of detectable cardiac abnormalities. After an average 9-year follow-up, a new diagnosis of
cardiomyopathy was made in five athletes (6%), including three with HCM, one with ARVC and one with DCM. Two athletes experienced adverse events (0.3% per year), including one cardiac arrest from HCM and one sudden death related to ARVC. Markedly abnormal ECGs, therefore, may represent the initial expression of cardiomyopathy, preceding by many years the phenotypic or morphological expression of structural heart disease.

Athletes presenting with distinctly abnormal ECGs (ie, deep TWI in the lateral leads) and no evidence of structural heart disease after a thorough work-up may be permitted to participate in competitive athletics. However, these athletes should undergo serial clinical evaluation on an annual basis, even in the absence of symptoms, including repeat imaging tests such as echocardiography and/or cardiac MRI to evaluate for the development of cardiomyopathy.

**Figure 8** Abnormal ECG in a patient with hypertrophic cardiomyopathy showing left atrial enlargement, defined as a prolonged P wave duration >120 ms in leads I or II with negative portion of the P wave ≥1 mm in depth and ≥40 ms duration in lead V1. This figure is only reproduced in colour in the online version.

**Figure 9** ECG (left panel) and cardiac MRI (right panel) of apical hypertrophic cardiomyopathy. Deep T wave inversions across the precordial leads are a characteristic ECG finding. The region of hypertrophy (red arrow) is isolated to the left ventricular (LV) apex, which can be challenging to detect by echocardiography. This figure is only reproduced in colour in the online version.
ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

ARVC is an inherited heart muscle disease characterised by fibro-fatty replacement of right ventricular myocardium and corollary life-threatening ventricular arrhythmias or SCD, mostly in young people and athletes. Progressive dilation/dysfunction predominantly involves the right ventricle with involvement of the left ventricle in late-stage disease. Variants with predominantly LV involvement are described in about 10% of patients (hence the alternative term of arrhythmogenic cardiomyopathy). Mutations in the desmosomal genes account for approximately 50% of ARVC cases. In addition, there is emerging evidence that intense endurance sports may lead to a similar phenotype (with similar prognosis) in the absence of desmosomal mutations, so-called exercise-induced ARVC, which may be the result of increased RV wall stress during exercise.

The prevalence of familial ARVC is estimated at 1:2000–1:5000 persons.

Contribution as a cause of SCD

According to data from the Veneto region of Italy where post-mortem investigation of young sudden death victims is performed systematically, ARVC is a leading cause of sport-related sudden death accounting for approximately one-fourth of fatalities in young competitive athletes. Data from the USA, notably without a mandatory registry for SCD in athletes, suggest that ARVC is a less common cause of SCD.

Diagnostic criteria

The original (1994) and the revised (2010) Task Force Criteria for diagnosis of ARVC are based on major and minor criteria encompassing familial/genetic, ECG, arrhythmic, morphofunctional ventricular and histopathological features. The diagnosis is fulfilled in the presence of two major criteria, one major plus two minor criteria, or four minor criteria from different groups.

Abnormal ECG findings in ARVC

Over 80% of patients with ARVC will have an abnormal ECG. ECG abnormalities include TWI in the anterior precordial leads, epsilon waves, delayed S wave upstroke, low-voltage in limb leads and premature ventricular beats with an LBBB morphology and superior axis. If there is primarily LV involvement, the TWI involves the lateral precordial leads and the premature ventricular beats can have an RBBB morphology.

T wave inversion

TWI in the anterior precordial leads (V1–V3/V4) is present in approximately 85% of patients with ARVC in the absence of RBBB. TWI occasionally extends to the left precordial leads V5–V6 or inferior limb leads II, III and aVF. TWI in V1–V3 or beyond in individuals >14 years of age (in the absence of complete RBBB) represent a major diagnostic criterion for ARVC, while TWI confined to just leads V1 and V2 in individuals >14 years of age (in the absence of complete RBBB) represents a minor diagnostic criterion. In Italian children ≥14 years with TWI in the anterior precordial leads beyond V2 (ie, V3 or V4), 3 of 26 (11%) fulfilled diagnostic criteria for ARVC (1 definite, 2 borderline).

Epsilon waves

Epsilon waves are defined as distinct low-amplitude potentials localised at the end of the QRS complex. Epsilon waves are challenging to detect and appear as a small negative deflection just beyond the QRS in V1–V3. The presence of epsilon waves in the right precordial leads V1–V3 represents a major diagnostic criterion for ARVC.

Delayed S wave upstroke

Delayed S wave upstroke of >55 ms in leads V1–V3 in the absence of complete RBBB represents a minor diagnostic criterion for ARVC. This feature is most commonly observed among ARVC patients with mild QRS prolongation (100–120 ms). The S wave upstroke is measured from the nadir...
of the S wave to the end of the QRS (including epsilon wave if present). Prolonged S wave upstroke may be present in up to 95% of patients with ARVC in the absence of RBBB.50

Low voltage in limb leads
Low voltage in limb leads, defined as a QRS amplitude ≤5 mm in each of the limb leads (I, II and III), also can be suggestive of ARVC (figure 11).

Premature ventricular contractions
Premature ventricular contractions (PVCs) originating from the right ventricle typically show an LBBB pattern with a predominantly negative QRS complex in V1. On the basis of the QRS axis in the limb leads the origin of the PVC can be further suggested. PVCs with LBBB morphology and an inferior axis (positive in the inferior leads) originate from the right ventricular outflow tract consistent with idiopathic right ventricular outflow tract arrhythmia which is a benign condition, non-familial and not associated with structural ventricular abnormalities. PVCs with an LBBB morphology and superior axis (negative in the inferior leads) originate from the right ventricular free wall or apex and are more suggestive of ARVC (figure 12).

Evaluation of suspected ARVC
Disease expression in ARVC is variable, and clinical manifestations vary with age and stage of disease.49 Similarly, the extent of ECG abnormalities are associated with the severity of disease.51 In patients with diagnosed ARVC (‘overt stage’) or known desmosome mutation positive subjects, 95% have an abnormal ECG marked by abnormal TWI, a prolonged S wave upstroke in the anterior precordial leads (V1–V3), and/or an epsilon wave.50–52 However, in cardiac screening, physicians may encounter asymptomatic athletes in the early ‘concealed stage’ of the disease, showing less pronounced ECG changes. 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. A combination of tests is needed to effectively make the diagnosis or to rule out ARVC. Echocardiography, ambulatory ECG (Holter) monitoring, signal-averaged ECG (SAECG), and ventricular angiography provided optimal evaluation, while cardiac MRI (false-positives) and biopsy (low-sensitivity) were considered less useful for diagnosis in suspected ARVC.53 54

The evaluation of major diagnostic ECG findings according to the 2010 Task Force criteria of TWI in the right precordial leads (V1–V3) or beyond in ages >14 years (in the absence of complete RBBB) should be extensive. In addition to a comprehensive symptom history, family history and physical examination, evaluation of TWI in V1–V3 or beyond should include echocardiography, Holter monitoring, SAECG, maximal exercise-ECG test and possibly a cardiac MRI.49 Isolated epsilon waves in the right precordial leads, a less specific major ECG criteria, still require an echocardiography and ECG monitoring for an arrhythmia (Holter or exercise-ECG test).49

Evaluation of minor diagnostic ECG-findings according to the Task Force criteria (without accompanying positive family history or alarming symptoms), may be less extensive.49 TWI in V1–V2 may require simply a careful personal and family history and physical examination.
Repolarisation variants in the anterior precordial leads in black/African athletes must be distinguished from pathological repolarisation changes found in ARVC. In ARVC, the ST segment is usually isoelectric prior to TWI, in contrast to the ‘domed’ ST segment elevation which is the hallmark feature of the normal repolarisation variant in black/African athletes (figure 13). TWI involving at least two consecutive precordial leads from V2 to V6 with an isoelectric ST segment, regardless of ethnicity, requires additional investigation.

