

Electrocardiographic Changes in Athletes of Black Ethnicity

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Abstract

Intensive participation in sport has positive physiological effects on the heart. The contractility of the heart improves, the ejection fraction increases and the muscle mass of the heart increases, thus leading to a greater cardiac output. Despite these positive effects, there is still an increased risk for acute cardiac events and an increase in cardiac muscle mass induced by sports is not always beneficial. It may cause a thickening of interventricular septum in course of hypertrophic cardiomyopathy or be a source of arrhythmia. The workload of the heart can be very high in some sports and may in some cases be the reason for sudden cardiac death. In these cases, there is often an underlying heart disease (cardiomyopathy) unknown before the actual event. Electrocardiographic examination (ECG) may reveal some of these diseases but although ECG examinations can be a useful tool to discover pathological conditions, there could be difficulties in interpreting different ECG patterns, especially in athletes. In some cases, athletes may exhibit ECG patterns that are similar to those in heart diseases such as cardiomyopathies (QRS-amplitudes, ST-segment elevation and T wave inversions in lateral leads). This pattern is even more common in athletes of African origins. Furthermore, cardiomyopathies such as hypertrophic cardiomyopathy (HCM) are more common among athletes with African heritage than in white athletes. Thus correct interpretation of ECG is crucial for several reasons: to distinguish between benign physiological ('athlete's heart') and pathological changes, to lower the risk of sudden cardiac death, and to save time and money by not undertaking further examination of the heart.

Keywords: Sport, athlete, ECG changes, gender, ethnicity

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Introduction

Intensive participation in sport has positive physiological effects on the heart. The effects of training can be detected on electrocardiographic recordings as bradycardia, repolarization changes and ventricular hypertrophy. Athletes generally show a 10-20% increase in left ventricular (LV) wall thickness and a 10% increase in LV size compared to controls. Sinus bradycardia is often occurring and is seen in up to 80% of athletes [1-3]. There are also repolarization changes in athletes presenting as ST segment elevation in electrocardiography (ECG) [1-4].

Normally ST segment elevations (STE) and T wave inversions (TWI) in the anterior (V1-V4) chest leads are considered abnormal, but statistics show that athletes of endurance sports might show TWI that can extend to V3 and this can be considered normal in athletes [5]. T wave inversions beyond these boundaries are considered abnormal but are often detected in athletes with African heritage with TWI in leads V1-V4, J point elevation and convex ST segment elevation [3,6]. Some athletes may have repolarization and structural effects on the ECG similar to cardiomyopathies. Black athletes show a higher prevalence of

voltage criteria for left and right ventricular hypertrophy and T wave inversions, compared with white athletes [3,4,7]. Among 842 cases of sudden cardiac death in athletes, hypertrophic cardiomyopathy (HCM) was found in 42% in African Americans and other minorities (164/390) whereas among white athletes HCM was found in 31% (138/452) [8]. Furthermore, wall thickness consistent with HCM is more common among black male athletes (13%) than white male athletes (2%) [3,9]. Hence, it is of a great importance to differentiate between normal and pathological changes in athletes with African heritage.

When it comes to female athletes, the repolarization changes are similar to male athletes. Women show a higher prevalence of T wave inversions in leads V1-V3 [10,11]. Despite this fact, there is a lower prevalence of voltage criteria for LV hypertrophy in women compared with men. As mentioned before, wall thickness consistent with HCM is present in 2% of white male athletes and 13% of black male athletes. Very few female athletes (1%) show dimensions and wall thickness similar to DCM and none reveal a LV wall thickness consistent with HCM [12]. The majority of studies investigating the effects of training on the heart are mainly



on male athletes even though the number of women participating in elite sports is increasing [13,14].

