

Review

Interpretation of the Electrocardiogram in Athletes

Keerthi Prakash, BSc(Hons), MBChB, MRCP, and Sanjay Sharma, BSc(Hons), MD, FRCP, FESC

Cardiovascular Sciences Department, St George's, University of London, London, United Kingdom

ABSTRACT

Regular intensive participation in sport results in electrical and structural alterations within the heart that can manifest on the surface electrocardiogram (ECG). In addition to the actual sporting discipline and the volume and intensity of exercise being performed, other factors play a role in the development of certain ECG patterns including sex, age, and ethnicity. In some instances, large male endurance athletes and those of African or Afro-Caribbean origin (black athletes), might exhibit ECG patterns that overlap with those seen in patients with cardiomyopathy and channelopathies, which are recognized causes of exercise-related sudden cardiac death. The ability to distinguish accurately between benign physiological electrical alterations and pathological ECG changes is crucial to prevent the unnecessary termination of an athlete's career and to minimize the risk of sudden death. Several recommendations currently exist to aid the physician in the interpretation of the athlete's ECG. In this review we discuss which ECG patterns can safely be considered benign as opposed to those that should prompt the physician to consider cardiac pathology.

RÉSUMÉ

Une participation intensive et régulière à des activités sportives peut entraîner l'apparition de modifications électriques et structurelles du cœur pouvant être décelées par un électrocardiogramme (ECG) de surface. En plus du type de discipline sportive pratiquée et de la quantité et de l'intensité d'exercice effectué, divers facteurs jouent un rôle dans l'apparition de certains tracés d'ECG, notamment le sexe, l'âge et l'origine ethnique. Dans certains cas, les athlètes de sexe masculin et de grande taille qui pratiquent un sport d'endurance et les athlètes d'origine africaine ou afro-caribéenne (de race noire) peuvent présenter des tracés d'ECG semblables à ceux des patients atteints d'une cardiomyopathie ou d'une canalopathie, lesquelles, on le sait, peuvent entraîner une mort subite d'origine cardiaque liée à l'exercice. La capacité de distinguer clairement une modification électrique physiologique bénigne d'une modification pathologique à l'ECG est cruciale pour éviter de mettre inutilement fin à la carrière d'un athlète et de minimiser le risque de mort subite. Il existe actuellement plusieurs recommandations pour aider le médecin à interpréter les tracés d'ECG d'un athlète. Nous vous présentons dans cet ouvrage une revue des tracés d'ECG qui peuvent d'emblée être considérés comme bénins ainsi que de ceux qui doivent amener le médecin à envisager la présence d'une pathologie cardiaque.

The heart of an athlete adapts physiologically over time to facilitate the generation of large and sustained increases in cardiac output. The resultant increases in vagal tone and enlarged cardiac dimensions often manifest as changes on the athlete's electrocardiogram (ECG). Sinus bradycardia and voltage criteria for left ventricular hypertrophy (LVH) are common features of the athlete's ECG, however, some electrical patterns in athletes might overlap with ECG changes observed in cardiac diseases that predispose the athlete to an increased risk of sudden cardiac death. The ability to distinguish between benign physiological ECG patterns and those considered to represent potentially serious disease is crucial for the livelihood of the athlete. It is prudent to emphasize that a

normal ECG does not exclude serious disease and should not provide any reassurance in an athlete who experiences symptoms consistent with cardiac disease.

The Normal Athlete's ECG

Bradycardia

Sinus bradycardia is seen in up to 80% of athletes,¹⁻³ however, a heart rate < 35 beats per minute (bpm) is rare and tends to only occur in endurance athletes.⁴ Sinus arrhythmia is noted in approximately 70% of athletes and first-degree atrioventricular (AV) heart block is present in 5%–13% of athletes.^{1,3,5} Mobitz type 1, junctional rhythm, and sinus pauses are also common (31%, 20%, and 37%, respectively)⁶ but predominantly at night. With increased sympathetic tone during exercise, these patterns invariably revert to sinus rhythm. A sinus pause > 2 seconds is exceptionally rare during waking hours, and as in nonathletes, should warrant further investigation for underlying conduction disease. A wandering atrial pacemaker (presence of > 2

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Corresponding author: Dr Sanjay Sharma, Cardiovascular Sciences Department, St George's, University of London, Cranmer Terrace, London SW17 0RE, United Kingdom. Tel.: +07930407772; fax: +02087250794.

E-mail: sasharma@sgul.ac.uk

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types of P-wave morphology) and an ectopic atrial rhythm are also recognized and related to increased vagal tone.

Higher degrees of AV block (Mobitz type 2 and third-degree AV block) are infrequent and are seen in only 0.5% of endurance athletes.⁶ Such findings warrant exclusion of underlying cardiac conduction disease, particularly if symptomatic with syncope or dizziness. The 2:1 AV block pattern can either be seen in Mobitz type 1 or Mobitz type 2. Although 2:1 Mobitz type 1 is usually associated with a narrow QRS complex because the conduction delay occurs at the AV node, 2:1 Mobitz type 2 is usually associated with a broad QRS complex. In asymptomatic athletes we recommend a maximal exercise test to ensure reversion to sinus rhythm and to assess chronotropic response.