**DILATED CARDIOMYOPATHY**

DCM is a heart muscle disorder characterised by weakened myocardial contraction, which over time leads to cavity enlargement and eccentric heart muscle hypertrophy. It is a common

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**Figure 12** ECG from a patient with arrhythmogenic right ventricular cardiomyopathy. Note multiple premature ventricular complexes with a left bundle branch pattern and superior axis (negative QRS vector in inferior leads). This figure is only reproduced in colour in the online version.

**Figure 13** (A) Normal variant repolarisation changes in a black/African athlete characterised by domed ST segment elevation and T wave inversion in V1–V4. (B) Pathological T wave inversion in V1–V3. Note the isoelectric ST segment. The absence of ST segment elevation prior to T wave inversion makes this ECG abnormal. Additional testing is required to rule out arrhythmogenic right ventricular cardiomyopathy. This figure is only reproduced in colour in the online version.
cause of heart failure, the inability of the heart to meet the demands of the body. DCM can be caused by a variety of etiologies ranging from coronary artery disease, infections/myocarditis, toxins (alcohol and other drugs), metabolic or endocrine abnormalities, autoimmune disorders, infiltrative processes and a variety of genetic/inheritable disorders. In many cases, a distinct cause of the compromised heart muscle is never found. DCM can be asymptomatic, or it can result in symptoms of exercise intolerance, shortness of breath, swelling, congestive heart failure or SCD from ventricular arrhythmia.

Prevalence
The exact prevalence of DCM in the general population is not known and estimates depend on the population demographics and the cut-off used to define LV dysfunction. The majority of cases with early stages of LV dysfunction may be asymptomatic. DCM prevalence is increased with older age, male gender and the presence of cardiovascular risk factors. Among a relatively young, healthy population without coronary risk factors, asymptomatic LV dysfunction was present in about 0.2% of individuals. DCM accounts for about 2% of cases in a series of young athletes with sudden cardiac arrest.

Diagnostic criteria
DCM is diagnosed on non-invasive imaging (echocardiography or cardiac MRI) by detecting LV chamber enlargement and decreased LV systolic (contractile) function. Systolic function is usually quantified using either fractional shortening (FS) or ejection fraction (EF), but there is no consensus cut-off definition of DCM based on these parameters. Normal ranges vary by lab and imaging modality.

Abnormal ECG findings in DCM
Several studies have examined ECG abnormalities in patients with asymptomatic DCM, but few have reported findings among non-ischaemic DCM or among individuals with asymptomatic LV dysfunction. Overall, approximately 90% of individuals with DCM have abnormal ECG findings (figures 14 and 15). Patients with prior myocardial infarction may manifest pathological Q waves. Among individuals with non-ischaemic cardiomyopathy, the most common abnormalities seen include voltage criteria for LVH (33–40%), TWIs (25–45%), LAE (15–33%), LAD (15–25%), pathological Q waves (10–25%), LBBB (9–25%), premature ventricular contractions (5–10%) and RBBB (4%). These findings are non-specific for DCM. Goldberger has proposed a triad of findings that may offer more specificity: (1) LVH in the anterior precordial leads; (2) low limb lead voltage and (3) poor precordial R wave progression. Given the overlap of some of these findings with physiological ECG changes found in athlete’s heart, distinctly abnormal ECG criteria unrelated to regular training and requiring additional evaluation to rule out an underlying cardiomyopathy are listed in table 1.

Evaluation of suspected DCM
When DCM is suspected, further evaluation of LV size and function is required. Echocardiography provides assessment of cardiac structure and function, including FS and/or EF, and is the first test of choice under most circumstances. Contrast echocardiography or cardiac MRI should be considered when image resolution is reduced.

Athletes may have LV chamber enlargement as a part of physiological adaptation to exercise. This is most often seen in

Figure 14 ECG from a patient with idiopathic dilated cardiomyopathy. Note the resting sinus tachycardia, left atrial enlargement, T wave inversions in the lateral limb (I and aVL) and precordial (V5–V6) leads and deep S waves in V1–V3 (part of Goldberger’s triad). This figure is only reproduced in colour in the online version.
athletes participating in endurance sports such as cycling, cross-country skiing or rowing. Mild reduction in L V contractility (EF 40–50%) is seen in a minority of athletes with L V cavity dilation, but is not an invariable component of physiological adaptation to exercise. Stress echocardiography can assess myocardial performance at submaximal or peak exercise which may help differentiate those with low normal or borderline systolic function, as systolic function is more likely to normalise in athlete’s heart than in DCM. Thus, L V dilatation and measures of systolic function should be interpreted carefully and in the context of the athlete’s level and amount of endurance training. All patients with DCM should be referred to a cardiologist for further aetiological evaluation, including assessment for myocardial ischaemia and infiltrative disorders.

LV NON-COMPACTION
LVNC is a heart muscle disorder in which loosely organised myocardial fibres fail to condense into a compact layer resulting in increased myocardial trabeculations and thinning of the compact myocardium (figure 16). LVNC can occur along with other congenital or embryological abnormalities, or can be found in isolation, and can be due to underlying gene mutations. However, the majority of LVNC remains genetically elusive. This disturbance of myocardial structure leads to progressive weakening of heart muscle contraction (lower EF) with ventricular dilation. Therefore, LVNC should be distinguished from DCM. Blood clots may also form within the trabecular recesses, increasing the risk for embolic strokes.

Prevalence
The exact prevalence of isolated LVNC is unknown, but is thought to be <0.1–0.2%. Reasons for the uncertainty in prevalence include challenges in imaging the non-compaction and disagreement regarding diagnostic criteria.

Contribution as a cause of SCD
LVNC is associated with an increased risk of abnormal heart rhythms and sudden cardiac arrest. LVNC is a rare (<1%) cause of SCD in a series of young athletes.

Diagnostic criteria
Several sets of diagnostic criteria for isolated LVNC exist, but remain controversial. These criteria are based generally on echocardiography or cardiac MRI findings of an increased ratio of trabeculations to compact myocardium. These criteria have
been called into question recently for being non-specific, particularly among black individuals who have relatively greater degrees of myocardial trabeculations and among athletes.\(^7^4\)

**Abnormal ECG findings in isolated LVNC**

ECG abnormalities in isolated LVNC are common but non-specific (figures 17 and 18). In a series of 78 patients with a clinical diagnosis of LVNC, only 13% had a normal ECG.\(^7^5\) The most common abnormalities in this series included repolarisation changes (72%), QT prolongation (52%), ST segment depression (51%), TWI (41%), LVH voltage criteria (38%), IVCD (31%) including LBBB (19%) and RBBB (3%), and LAE (26%).\(^7^5\) Given the overlap of some of these findings with physiological ECG changes found in athlete’s heart, abnormal ECG criteria requiring additional evaluation to rule out an underlying cardiomyopathy are listed in table 1.

**Evaluation of suspected isolated LVNC**

Echocardiography is usually the first investigation in the evaluation of ECG abnormalities suggestive of cardiomyopathy. The diagnosis and evaluation of suspected LVNC is quite challenging, and therefore patients should be referred to a cardiovascular specialist familiar with LVNC. Cardiac MRI provides a more detailed and accurate assessment of myocardial trabeculations and is recommended in cases with concerning or borderline findings on echocardiography. Each of these findings, particularly when observed in an asymptomatic athlete with no family history suggestive of heritable heart disease, has a low-positive predictive value for the cardiomyopathic conditions associated with an increased risk of SCD during exercise. As such, none of these ECG patterns, when found in isolation in asymptomatic athletes, clearly necessitate further evaluation. However, in athletes with cardiovascular-related symptoms or a family history of sudden death or suspected cardiomyopathy, each of these findings should prompt additional evaluation to evaluate for cardiomyopathy.