Review

Comparison of ECG between healthy athletes and cardiomyopathy patients

The similarities in the ECG pattern between athletes and patients with cardiomyopathies (for example HCM) are of great interest in interpreting any pathophysiological finding. This is even more crucial in assessment of ECG registration from athletes of black ethnicity. Earlier we discussed the possibility of healthy athletes to exhibit J point elevation, STE and TWI in anterior leads (V1-V4) and that it could be considered normal in athletes. This could also be seen in patients with HCM, although in these patients TWI are usually more frequently deeper and are extending to lateral (V5-V6, I, aVL) and/or involving inferior (II, III, aVF) leads [15]. ST segment depression is also quite a common abnormality in HCM, but very rare in healthy athletes. This finding is reported in approximately 50% of patients with HCM, but below 1% in healthy athletes [2,3,16-18]. This makes ST segment depression a more reliable indicator of disease.

In summary, to differentiate between ECG pattern in healthy athletes and patients with HCM, in the athletes it is more likely that TWI is limited to leads V1-V4 and associated with J point elevation and STE [3]. Patients with HCM also present ST depressions more often [2,3,16-18].

Sudden cardiac death

The U.S. National Registry of Sudden Death in Athletes, 1980-2011, showed interesting results. Among the 2406 deaths, 842 were autopsy-confirmed cardiovascular diagnoses, where males exceeded females 6.5-fold. Also, HCM was the most common cause of sudden death, occurring in 36% of these athletes, and accounting for 39% of male SCD, almost 4 times more common than among females (11%) [8]. The most common diseases causing SCD in sports being reported are cardiomyopathy and atherosclerotic coronary artery disease [19]. There is also a notable relationship between ethnicity and SCD where African-American and other minority athletes accounted for almost 50% of confirmed cardiovascular deaths, as well as more than 50% due to HCM [20].

Females are at a lower risk than males: the male to female ratio for sudden cardiac death is 10:1 [21]. This ratio could partly be explained by the fact that females have a lower participation rate in sports than men. But as we discussed earlier, the number of female participants in elite sports is increasing. Despite this fact, there seems to be an increased risk for SCD in males for various reasons, for example high concentrations of androgens and the possibility of ignoring warning symptoms [19]. Other possible causes of gender disproportionality could be the intensity of training which can be greater in males who are generally capable of more demanding physical exertion [22-24].

SCD occurs in males in many different sports, most commonly football and basketball. In females too, SCDs occur most commonly in soccer and basketball [8].

Pre-participation screening

There is an ongoing discussion concerning whether there should be mandatory screening of athletes for cardiac disease. The

main issue of ECG as part of pre-participation screening is that ECG may be abnormal in up to 50% of athletes. Improvements of criteria and the definition of what ECG changes are physiological (athlete's heart), and what are pathological, have shown that the number of false-positives will decrease, thus reducing expensive investigations [25].

Proposed criteria from European Society of Cardiology for deviant ECG were applied to ECGs in a study reported by Peliccia et al. which showed an increase of the specificity by approximately 70% [26]. This increase was primarily in athletes with voltage criteria for left ventricular hypertrophy. Even though there has been improvements of ECG-interpretation using criteria by the European Society of Cardiology and the Seattle recommendations, ECG does not always detect cardiac anomalies in athletes. Therefore it is of great importance to have access to other methods for cardiac assessment such as echocardiography and Magnetic Resonance Imaging (MRI). Using these methods makes it more reliable to differentiate between exercise-induced physiological hypertrophy and hypertrophic cardiomyopathy. But still ECG can be used as a screening method and there are several recommendations / guidelines regarding ECG changes in athletes:

Seattle recommendations/criteria [26-28]

Anterior TWI (V1-V4) in black athletes is considered normal provided it was preceded by convex ST elevation. TWI in V1-V2 is considered normal in Caucasian athletes.

European Society of Cardiology recommendations/criteria [25]

ECGs were divided into two different categories. Category 1 (class 1), which are benign training-related changes, and Category 2 (class 2), which are non-training/pathological changes.