Repolarization changes

Repolarization changes encompass ST segment elevation (STE) with tall T waves and are seen in approximately 60% of athletes.^{1-3,7} Although Caucasian (white) athletes show concave STE, nearly 40% of African or Afro-Caribbean (black) athletes demonstrate convex anterior STE (V_1 - V_4), often associated with either biphasic or deep T wave inversion (TWI) in the same leads (Fig. 1).^{3,8}

There has been much discussion about the definition of the early repolarization patterns (ERPs). A recent consensus document has clearly explained the definition and methods to accurately assess for the presence of ERPs.⁹ ERPs include J point elevation, a notched J point, and slurring of the S wave, which is due to a J point being buried within the S wave (Fig. 2). An ERP is likely to be associated with vagal tone because it is associated with slower heart rates and disappears on exercise or after a period of detraining.¹⁰ An ERP is seen in nearly 25% of white athletes and 40% of black athletes and is predominantly associated with ascending ST segments. Less than 5% of athletes have been found to have ERPs with horizontal ST segments.¹¹ Over the past 2 decades concern has been raised from some studies that reported an association between the presence of ERPs in the inferior or lateral leads and idiopathic ventricular fibrillation (VF).^{12,13} More recently, ERPs in the presence of horizontal or down-sloping ST segments in a case-control series of idiopathic VF in a middle-aged population, have been found to be most strongly associated with idiopathic VF.¹⁴ Only 1 study comprised of just 21 athletes who had suffered a sudden idiopathic cardiac arrest, suggested that the presence of J point elevation and/or slurred QRS in the inferolateral leads without STE was more common in those who had a cardiac arrest. During a follow-up period of over a median of 36 months, there was no significant difference in the frequency of recurrent ventricular arrhythmia between those with and without ERPs.¹⁵ It should be noted, however, that the prevalence of ERPs in the control group (7.6%) was lower than in other studies. Hence we believe that at present, there is no substantive evidence that an ERP is a marker of arrhythmogenic risk in athletes. Our practice is to consider investigation of athletes with ERPs associated with down-sloping or horizontal ST segments if there is a history of syncope without warning or if there is a family history of premature cardiac death.

Voltage criteria for LVH

Increased ventricular wall thickness and increased cardiac cavity dimensions contribute to the physiological adaptation of an athlete's heart. Unsurprisingly, voltage criteria for LVH is common, and is present in as many as 70% of athletes,¹⁶ particularly in slender male athletes (with thin chest walls)¹ and in black male athletes. The Sokolow-Lyon criteria is the most commonly used definition for voltage criteria for LVH (S wave V_1 + largest R wave $V_5/V_6 \geq 3.5$ mV or R wave aVL ≥ 1.1 mV). Although other pathological causes for LVH can be present in athletes, investigation should only be pursued in the presence of other ECG changes indicative of pathological LVH, such as ST depression, pathological Q waves, and TWI, or, a family history of hypertrophic cardiomyopathy (HCM).^{17,18}

Partial right bundle branch block

Right ventricular dilation is another common feature of athletic cardiac adaptation. The resultant increased conduction time through the His-Purkinje fibres is believed to be the mechanism behind partial right bundle branch block (RBBB; rSR' in V_1 , QRS < 120 ms), which is present in up to 30% of athletes.^{1,2,7} This ECG pattern does not require investigation in asymptomatic athletes.

Effects of Sports, Age, Sex, and Ethnicity

Endurance athletes

Endurance athletes such as those who compete in long-distance running, cycling, swimming, rowing, or canoeing show the highest prevalence of bradyarrhythmia, repolarization changes, and voltage criteria for ventricular hypertrophy. The prevalence of ECG changes is similar in men and women other than a lower prevalence of voltage criteria for LVH and ERPs in women.¹⁹

Although TWI in the chest leads (with the exception of V_1 - V_2) is considered abnormal, there are data to suggest that up to 14% of endurance athletes show anterior TWI, which might extend to V_3 .¹⁹ Ordinarily, anterior TWI that involves leads beyond V_2 would raise concern about the possibility of arrhythmogenic right ventricular cardiomyopathy (ARVC), with some investigators who speculated that extensive participation in sport can result in "exercise-induced ARVC."^{20,21} However, our experience suggests that endurance athletes with physiological anterior TWI show J point elevation and high ST segments that precede the anterior TWI, whereas patients with ARVC show a nonelevated J point and isoelectric ST segments (Fig. 3) in addition to small voltage complexes in the precordial leads (< 1.8 mV), ≥ 1 ventricle ectopic on the resting ECG, and pathological Q waves.²²

Adolescent athletes

Similar to adult athletes, the prevalence of sinus bradycardia, sinus arrhythmia, and voltage criteria for LVH is higher in adolescent athletes compared with nonathletic adolescents.¹ In addition to the ECG changes that result from physiological adaptation to exercise, adolescent athletes also exhibit a higher prevalence of voltage criteria for left and

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