**Right bundle branch block**

RBBB is defined as a QRS complex ≥120 ms in association with a terminal (final component of the QRS complex) R\(^0\) wave in lead V1 and terminal S waves in leads I, aVL and V6 (figure 19). The R\(^0\) may extend into lead V2 but is typically absent in other precordial leads. T waves in typical RBBB are in the same direction as the terminal QRS forces and are thus inverted in leads with an R\(^0\) (V1±V2). A QRS complex duration of
100–119 ms with these morphological features is termed an incomplete RBBB.

Although RBBB may be present in various forms of heart disease, complete and incomplete RBBB are found commonly among trained athletes without underlying heart disease. This ECG pattern has been shown to reflect the exercise-induced right ventricular remodelling common in endurance sport athletes. In asymptomatic athletes with an isolated complete or incomplete RBBB, no further diagnostic evaluation is required. In contrast, athletes presenting with symptoms suggestive of cardiomyopathy, a family history of sudden death or suspected cardiomyopathy, an RBBB with atypical features (extensive TWIs, ST-segment elevation or a markedly prolonged R'), or a RBBB in conjunction with other abnormal ECG findings should be further evaluated.

**Non-Specific intra-ventricular conduction delay**

IVCD is defined as a QRS complex >110 ms that does not have morphological features consistent with either LBBB or RBBB. IVCD has been documented among patients with...
cardiomyopathy, but is also frequently seen in healthy athletes. The physiology underlying IVCD in athletes remains incompletely understood but likely includes some combination of neurally mediated conduction fibre slowing and increased myocardial mass.

Digital analysis of QRS duration can outperform standard visual measurement because the first onset and last offset in all of the leads can be considered. In asymptomatic athletes with an isolated IVCD with a QRS duration of 100–139 ms, no further diagnostic evaluation is required. In contrast, athletes presenting with symptoms suggestive of cardiomyopathy, a family history of sudden death or suspected cardiomyopathy, an IVCD with marked QRS prolongation (≥140 ms) or an IVCD in tandem with other abnormal ECG findings should be further evaluated.

Isolated premature ventricular contractions
PVCs are electrical impulses that originate from myocardial tissue below the AV node. They are defined as QRS complexes >100 ms that are not preceded by a triggering p-wave. PVCs may reflect pathological myocardial ‘irritability’ due to a cardiomyopathy, an underlying systemic disease process, or may be a completely benign normal variant. PVCs are common in athletes with high vagal tone and resting bradycardia and may increase in frequency in parallel with physical fitness. A single PVC captured during a routine 12-lead ECG in an asymptomatic athlete does not require further evaluation, unless the athlete performs a high-intensity endurance sport (mainly cycling, triathlon, rowing or swimming). In this select group of high-intensity endurance athletes, a single PVC, especially if it has an LBBB morphology, may be a hallmark of ‘exercise-induced’ ARVC, and further evaluation should be considered. The presence of PVCs in an athlete with cardiovascular symptoms or a family history of sudden death or suspected cardiomyopathy should prompt further evaluation. In addition, multiple PVCs (2 or more) during a single ECG tracing (10 s), multifocal PVCs or PVCs found in tandem with other abnormal ECG findings should be further evaluated.

PULMONARY HYPERTENSION
Pulmonary hypertension (PHT) is caused by a variety of aetiologies that result in elevation in the pulmonary artery pressure (mean pulmonary artery pressure greater than or equal to 25 mm Hg) and elevation in the pulmonary vascular resistance. As a result of the increased afterload on the right heart, patients are predisposed to develop right heart failure and are at risk for sudden death. PHT is a rare cause of sudden death in athletes but may be suggested by ECG abnormalities and thus a clinically relevant finding in the cardiovascular care of athletes.

ECG findings in pulmonary hypertension
The ECG findings in PHT are due to physiological and anatomic adaptations of the right heart as a result of elevated pulmonary artery pressures and/or pulmonary vascular resistance. Findings suggestive of PHT include right ventricular hypertrophy (RVH), right axis deviation, right ventricular strain and right atrial enlargement (figure 20). In adults with idiopathic PHT, 87% demonstrated RVH and 79% demonstrated right axis deviation. However, in patients with PHT, the ECG remains an inadequate screening tool to completely rule out the presence of this disease.

Figure 20  ECG from a patient with pulmonary hypertension demonstrating evidence of right-axis deviation >120° (A), right ventricular hypertrophy (B), right atrial enlargement (C) and right ventricular strain (D). This figure is only reproduced in colour in the online version.

Right axis deviation
Right-axis deviation is defined as a frontal plane QRS axis of >120° (figure 20).

Right atrial enlargement
Right atrial enlargement is defined as a P wave greater than or equal to 2.5 mm in leads II, III and aVF (figure 20).

RV strain
Right ventricular ‘strain’ is defined as ST depression and TWI in the right precordial leads (V1–V3) (figure 20). As with LVH, these ST-T changes are referred to as ‘secondary ST-T abnormalities.’

Evaluation of suspected pulmonary hypertension
Evaluation should include clinical assessment with appropriate diagnostic testing. Pulmonary artery pressures often can be assessed by Doppler echocardiography, and both echocardiography and cardiac MRI can evaluate RVH and function, and assess for secondary causes of PHT such as intracardiac shunts. Definitive diagnosis of PHT is made by cardiac catheterisation.

CONCLUSIONS
The cardiomyopathies are a heterogeneous group of heart muscle diseases associated with important clinical implications. In aggregate, HCM, ARVC, DCM and LVNC underlie the majority of autopsy-positive sudden death cases in young athletes. Each of these cardiomyopathies can manifest in athletes with a broad spectrum of clinical severity ranging from completely asymptomatic to markedly symptomatic disease with associated exercise limitations. The ECG plays an important role in the cardiovascular assessment of athletes given its capacity to detect cardiomyopathies. As delineated in this paper, there is a concise list of ECG findings that may indicate the presence of an underlying cardiomyopathic condition. Importantly, these ECG findings are not characteristic of the benign exercise-induced cardiac remodelling common in athletes, and, thus, the ECG can be useful for differentiating physiological cardiac enlargement in athletes from pathological myocardial disease.

Clinicians charged with the cardiovascular care of athletes should be familiar with the ECG findings associated with cardiomyopathy. During pre-participation screening that includes the use of ECG, asymptomatic athletes with any of these abnormal findings should undergo further testing. Athletes presenting with symptoms that may be indicative of an underlying cardiomyopathy (ie, exercise intolerance, inappropriate exertional dyspnoea, chest pain, palpitations or syncope) should undergo a prompt evaluation including an ECG. The symptomatic athlete with an ECG suggestive of a cardiomyopathy requires a comprehensive and definitive assessment that will include some combination of non-invasive cardiac imaging, exercise testing and ambulatory rhythm monitoring. This evaluation should be conducted by a sports medicine team that includes a cardiovascular specialist familiar with cardiomyopathic diseases and with experience in caring for athletes.

Additional resources
For a free online training module on ECG interpretation in athletes, please visit: http://learning.bmj.com/ECGathlete
For the November 2012 BJSM supplement on ‘Advances in Sports Cardiology,’ please visit: http://bsjm.bmj.com/content/46/Suppl_1.toc

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Abnormal electrocardiographic findings in athletes: recognising changes suggestive of cardiomyopathy

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Abnormal electrocardiographic findings in athletes: recognising changes suggestive of primary electrical disease


This document was developed in collaboration between the American Medical Society for Sports Medicine (AMSSM), the Section on Sports Cardiology of the European Association for Cardiovascular Prevention and Rehabilitation (EACPR), a registered branch of the European Society of Cardiology (ESC), the FIFA Medical Assessment and Research Center (F-MARC), and the Pediatric & Congenital Electrophysiology Society (PACES).