Category 1 changes consisted of physiological ECG patterns such as:

- Sinus bradycardia
- First degree AV block
- Incomplete RBBB
- Early repolarization
- Isolated voltage criteria for LVH.

Category 2 changes consisted of non-training/pathological ECG changes such as:

- ST segment depression
- Pathological Q waves
- Ventricular pre-excitation
- TWI beyond V1 in white and beyond V4 in black athletes
- LBBB
- QTc \geq 470msec (males)
- QTc \geq 480msec (females)
- Brugada like early repolarization
- Atrial/ventricular arrhythmia
- \geq 2 PVCs per 10sec tracing

Refined criteria for borderline variants

Both the ESC and Seattle guidelines have been proven effective in interpretation of athletes' ECG. But the two guidelines only include one normal and one abnormal category. A more refined criterion however includes a borderline category consisting of : 1) left axis deviation; 2) right axis deviation; 3) voltage criteria for left

atrial enlargement; 4) voltage criteria for right atrial enlargement; 5) voltage criteria for right ventricular hypertrophy; and 6) TWI preceded by convex ST segment elevation up to V4 in black athletes. In these borderline variants, the approach is to only investigate in symptomatic athletes, in athletes with a family history of premature cardiac disease or sudden cardiac death, or if there is a combination of any 2 of the criteria in the same athlete, for example left axis deviation and voltage criteria for left atrial enlargement [30].

The refined criteria showed an improved specificity in white and black athletes compared to the ESC and Seattle recommendations. In comparison with the ESC recommendations, specificity improved from 74% to 94% in white athletes and 40% to 84% in black athletes with the refined criteria [30,31].

Conclusions

Participation in sports has physiological, electrical and structural effects on the athlete's heart. These effects can be manifested on cardiac assessment such as ECG. An athlete's ECG can however manifest differently by overlapping with the ECG appearance of cardiac diseases, such as cardiomyopathies. Studies have shown that the frequency of the deviant ECG manifestations is higher among athletes with African heritage in comparison to white athletes. At the same time, cardiomyopathies such as HCM are also more common in people and athletes with African heritage. This makes it even more important to be able to differentiate between physiological and pathological findings.

Studies have shown that repolarization changes in the ECG differ between white athletes and black athletes. The repolarization changes in healthy white athletes could encompass STE in V1-V4 and TWI in V1-V2, aVF or III. Beyond these leads it is considered abnormal in white athletes [1-4,6,7], but in black athletes, STE with TWI is more common than in white athletes. Also, these repolarization changes beyond V1-V2, aVF or III are often detected in blacks. It is also relatively common with TWI in the inferior and lateral leads in black athletes. Many of these athletes were ultimately diagnosed with a cardiomyopathy. Such repolarization changes in black athletes should therefore be further investigated to exclude an underlying cardiomyopathy [3,32,33], but the problem that repolarization changes in black athletes are similar to those in HCM remains. J point elevation, STE and TWI in anterior leads could be seen in healthy athletes but also in HCM patients. However, TWI are extending more to lateral (V5-V6, I, aVL) and/or inferior (II, III, aVF) leads in patients with HCM. Also, ST segment depression is rare in healthy athletes, but relatively common in HCM. An ST segment depression in an athlete would be considered an indicator of disease [2,3,15-18].

Due to the recommendations from ESC and Seattle, the specificity has increased and the occurrence of false-positives has decreased among both white and black athletes. There are also refined criteria for the borderline variants that have shown an improvement in the specificity in white and black athletes. The fact that using the refined criteria improved specificity from 40% to 84% in black athletes underlines the importance of increasing the use of these criteria. This would be likely to result in less time and money being spent on further investigations and possibly in fewer cases of sudden cardiac death [30,31].

In healthy female athletes, the repolarization changes are similar to the males'. The PR interval and QRS duration are shorter in women. There is also a higher prevalence of TWI in leads V1-V3, but the prevalence of LV hypertrophy is lower in females than in males [10,11]. However, more studies on female athletes, both white and black, are needed. The number of females participating in endurance sports is increasing and it is of importance to understand the impact of exercise on the female heart.