ABSTRACT
Cardiac channelopathies are potentially lethal inherited arrhythmia syndromes and an important cause of sudden cardiac death (SCD) in young athletes. Other cardiac rhythm and conduction disturbances also may indicate the presence of an underlying cardiac disorder. The 12-lead ECG is utilised as both a screening and a diagnostic tool for detecting conditions associated with SCD. Fundamental to the appropriate evaluation of athletes undergoing ECG is an understanding of the ECG findings that may indicate the presence of a pathological cardiac disease. This article describes ECG findings present in primary electrical diseases afflicting young athletes and outlines appropriate steps for further evaluation of these ECG abnormalities. The ECG findings defined as abnormal in athletes were established by an international consensus panel of experts in sports cardiology and sports medicine.

INTRODUCTION
Inherited primary arrhythmia syndromes are known causes of sudden cardiac death (SCD) in young athletes. Channelopathies represent a heterogeneous group of genetically distinct cardiovascular disorders associated with sudden death and ventricular arrhythmias from disturbed function of the cardiac ion channel.1–3 Ventricular pre-excitation and other disturbances of cardiac conduction also are associated with diseases predisposing to SCD in young athletes.4 Primary electrical disorders may present with disease-related symptoms or be asymptomatic and thus only identified by abnormal testing during pre-participation screening. The 12-lead ECG is commonly abnormal among athletes with pathological cardiac disease, and clinicians responsible for ECG interpretation in athletes must be familiar with key findings associated with conditions at risk for SCD. This paper will review ECG findings associated with primary electrical diseases relevant to the care of the young athlete. Initial testing for further evaluation of abnormal ECG findings is also presented.

DISTINGUISHING NORMAL FROM ABNORMAL
A challenge in the use of ECG for screening or diagnostic evaluations in athletes is the ability to accurately differentiate findings suggestive of a potentially lethal cardiovascular disorder from benign physiological adaptations occurring as the result of regular, intense training (ie, athlete’s heart). Several reports have outlined ECG criteria intended to distinguish normal ECG findings in athletes from ECG abnormalities requiring additional evaluation.5–10

On 13–14 February 2012, an international group of experts in sports cardiology and sports medicine convened in Seattle, Washington, to define contemporary standards for ECG interpretation in athletes. The objective of the meeting was to assist physicians distinguish normal ECG alterations in athletes from abnormal ECG findings that require additional evaluation for conditions that predispose to SCD.11 A review of normal ECG findings in athletes is presented separately.12

In this paper, abnormal ECG findings suggestive of an ion channel or conduction disorder associated with SCD in athletes are presented including congenital long and short QT syndromes (LQTS and SQTS), catecholaminergic polymorphic ventricular tachycardia (CPVT), Brugada syndrome (BrS), ventricular pre-excitation, supraventricular tachycardias (SVT), atrioventricular (AV) blocks and premature ventricular contractions (PVCs). Table 1 summarises a list of abnormal ECG findings unrelated to athletic training that may suggest the presence of a pathological cardiac disorder and should trigger additional evaluation in an athlete.

THE CONGENITAL QT SYNDROMES
Congenital long QT syndrome (LQTS) and short QT syndrome (SQTS) are potentially lethal,
genetically mediated ventricular arrhythmia syndromes with the hallmark electrocardiographic feature of either QT prolongation (LQTS) or markedly shortened QT intervals (SQTS) (Figures 1–3).

Symptoms if present include arrhythmic syncope, seizures or aborted cardiac arrest/sudden death stemming from either torsades de pointes (LQTS) or ventricular fibrillation (SQTS).

The pathophysiology of the QT syndromes is understood as either delayed ventricular repolarisation (LQTS) or accelerated ventricular repolarisation (SQTS) originating primarily from loss-of-function (LQTS) or gain-of-function (SQTS) mutations in genes encoding voltage-gated potassium channels (Kv7.1 and Kv11.1) that govern phase 3 repolarisation in the ventricular myocytes. Currently, 13 LQTS-susceptibility genes and 3 SQTS-susceptibility genes have been identified and account for over 75% of LQTS and <20% of SQTS.1

Prevalence
SQTS is extremely uncommon affecting less than 1:10 000 individuals. Although once considered similarly rare, LQTS is now estimated to affect 1 in 2000 individuals and given the subpopulation of so-called ‘normal QT interval’ or ‘concealed’ LQTS, this may be an underestimate.13

Contribution as a cause of SCD
Among individuals between 1 and 40 years of age, approximately 25–40% of sudden unexpected deaths are classified as autopsy negative sudden unexplained death (SUD) lacking necropsy findings to establish the cause and manner of death.14–16 Here, cardiac channelopathies such as LQTS, SQTS, CPVT and BrS are considered as possible culprits and have been implicated by postmortem genetic testing as the root cause for up to 25–35% of SUD in selected cohorts.17–19 LQTS is the most common channelopathy responsible for about 15–20% of SUD.20 SQTS is a very rare cause of autopsy negative SUD.14

In a series of young athletes with SCD from the USA (n=1049), a precise cause of death was identified in 690 cases.4 LQTS was implicated in less than 4% of cases (23/690) with an identified cause.4 However, this estimate does not include one-third of the total cases in the series (359/1049) with no precise diagnosis but in whom post-mortem genetic testing was not documented. Thus, it is likely ion channelopathies or

Table 1 Abnormal ECG findings suggestive of primary electrical disease

<table>
<thead>
<tr>
<th>Abnormal ECG Finding</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Ventricular pre-excitation</td>
<td>PR interval &lt;120 ms with a delta wave (slurred upstroke in the QRS complex) and wide QRS (≥120 ms)</td>
</tr>
<tr>
<td>Long QT interval*</td>
<td>QTc ≥470 ms (male)</td>
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<tr>
<td>Short QT interval*</td>
<td>QTc ≤320 ms</td>
</tr>
<tr>
<td>Brugada-like ECG pattern</td>
<td>High take-off and downsloping ST segment elevation followed by a negative T wave in ≥2 leads in V1–V3</td>
</tr>
<tr>
<td>Profound sinus bradycardia</td>
<td>&lt;30 bpm or sinus pauses ≥3 s</td>
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<tr>
<td>Atrial tachyarrhythmias</td>
<td>Supraventricular tachycardia, atrial-fibrillation, atrial-flutter</td>
</tr>
<tr>
<td>Premature ventricular contractions</td>
<td>≥2 PVCs per 10 s tracing</td>
</tr>
<tr>
<td>Ventricular arrhythmias</td>
<td>Couplets, triplets, and non-sustained ventricular tachycardia</td>
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</tbody>
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*The QT interval corrected for heart rate is ideally measured with heart rates of 60–90 bpm. Consider repeating the ECG after mild aerobic activity for borderline or abnormal QTc values with a heart rate <50 bpm.

Note: These ECG findings are unrelated to regular training or expected physiological adaptation to exercise, may suggest the presence of pathological cardiovascular disease, and require further diagnostic evaluation.

Figure 1 ECG demonstrating a profoundly prolonged QT interval. The computer derived a QTc measurement of 362 ms which was inaccurate. Manual measurement of the QTc was 760 ms.
Accessory electrical pathways represent a larger percentage of SCD in young athletes than previously reported.

**Diagnostic criteria**
Both QT syndromes are diagnosed based on a combination of symptoms, family history, electrocardiographic findings and genetic testing. The Gollob score is used for SQTS while the Schwartz-Moss score is used to invoke low, intermediate and high probability for LQTS. For LQTS, genetic testing is recommended for: (1) any patient where a cardiologist has an index of suspicion for LQTS (intermediate or high probability score), or (2) an asymptomatic patient with no family history.
but an incidental ECG finding with a QTc >480 ms prepuberty and >500 ms postpuberty that is confirmed on repeat ECG testing. Genetic testing may be considered for individuals with an incidental QTc finding (repeated) of ≥460 ms prepuberty and ≥480 ms postpuberty.