Declarations of interest

The authors declare no conflicts of interest.

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References

1. Sharma S, Whyte G, Elliott P, et al. Electrocardiographic changes in 1000 highly trained junior elite athletes. *Br J Sports Med* 1999;33: 319-24. doi: 10.1136/bjism.33.5.319
2. Papadakis M, Basavarajaiah S, Rawlins J, et al. Prevalence and significance of T-wave inversions in predominantly Caucasian adolescent athletes. *Eur Heart J* 2009;30:1728-35. doi: 10.1093/eurheartj/ehp164.
3. Papadakis M, Carre F, Kervio G, et al. The prevalence, distribution, and clinical outcomes of electrocardiographic repolarization patterns in male athletes of African/Afro-Caribbean origin. *Eur Heart J* 2011;32:2304-13. doi: 10.1093/eurheartj/ehr140.
4. Di Paolo FM, Schmied C, Zerguini YA, et al. The athlete's heart in adolescent Africans: an electrocardiographic and echocardiographic study. *J Am Coll Cardiol* 2012;59(11):1029-36. doi: 10.1016/j.jacc.2011.12.008.
5. Brosnan M, La Gerche A, Kalman J, et al. Comparison of frequency of significant electrocardiographic abnormalities in endurance versus nonendurance athletes. *Am J Cardiol* 2014;113(9):1567-73. doi: 10.1016/j.amjcard.2014.01.438.
6. Sheikh N, Papadakis M, Carre F, et al. Cardiac adaptation to exercise in adolescent athletes of African ethnicity: an emergent elite athletic population. *Br J Sports Med* 2013;47(9):585-92. doi: 10.1136/bjsports-2012-091874.
7. Noseworthy PA, Weiner R, Kim J, et al. Early repolarization pattern in competitive athletes: clinical correlates and the effects of exercise training. *Circ Arrhythm Electrophysiol* 2011;4(4):432-40. doi: 10.1161/CIRCEP.111.962852.
8. Barry J, Maron, MD, Tammy S. Haas, RN, Aneesha Ahluwalia, Caleb J. Murphy, BS, and Ross F. Garberich, MSc. Demographics and epidemiology of sudden deaths in young competitive athletes: From the U.S. national registry. Hypertrophic Cardiomyopathy Center. Minneapolis Heart Institute Foundation. *Am J Med.* 2016;129(11):1170-1177. doi: 10.1016/j.amjmed.2016.02.031.
9. Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. *N. Engl. J. Med.* 1991;324(5), 295-301. doi: 10.1056/NEJM199101313240504
10. Storstein L, Björnstad H, Hals O, Meen HD. Electrocardiographic findings according to sex in athletes and controls. *Cardiology* 1991;79(3), 227-236. doi: 10.1159/000175017
11. Malhotra A, Dhutia H, Gati S, Dores H, Millar L, Merghani R, Merghani A, Walker M, Papadakis M, Sharma S. Prevalence and significance of anterior T wave inversion in females. (Clinical Abstract 103) *Heart* 2014;100 (Suppl. 3), A60-A60. doi: 10.1136/heartjnl-2014-306118.103
12. Pelliccia A, Maron BJ, Culasso F, Spataro A, Caselli G. Athlete's heart in women. Echocardiographic characterization of highly trained elite female athletes. *JAMA* 1996;276(3), 211-215. doi: 10.1001/jama.1996.03540030045030
13. Whyte GP, George K, Nevill A, Shave R, Sharma S, McKenna WJ. Left ventricular morphology and function in female athletes: a meta-analysis. *Int J Sports Med* 2004;25(5), 380-3. DOI 10.1055/s-2004-817827
14. International Olympic Committee. Factsheet: Women in the Olympic Movement – Update May 2014. International Olympic Committee, Lausanne, Switzerland (2014).
15. Calore C, Zorzi A, Sheikh N, Nese A, Facci M, Malhotra A, Zaidi A, Schiavon M, Pelliccia A, Sharma S, Corrado D. Electrocardiographic anterior T-wave inversion in athletes of different ethnicities: differential diagnosis between athlete's heart and cardiomyopathy. *Eur Heart J* 2016;37(32):2515-27. doi: 10.1093/eurheartj/ehv591.
16. Lakdawala NK, Thune JJ, Maron BJ, Cirino AL, Havndrup O, Bundgaard H, Christiansen M, Carlsen CM, Dorbval JF, Kwong RY, Colan SD, Køber LV, Ho CY. Electrocardiographic features of sarcomere mutation carriers