### Calculating the QTc

Whether the ECG was obtained for screening or diagnostic purposes, the heart rate corrected QT interval (QTc) derived by the computer must be confirmed manually because the accuracy of the computer generated QTc is only about 90–95%. Notably, the computer-derived QTc for the ECG in figure 1 was off by about 400 ms as the true QTc was around 760 ms but was read as 360 ms. Although this is an extreme example of an inaccurate computer QTc calculation, studies have also suggested that the ability of cardiologists and even heart rhythm specialists to accurately measure the QTc is suboptimal. However, an accurate assessment of the QTc can be taught and achieved by adhering to the following six principles.

First, most ECG machines utilise the Bazett’s heart rate correction formula (QTc=QT/√RR; note the RR interval is measured in seconds). 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 the population-based QTc distributions most frequently used Bazett-derived QTc values.

Second, Bazett’s formula loses accuracy at slow heart rates and can underestimate the individual’s inherent QTc at heart rates <60 bpm, especially at heart rates <50 bpm. Accordingly, if an athlete has a heart rate <50 bpm, repeat the ECG after some mild aerobic activity to get his/her heart rate into a range (60–90 bpm) where the formula is most accurate.

Third, if there is beat-to-beat variation in heart rate (sinus arrhythmia) which is common among athletes, do not take the maximum QT interval on the ECG and divide it by the square root of the shortest RR interval, which will grossly overestimate the QTc. Instead, it is more accurate to derive an average QT interval and average RR interval.

Fourth, to perform a manual confirmation, the critical issue is identifying the end of the T wave since the onset of the QRS is seen easily. The rhythm strip at the bottom of the ECG generally includes leads II, V1 and/or V5, and lead II and V5 usually provide the best delineation of the T wave.

Fifth, it is incorrect to include the commonly seen low-amplitude U wave in the QT calculation. Such U wave inclusion will inflate greatly the QTc. Instead, follow the ‘Teach-the-Tangent’ or ‘Avoid-the-Tail’ method as shown in figure 4.

Sixth, the morphology of the T waves, not just the length of the QT interval, can suggest the presence of a QT syndrome. As shown in figure 5, a notched T wave in the lateral precordial leads may be a tip off to LQTS 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 Bazett’s calculation. As such, the computer generated QTc has been confirmed manually. If, however, the manually measured QT interval is >10 ms different than the computer’s QT measurement, calculate an average RR interval and recalculate the QTc using Bazett’s formula.

QTc cut-offs: how long is too long? How short is too short? Figure 6 shows the overlap between the distribution of QTc values in population-derived cohorts of healthy individuals.
compared to patients with genetically confirmed LQTS.\textsuperscript{29–31} Considering that 25–40% of genotype positive individuals (mostly relatives of the index cases/probands) have normal QT interval/concealed LQTS, it must be acknowledged that no screening programme will identify all persons with either LQTS or SQTS.\textsuperscript{32} Instead, the QTc cut-off values, where the QTc measurement requires further evaluation, must be chosen carefully to balance the frequency of abnormal results and the positive predictive value that an SQTS or LQTS host has been identified.

Published definitions of a ‘prolonged QTc’ requiring further evaluation have varied. Guidelines from the European Society of Cardiology for ECG interpretation in athletes define a QTc value of >440 ms in men and >460 ms in women (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.\textsuperscript{6} In the USA, the AHA/ACC/HRS guideline has dropped the term ‘borderline’ QT prolongation and instead now annotates a QTc ≥450 ms in men and ≥460 ms in women as ‘prolonged QTc’.\textsuperscript{33} Concern has been raised that these QTc cut-offs will produce a high number of false-positive test results if followed in a screening population of athletes.\textsuperscript{29} However, one study of 2000 elite athletes age 14–35 in the UK found that only 0.4% of athletes had a QTc >460 ms, and another study detailing ECG findings in 32,561 young adults from the USA undergoing screening reported only 0.3% had a QTc >460 ms.\textsuperscript{10,34} Nonetheless, these QTc thresholds (440–460 ms) represent the approximate 90–95th percentile values for QTc distribution in the general population, and utilisation of these QTc cut-offs in a screening programme of athletes will have a <1% positive predictive value for LQTS in the absence of any personal or family history to indicate disease.\textsuperscript{29,31} In 2011, an international statement on ECG interpretation in athletes recommended that all athletes
with a QTc >470 ms in men and >480 ms in women undergo further evaluation for LQTS to better balance false-positive and false-negative findings.\(^7\)

Accordingly, it seems prudent to shift the QTc cut-off values that should trigger further evaluation in asymptomatic athletes with no concerning family history. This consensus group recommends cut-off values around the 1st percentile (QTc ≤320 ms) for a short QTc and around the 99th percentile (>240 ms in men and >240 ms in postpubertal women) to indicate a prolonged QTc. These cut-offs will improve the positive predictive value if ECG is used for athlete screening while still identifying the most overt QT abnormalities and those individuals most likely to experience QTc-related adverse events. These cut-offs are also consistent with thresholds defined by the 36th Bethesda Conference.\(^35\)

However, it is critical that an athlete should not be obligated to a diagnosis of either SQTS or LQTS for falling below or above these QTc cut-off values, but rather these cut-off values should trigger the need for further evaluation. In other words, a prolonged QTc measurement on a single ECG does not equal LQTS. Further evaluation as outlined below and the use of scoring systems that account for personal symptoms, family history, as well as electrocardiographic features are helpful in clarifying the diagnosis.

QTc cut-offs: relative versus absolute risk of a QT syndrome
By definition, the 99th percentile cut-off for genetically confirmed LQTS assumes 1% of those with a value outside of this QTc threshold (>240 ms in men, >280 ms in women) have a false-positive result. If one assumes that the prevalence of LQTS is 1 in 2000 individuals, and approximately half of these individuals will have a QTc above and half below these QTc thresholds (1:4000), then the positive predictive value of detecting true disease for a QTc outside the cut-off value is about 2.5%.\(^29\) \(^30\) However, once a prolonged QTc is identified, that individual has a 1 in 40 (rather than 1:2000) chance of having true disease. In other words, with no additional corroborative evidence, a single prolonged QTc value above the defined cut-off would suggest a 50-fold increase in relative risk for LQTS but only a 2.5% absolute risk. However, in an athlete with a QTc >500 ms, the predictive value now favours the presence of not only LQTS but possibly higher risk LQTS.\(^34\) \(^36\)

Evaluation of a possible long or short QT syndrome
An athlete identified as crossing the aforementioned QTc thresholds (>240 ms men, ≥480 ms women) should have their personal history (exercise/emotion/auditory-triggered syncope or seizures) and family history (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) reviewed. If their personal/family history is positive, then the athlete should be referred to a heart rhythm specialist for further evaluation. If the personal/family history is negative, then a repeat ECG should be obtained. If the follow-up ECG is within the QTc cut-off values, then no additional evaluation is needed and the athlete should be reassured and may continue sports participation.

On the other hand, if the repeat ECG still exceeds the QTc cut-off values, then a screening ECG of the athlete’s first-degree relatives (parents and siblings) should be considered and the athlete should be referred to a heart rhythm specialist or cardiologist as the possibility for newly discovered LQTS or SQTS has increased. Reversible, extrinsic factors, such as electrolyte abnormalities (hypokalaemia) or the presence of QT prolonging medications, must also be evaluated. If an athlete’s ECG shows a QTc ≥500 ms and no reversible causes are identified, then the athlete should be referred immediately to a heart rhythm specialist or cardiologist as the probability of LQTS and future adverse events has increased.\(^36\) Further testing including provocative treadmill stress and/or epinephrine QT stress testing along with genetic testing need to be considered carefully and should be performed and interpreted by a cardiologist familiar to the disease.