- with and without clinically overt hypertrophic cardiomyopathy. *Am J Cardiol* 2011;108(11):1606-13. doi: 10.1016/j.amjcard.2011.07.019.
17. Le VV, Wheeler MT, Mandic S, Dewey F, Fonda H, Perez M, Sungar G, Garza D, Ashley EA, Matheson G, Froelicher V. Addition of the electrocardiogram to the preparticipation examination of college athletes. *Clin J Sport Med* 2010;20(2): 98-105. doi: 10.1097/JSM.0b013e3181d44705.
 18. Marek J, Bufalino V, Bufalino V, Davis J, Marek K, Gami A, Stephan W, Zimmerman F. Feasibility and findings of large-scale electrocardiographic screening in young adults: data from 32561 subjects. *Heart Rhythm* 2011;8(10): 1555-9. doi: 10.1016/j.hrthm.2011.04.024.
 19. Olivetto I, Maron MS, Adabag AS, Casey SA, Vargiu D, Link MS, Udelson JE, Cecchi F, Maron BJ. Gender-related differences in the clinical presentation and outcome of hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2005;46(3), 480-487. doi: 10.1016/j.jacc.2005.04.043
 20. Maron BJ, Carney KP, Lever HM, Lewis JF, Barac I, Casey SA, Sherrid MV. Relationship of race to sudden cardiac death in competitive athletes with hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2003;4(6):974-980. doi: 10.1016/S0735-1097(02)02976-5
 21. Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. *Circulation* 2009;119(8), 1085-1092. doi:10.1161/CIRCULATIONAHA.108.804617.
 22. Maron BJ. Sudden death in young athletes. *N Engl J Med* 2003;349:1064-1075. doi: 10.1056/NEJMra022783
 23. Maron BJ, Shirani J, Poliac LC, Mathenge R, Roberts WC, Mueller FO. Sudden death in young competitive athletes: Clinical, demographic and pathological profiles. *JAMA* 1996;276(3):199-204. doi:10.1001/jama.1996.03540030033028
 24. Maron BJ. Historical perspectives on sudden deaths in young athletes with evolution over 35 years. *Am J Cardiol* 2015;116(9):1462-1468. doi: 10.1016/j.amjcard.2015.07.072.
 25. Corrado D1, Pelliccia A, Heidbuchel H, Sharma S, Link M, Basso C, Biffi A, Buja G, Delise P, Gussac I, Anastasakis A, Borjesson M, Björnstad HH, Carré F, Deligiannis A, Dugmore D, Fagard R, Hoogsteen J, Mellwig KP, Panhuyzen-Goedkoop N, Solberg E, Vanhees L, Drezner J, Estes NA 3rd, Iliceto S, Maron BJ, Peidro R, Schwartz PJ, Stein R, Thiene G, Zeppilli P, McKenna WJ; Section of Sports Cardiology, European Association of Cardiovascular Prevention and Rehabilitation. Recommendations for interpretation of 12-lead electrocardiogram in the athlete. *European Heart Journal* 2010; 31(2), 243-259. doi: 10.1093/eurheartj/ehp473.
 26. Pelliccia A, Maron BJ, Culasso F, Di Paolo FM, Spataro A, Biffi A, Caselli G, Piovano P. Clinical significance of abnormal electrocardiographic patterns in trained athletes. *Circulation* 2000;102(3): 278-84.
 27. Drezner JA, Fischbach P, Froelicher V, Marek J, Pelliccia A, Prutkin JM, Schmied CM, Sharma S, Wilson MG, Ackerman MJ, Anderson J, Ashley E, Asplund CA, Baggish AL, Börjesson M, Cannon BC, Corrado D, DiFiori JP, Harmon KG, Heidbuchel H, Owens DS, Paul S, Salerno JC, Stein R, Vetter VL. Normal electrocardiographic findings: recognizing physiological adaptations in athletes. *Br J Sports Med* 2013;47(3):125-36. doi: 10.1136/bjsports-2012-092068
 28. Drezner JA, Ackerman MJ, Cannon BC, Corrado D, Heidbuchel H, Prutkin JM, Salerno JC, Anderson J, Ashley E, Asplund CA, Baggish AL, Börjesson M, DiFiori JP, Fischbach P, Froelicher V, Harmon KG, Marek J, Owens DS, Paul S, Pelliccia A, Schmied CM, Sharma S, Stein R, Vetter VL, Wilson MG. Abnormal electrocardiographic findings in athletes: recognizing changes suggestive of primary electrical disease. *Br J Sports Med* 2013;47(3):153-75. doi: 10.1136/bjsports-2012-092070
 29. Drezner JA, Ashley E, Baggish AL, Börjesson M, Corrado D, Owens DS, Patel A, Pelliccia A, Vetter VL, Ackerman MJ, Anderson J, Asplund CA, Cannon BC, DiFiori J, Fischbach P, Froelicher V, Harmon KG, Heidbuchel H, Marek J, Paul S, Prutkin JM, Salerno JC, Schmied CM, Sharma S, Stein R, Wilson M. Abnormal electrocardiographic findings in athletes: recognizing changes suggestive of cardiomyopathy. *Br J Sports Med* 2013;47(3):137-52. doi: 10.1136/bjsports-2012-092069.
 30. Sheikh N, Papadakis M, Ghani S, Ghani S, Zaidi A, Gati S, Adami PE, Carré F, Schnell F, Wilson M, Avila P, McKenna W, Sharma S. Comparison of electrocardiographic criteria for the detection of cardiac abnormalities in elite black and white athletes. *Circulation* 2014; 129(16):1637-49. doi: 10.1161/CIRCULATIONAHA.113.006179.
 31. Malhotra A, Walker M, Dhutia H. ECG interpretation in the athlete: a comparison of ethnic groups when three different criteria are applied. (Abstract 416) *Eur J Prev Cardiol* 2015;22:84-5.
 32. Schnell F, Riding N, O'Hanlon R, Axel Lentz P, Donal E, Kervio G, Matelot D, Leurent G, Doutreleau S, Chevalier L, Guerard S, Wilson MG, Carré F. The recognition and significance of pathological T-wave inversions in athletes. *Circulation* 2014;131(2):165-73. doi: 10.1161/CIRCULATIONAHA.114.011038.
 33. Wilson MG, Sharma S, Carré F, Charron P, Richard P, O'Hanlon R, Prasad SK, Heidbuchel H, Brugada J, Salah O, Sheppard M, George KP, Whyte G, Hamilton B, Chalabi H. Significance of deep T-wave inversions in asymptomatic athletes with normal cardiovascular examinations: practical solutions of managing the diagnostic conundrum. *Br J Sports Med* 2012;46(suppl 1):i51-8. doi: 10.1136/bjsports-2011-090838
 34. Shewan LG, Coats AJS, Henein M. Requirements for ethical publishing in biomedical journals. *International Cardiovascular Forum Journal* 2015;2:2 DOI: 10.17987/icfj.V2i1.4