**CATECHOLAMINERGIC POLYMORPHIC VENTRICULAR TACHYCARDIA**
CPVT is an inherited arrhythmogenic disorder characterised by ventricular ectopy induced by exercise or emotional stress. CPVT is a primary electrical disease involving cardiac channels, particularly the RYR2-encoded cardiac ryanodine receptor/calcium release channel, and typically occurs in patients with structurally normal hearts. Exercise or acute emotion can lead to progressive ventricular ectopy eventually causing a fast ventricular tachycardia. This tachycardia may lead to syncope and in some cases ventricular fibrillation and sudden death. The average age of presentation of CPVT is between 7 and 9 years old, but onset as late as the fourth decade of life has been reported.\(^37\) If untreated, approximately 30% of individuals experience cardiac arrest and up to 80% have at least one episode of syncope.\(^38\)

**Prevalence and contribution as a cause of SCD**
The prevalence of CPVT is estimated to be around 1 in 10 000 people, although the true prevalence of this condition is not known.\(^39\) The incidence of SCD in athletes from CPVT is also not known. However, one study showed a prevalence of 9.4% in adults with sudden unexplained death and a pooled analysis found 4–10% of autopsy negative SCD could be attributed to CPVT.\(^20\) \(^40\) \(^41\)

**Diagnostic criteria and ECG findings in CPVT**
CPVT should be considered in any person who experiences syncope during exercise or extreme emotion, particularly in those who experience syncope during maximal exertion or have repeated episodes of syncope with exercise. CPVT cannot be diagnosed on the basis of a resting ECG, as the resting ECG is normal. An echocardiogram is also typically normal. As ventricular ectopy occurs only in the face of increased emotion or exercise, exercise stress testing is the key test in the evaluation of CPVT. As exercise workload increases, there is typically an increase in the amount of ventricular ectopy which ultimately may result in polymorphic ventricular tachycardia\(^42\) (figure 7). This graded, exercise-induced ventricular ectopy differentiates CPVT from benign PVCs which typically suppress with exercise.

The ventricular tachycardia in CPVT can be bidirectional with a 180° rotation of the QRS complex alternating from beat to beat. However, exercise-induced bidirectional ventricular tachycardia is uncommonly seen in patients with genetically proven CPVT. Instead, as heart rate increases during exercise there will be increasing ventricular ectopy initially with isolated PVC’s, then ventricular bigeminy, progressing to ventricular couplets and ventricular tachycardia if exercise persists.\(^33\)

**Evaluation of possible CPVT**
Evaluation of possible CPVT should be referred to a cardiologist. An exercise stress ECG such as an exercise treadmill test

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Original articles

may be diagnostic in CPVT. Infusion of intravenous catecholamines (isoproterenol) during ECG monitoring or CPVT genetic testing may confirm the diagnosis in questionable cases. 

**BRUGADA SYNDROME**

BrS is a primary electrical disease which is characterised by the distinctive ECG pattern of 'high take-off' ST segment elevation in the right precordial leads and predisposes to ventricular fibrillation and sudden death in the absence of clinically demonstrable structural heart disease. Loss-of-function defects in the SCN5A gene, which encodes for the α-subunit of the sodium channel, accounts for approximately 20–25% of BrS and approximately 40% of BrS accompanied by prolonged PR intervals. 

**Prevalence and contribution as a cause of SCD**

The syndrome is estimated to account for up to 4% of all sudden deaths in the general population and 5–20% of sudden deaths victims with a structurally normal heart at autopsy. Ventricular fibrillation and sudden death in patients with BrS occurs more commonly during rest and sleep and is unrelated to exercise.

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**Figure 7** 15-year-old boy undergoing exercise stress test for evaluation of CPVT. Polymorphic ventricular ectopy is evident late in stage 3 of the exercise stress test. Heart rate 148 bpm. This figure is only reproduced in colour in the online version.

**Figure 8** Brugada pattern ECGs. Type 1 Brugada pattern ECG is defined as a high-take off and downsloping ST segment elevation ≥2 mm followed by a negative T-wave in at least two contiguous leads (V1–V3). Type 2 and 3 Brugada pattern ECGs 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.
Diagnostic criteria
In 2002, a consensus conference endorsed by the Heart Rhythm Society and the European Heart Rhythm Association proposed ECG criteria for the diagnosis of BrS. Three types of Brugada ECG (Br-ECG) patterns were defined: the ‘diagnostic’ (type 1) which is characterised by a ‘coved-type’ ST segment elevation in the right precordial leads, and the ‘non-diagnostic’ (types 2 and 3) which show a ‘saddle-back’ configuration. (figure 8) The type 1 Br-ECG may be unmasked or worsened by sodium channel blockers such as ajmaline, flecainide and procainamide. Higher placement of the V1 and V2 electrodes in the second intercostal space (rather than the fourth intercostal space) also can precipitate a type 1 Brugada ECG pattern. Conversion of type 2 and 3 Br-ECG to type 1 by sodium channel blocker administration is considered diagnostic for a positive Brugada ECG pattern and is used in clinical practice for diagnosis and management of BrS according to current guidelines.

ECG findings in Brugada syndrome
Type 1 Brugada pattern ECG is defined as a high-take off and downsloping ST segment elevation ≥2 mm followed by a negative T-wave in at least two contiguous leads (V1–V3) (figure 8). Type 2 and 3 Brugada pattern ECGs 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 (figure 8).

The downsloping ST elevation in Brugada type-1 ECG should be distinguished from the ‘convex’ ST segment elevation characteristic of early repolarisation in a trained athlete (figure 9). Measuring the ST elevation at the start of the ST segment/ J-point (STJ) and 80 ms after the start of the ST segment (ST80) can help differentiate the slope of the ST segment. In Brugada type-1 pattern the downsloping ST segment will have a STJ/ST80 ratio >1. In early repolarisation patterns in an athlete the initial upsloping of the ST segment will produce a STJ/ST80 ratio <1 (figure 9).

Evaluation of possible brugada syndrome
Patients with a type 1 Brugada pattern ECG should be referred to a cardiac electrophysiologist for further evaluation and management.

VENTRICULAR PRE-EXCITATION
The PR interval is the time required for the electrical impulse to travel from the sinus node through the AV node to the Purkinje fibres, and it is measured from the beginning of the P wave to the beginning of the QRS. Ventricular pre-excitation occurs when an accessory pathway of electrical activation bypasses the AV node. As a result, there is 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.

Prevalence and contribution as a cause of SCD
WPW pattern occurs in approximately 1 : 1000 athletes. The presence of an accessory pathway can predispose an athlete to sudden death if the athlete also goes into atrial fibrillation. Rapid conduction of atrial fibrillation across the accessory pathway can result in ventricular fibrillation. The risk of sudden death associated with asymptomatic WPW pattern in most population-based studies is 0.1% per year in adults. There is evidence to suggest a higher risk of sudden death in asymptomatic children and younger adults with WPW pattern. Overall, WPW accounted for 1% of cardiovascular deaths in a long-term registry of sudden death in athletes.

Diagnostic criteria and ECG findings in ventricular pre-excitation
WPW pattern is defined as a short PR interval (<120 ms), the presence of a delta wave (slurring of the initial QRS) and a wide QRS (>120 ms). (figure 10)

WPW pattern should be differentiated from a low atrial rhythm with a short PR interval that is a common finding in athletes (figure 11). The short PR is a result of the impulse being generated by an atrial focus outside the sinus node and closer to the AV node. Due to the proximity to the AV node, atrial conduction time is reduced and the PR interval is shortened. Findings that can help differentiate this from pre-excitation include an atypical P wave axis (negative P wave in the inferior leads) suggesting the atrium is activated from bottom to top rather than top to bottom as in sinus rhythm (figure 11). No further evaluation is recommended for asymptomatic athletes with only a short PR interval and no other ECG abnormality.

Figure 9 Brugada type-1 ECG (left) should be distinguished from early repolarisation 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 characterised by a STJ/ST80 ratio >1. Early repolarisation patterns in an athlete show an initial ‘upsloping’ ST segment elevation with STJ/ST80 ratio <1.
Evaluation of WPW

The diagnostic evaluation of asymptomatic athletes with WPW pattern remains controversial and is conducted usually by an electrophysiologist. Stratification methods for the risk of sudden death include invasive and non-invasive tests. Non-invasive measures of a low-risk accessory pathway include intermittent pre-excitation during sinus rhythm and abrupt, complete loss of pre-excitation during an exercise stress test.56 57 If non-invasive testing is inconclusive, electrophysiology testing should be considered. Characteristics of a high-risk pathway are generally determined during an electrophysiology study by the shortest pre-excited RR interval during induced atrial fibrillation. If the shortest pre-excited RR interval is measured as ≤250 ms [240 beats/min (bpm)] then the pathway is deemed high risk.52 Young athletes with a shortest pre-excited RR interval ≤250 ms should proceed with transcatheter ablation.58 An echocardiogram should also be considered due to the association of WPW with Ebstein’s anomaly and cardiomyopathy.

SUPRAVENTRICULAR TACHYCARDIAS

SVT are heart rhythms >100 bpm, originating from the sinus node, atrial tissue or involving the AV node. The most common SVT is sinus tachycardia, seen during exercise, anxiety, fever, infection, dehydration, hyperthyroidism, anaemia, pulmonary disease, heart failure, stimulant use and other causes. Paroxysmal SVT includes AV nodal re-entrant tachycardia (AVNRT), AV reciprocating tachycardia (AVRT), atrial tachycardia and other rare tachycardias (figure 12). AVNRT is a circuit involving a slow and fast pathway entering and exiting the AV node. The electrical circuit usually travels down the slow pathway and up the fast pathway, but can be reversed or even involve multiple slow pathways. AVRT involves an accessory AV bypass pathway where the circuit typically conducts down the AV node and up the bypass pathway, producing a narrow complex tachycardia. In WPW pattern, the circuit also may conduct down the bypass pathway and up the AV node. Since the ventricular myocardium is activated by the bypass tract and not the His-Purkinje system, there is a resultant wide complex tachycardia. More commonly in AVRT there is a concealed bypass pathway that only conducts retrograde and, therefore, is not seen on ECG. Lastly, atrial tachycardia occurs from an
abnormal focus within the atrium that activates faster than the sinus node.

Atrial fibrillation and flutter are other types of SVT. Atrial fibrillation is the most common abnormal SVT and is defined by the rapid and irregular atrial electrical activation with the resultant dysfunctional atrial mechanical activation. Atrial flutter is characterised by a macro-reentrant circuit within the atrium and is more organised than atrial fibrillation.

Prevalence of SVT

The incidence of SVT is difficult to determine because of the various study populations and ascertainment methods. Many studies have included elderly patients, with coronary disease or heart failure, which are not relevant to a younger athletic population. SVT is rarely found on a screening ECG, as most young athletes are symptomatic with SVT. In a study of 32,652 Italian subjects, 29 (0.09%) had SVT and 5 (0.02%) had atrial fibrillation.

Figure 12 Paroxysmal supraventricular tachycardia (SVT) refers to narrow complex tachycardias including atrioventricular nodal re-entrant tachycardia (AVNRT), atrioventricular reciprocating tachycardia (AVRT), atrial tachycardia and other rare tachycardias. Atrial fibrillation and flutter are types of SVT but do not fall into the paroxysmal classification. Here, there is a narrow complex tachycardia at 240 bpm, which was later found to be AVRT. This figure is only reproduced in colour in the online version.

Figure 13 The top row demonstrates atrioventricular nodal re-entrant tachycardia (AVNRT) and the bottom row is in normal sinus rhythm in the same patient. In lead II, retrograde P waves are present at the end of the QRS complex. In lead V1, a pseudo-R’ with right bundle branch block pattern is present due to the retrograde P waves. These deflections are not seen on the ECG when in sinus rhythm. This figure is only reproduced in colour in the online version.
fibrillation or flutter. Uncontrolled supraventricular arrhythmias led to disqualification in 73 of 42,386 subjects in the Veneto region of Italy from 1982 to 2004. In 32,561 screening ECGs, an ectopic atrial tachycardia was found in only 4 (0.01%) and atrial fibrillation in 2 (<0.01%). These studies suggest the presence of SVT to be quite rare on a screening ECG.

**Contribution as a cause of SCD**

SVT, atrial fibrillation and atrial flutter very rarely lead to SCD, but are more likely to lead to symptoms (ie, palpitations) that prevent intense physical activity due to uncontrolled ventricular rates or lack of adequate AV mechanical synchronisation. Another concern is whether SVT or atrial fibrillation/flutter is reflective of an underlying cardiomyopathy or channelopathy that would place the athlete at risk for SCD. These may include hypertrophic or other cardiomyopathies, BrS, myocarditis, short QT syndrome or WPW.

**ECG findings in SVT**

SVT is usually narrow complex, but in the presence of a bundle branch block can be wide complex. It is often difficult to see P waves in AVNRT, since the atrium and ventricles activate near simultaneously, but it is sometimes possible to see inverted P waves in the inferior leads and a pseudo R' in V1 suggesting right bundle branch block, which are not present when in sinus rhythm (figure 13). AVRT will also usually show retrograde P waves, but they do not necessarily have to be inverted in the inferior leads. Some patients with AVRT will have a WPW pattern on their baseline ECG. Atrial tachycardia demonstrates a regular atrial rhythm which is faster than 100 bpm with P waves all of the same morphology.

The ECG in atrial fibrillation shows fibrillation waves instead of P waves (figure 14). These vary in size, morphology and frequency, but are usually low amplitude with changing shape and rate. The ventricular response to atrial fibrillation is irregular with varying QRS intervals. Atrial flutter, however, has regular atrial activity characterised by flutter waves. Counterclockwise typical atrial flutter, the most common type, shows negative, sawtooth flutter waves in leads II, III and aVF and a positive deflection in lead V1 (figure 15). The atrial activity in atrial flutter is almost always continuous with no isoelectric segment.

**Evaluation of SVT**

If paroxysmal SVT is seen, carotid sinus massage, Valsalva maneuver or facial dunking in an ice bath should be completed while recording the ECG to determine if the rhythm terminates (suggestive of AVNRT or AVRT) or ventricular rate slows to reveal hidden P waves (suggestive of an atrial tachycardia or atrial flutter). Once the patient is no longer in SVT, a baseline ECG should be completed to assess for WPW, and if seen, an electrophysiologist should be consulted to discuss electrophysiology study and possible ablation. If the baseline ECG is normal, it is reasonable to obtain an echocardiogram to look for structural heart disease and consider an electrophysiology consultation to discuss possible ablation.

If the rhythm is atrial fibrillation or atrial flutter, an echocardiogram and 24 h Holter monitor should be completed to look for structural heart disease and assess the heart rate throughout the day, including with exercise. If a Holter cannot be performed during exercise, treadmill testing should be completed to assess maximum heart rate while in atrial fibrillation/flutter. Thyroid, liver and renal laboratory testing also should be completed.

**OTHER ABNORMAL ECG FINDINGS**

**Profound sinus bradycardia**

Diagnostic criteria

Sinus bradycardia is one of the hallmark features of a well-conditioned athlete’s heart. It is the result of increased vagal...
tone and possible structural atrial remodelling. The sinus rate only rarely falls below 30 bpm or shows pauses of ≥3 s during an ECG recording at rest.

**Evaluation**

Profound sinus bradycardia (<30 bpm) at rest in an athlete should be evaluated further but is not necessarily pathological (figure 16). If asymptomatic and the sinus rate quickly accelerates with an increase in sympathetic tone (ie, small exercise load), then additional testing is not usually necessary. The presence of symptoms, such as decreased exercise capacity or a predisposition for vasovagal syncope, may prompt additional testing to exclude primary sinus node disease. One might also consider temporary cessation of sports activity to evaluate

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**Figure 15** ECG demonstrating atrial flutter, as evidenced by regular, sawtooth flutter waves in the inferior leads and a positive deflection in V1. No isoelectric segment is present between the flutter waves. The QRS response is regular, in this case a 4:1 pattern. This figure is only reproduced in colour in the online version.

**Figure 16** ECG in an asymptomatic long-distance runner showing profound sinus bradycardia (30 bpm) compatible with high vagal tone. The athlete showed a normal chronotropic response during exercise testing.
reversibility, although even adaptive athlete’s heart sinus brady-cardia may not be fully reversible.62 63

**Profound first-degree AV block**

**Diagnostic criteria**

The high vagal tone in athletes also leads to a slowing of AV nodal conduction, and hence a lengthening of the PR interval. It is not uncommon to see PR intervals longer than 200 ms in athletes at rest. Even significant PR prolongation ≥300 ms may occur, although this by itself is not necessarily pathological and is usually asymptomatic.

**Evaluation**

In asymptomatic athletes with a profound first-degree AV block (≥300 ms), the athlete should undergo a minimal exercise load (ie, like climbing a flight of stairs) to increase sympathetic tone. If this results in shortening and normalisation of the PR interval, the PR prolongation is due to functional (vagal) mechanisms and hence benign. If the PR interval does not normalise to ≤200 ms with exercise, a structural cause of AV conduction disturbance (such as Lyme disease or sarcoidosis) should be investigated. Athletes with a profound first-degree AV block (≥300 ms) who have symptoms (ie, syncope, palpitations) or a positive family history of cardiac disease or sudden death require additional evaluation to rule out pathological causes of heart block.

**Mobitz type II second-degree AV block**

**Diagnostic criteria**

An abrupt loss of P wave conduction (P wave with no ensuing QRS complex), without prior PR prolongation, represents Mobitz type II second-degree AV block (figure 17). If Mobitz type II or more advanced types of AV block including 2:1 or 3:1 occur during sinus rhythm, it may be indicative of underlying structural heart disease.

**Evaluation**

Suspected Mobitz type II second-degree AV block or other more advanced types of AV block (2:1 or 3:1 block) should first be differentiated from Wenckebach (Mobitz type I) second-degree AV block. Wenckebach (Mobitz type I) block is present when there is PR prolongation before a blocked P wave and a shorter PR in the first conducted beat after the block. Mobitz type I second-degree AV block is usually a functional block from increased vagal tone and does not constitute pathology in an athlete. Further diagnostic evaluation can be done with an ECG after minor exercise, as a slight increase in sympathetic tone will resolve the conduction disturbance in physiological cases. A Holter monitor (or other form of long-term ECG recording) also can assist in clarifying the type of AV block. Mobitz type II or higher degree (2:1 or 3:1) AV block requires further evaluation for underlying pathological cardiac disease.

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**Figure 17** ECG showing Mobitz type II second-degree AV block. Note the presence of P waves with loss of conduction and no QRS complex (arrows) and without PR prolongation in the beats prior, nor PR shortening in the beats after (which would suggest Mobitz type I). Mobitz type II second-degree AV block in an athlete is not due to increased vagal tone and should prompt evaluation for underlying conduction disease. This figure is only reproduced in colour in the online version.
Third-degree AV block/complete heart block

Diagnostic criteria
Complete heart block is not an expression of athlete’s heart and should be considered an abnormal finding requiring additional evaluation.

Evaluation
With true third-degree AV block, there are more P waves than QRS complexes and the ventricular rhythm is perfectly regular due to an undisturbed junctional pacemaker (figure 18). Complete heart block can easily be

Figure 18  ECG showing third-degree (complete) AV block and a junctional escape rhythm. With third-degree AV block, there are more P waves than QRS complexes and the ventricular rhythm is perfectly regular due to an undisturbed junctional pacemaker. Complete heart block is not an expression of athlete’s heart and requires additional evaluation. This figure is only reproduced in colour in the online version.

Figure 19  ECG of a 35-year-old cyclist shows two premature ventricular contractions which should trigger further evaluation for underlying structural heart disease and/or more complex arrhythmias. This asymptomatic athlete had inducible ventricular tachycardia during EP study, and later received appropriate shocks from an implanted ICD.
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.

≥2 premature ventricular contractions

Diagnostic criteria

When two PVCs are recorded on a baseline (10 s) ECG, the likelihood is very high that the athlete has >2000 PVCs per 24 h. In athletes with >2000 PVCs per 24 h, underlying structural heart disease which may predispose to more life-threatening ventricular arrhythmias was found in 30% of cases, compared to only 3% of athletes with 100–2000 PVCs, and 0% of athletes with <100 PVCs on a 24 h Holter. Over half of the athletes with >2000 PVCs also had bursts of non-sustained ventricular tachycardia. Therefore, a structural cardiac abnormality should be ruled out in athletes with >2000 PVCs per 24 h.

Evaluation

Documentation of ≥2 PVCs on baseline ECG should prompt more extensive evaluation to exclude underlying cardiac disease (figure 19). However, excluding pathology may be difficult and the extent of the evaluation is controversial. At a minimum, a 24 Holter monitor, echocardiogram and exercise stress test should be done. If the Holter and echocardiogram are normal and the PVCs suppress with exercise, some experts recommend no further evaluation for an asymptomatic athlete. However, in cases with >2000 PVCs per 24 h or episodes of non-sustained ventricular tachycardia, and depending on the level of clinical concern, morphology of the PVCs and type of sport, additional evaluation may also include cardiac MRI and more extensive electrophysiological (EP) evaluation with signal averaged ECG, long-term ECG recording, invasive EP study and/or cardiac biopsy. Therefore, many such cases require referral to a heart rhythm specialist.

Considerations in high-level endurance athletes with PVCs

In high-level adult endurance athletes (such as cyclists, triathlon athletes, marathon runners and rowers), concern has been raised about right ventricular changes that may resemble familial arrhythmogenic right ventricular cardiomyopathy (ARVC), but in the absence of demonstrable desmosomal mutations or a familial history. There is evolving evidence that persistent high volume and pressure load on the right ventricle from such long-term endurance exercise may result in ‘exercise-induced’ ARVC in such athletes. Its prognosis is not benign and may result in major ventricular arrhythmias or sudden death, although many athletes with exercise-induced ARVC initially present with minor arrhythmias or symptoms. PVCs originating from the right ventricle typically show a left bundle branch block (LBBB) pattern with a predominantly negative QRS complex in V1. Therefore, in high-level adult endurance athletes, it may be reasonable to consider a single PVC, especially with LBBB morphology and superior axis, sufficient to warrant further investigation similar to that discussed above.

CONCLUSIONS

The ECG plays an important role in the cardiovascular assessment of athletes given its capacity to detect inherited primary arrhythmia syndromes and other diseases of disturbed cardiac conduction. As outlined in this paper, there is a concise list of ECG findings that are associated with the presence of a primary cardiac channelopathy or other disorder predisposing to ventricular arrhythmias. Clinicians charged with the cardiovascular care of athletes should be familiar with abnormal ECG findings indicative of primary electrical disease. During preparticipation screening that includes the use of ECG, asymptomatic athletes with any of these abnormal findings should undergo additional testing. Athletes presenting with symptoms (ie, palpitations, exercise, or emotion related syncpe or seizure-like activity) should undergo prompt evaluation including an ECG. The evaluation of an athlete with abnormal ECG findings is conducted ideally in consultation with a cardiovascular specialist familiar with primary electrical diseases and with experience caring for athletes.

Additional Resources

For a free online training module on ECG interpretation in athletes, please visit: http://learning.bmj.com/ECGathlete. For the November 2012 BJSM supplement on ‘Advances in Sports Cardiology’, please visit: http://bjsm.bmj.com/content/46/Suppl_1.toc.

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