



LJMU Research Online

Morrison, B, Mohammad, A, Oxborough, D, Somauroo, J, Lindsay, S, Drane, AL, Shave, R and George, KP

The 12-lead electrocardiogram of the elite female footballer as defined by different interpretation criteria across the competitive season.

<http://researchonline.ljmu.ac.uk/id/eprint/15379/>

Article

Citation (please note it is advisable to refer to the publisher's version if you intend to cite from this work)

Morrison, B, Mohammad, A, Oxborough, D, Somauroo, J, Lindsay, S, Drane, AL, Shave, R and George, KP (2021) The 12-lead electrocardiogram of the elite female footballer as defined by different interpretation criteria across the competitive season. European Journal of Sport Science. ISSN 1536-

LJMU has developed [LJMU Research Online](#) for users to access the research output of the University more effectively. Copyright © and Moral Rights for the papers on this site are retained by the individual authors and/or other copyright owners. Users may download and/or print one copy of any article(s) in LJMU Research Online to facilitate their private study or for non-commercial research. You may not engage in further distribution of the material or use it for any profit-making activities or any commercial gain.

The version presented here may differ from the published version or from the version of the record. Please see the repository URL above for details on accessing the published version and note that access may require a subscription.

For more information please contact researchonline@ljmu.ac.uk

<http://researchonline.ljmu.ac.uk/>

The 12-lead electrocardiogram of the elite female footballer as defined by different interpretation criteria across the competitive season

Barbara Morrison MSc^{1,2}, Aleah Mohammad (Mphil)², David Oxborough PhD², John Somauroo BMedSci², Sara Lindsay (MD)², Aimee L Drane (PhD)³, Rob Shave PhD⁴, Keith George PhD²

¹SportsCardiologyBC, University of British Columbia, Vancouver, British Columbia, Canada

²Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Liverpool, United Kingdom.

³Cardiff School of Sport and Health, Cardiff Metropolitan University, Cardiff, UK

⁴School of Health and Exercise Sciences, University of British Columbia, Okanagan, Canada

ORCID iD

B. Morrison - <https://orcid.org/0000-0003-1648-4495>

K. George - <https://orcid.org/0000-0002-5119-6651>

D. Oxborough - <https://orcid.org/0000-0002-1334-3286>

A.Drane - <https://orcid.org/0000-0002-5208-917X>

Address for Correspondence:

Professor Keith George
Liverpool John Moores University
Egerton Court,
2 Rodney Street,
Liverpool,
L1 2UA

Email: K.George@ljmu.ac.uk

Tel: 01512313366

Abstract

PURPOSE: Pre-participation screening (PPS), using a 12-lead electrocardiogram (ECG), is recommended to identify athletes at risk of sudden cardiac death. ECG interpretation criteria have been developed to address the concern arising from high false-positives in athletes. There are limited ECG data in elite female footballers. The aims of this study were to 1) compare the ECG outcomes using three published ECG criteria (European Society of Cardiology [ESC], Seattle, International) in elite female footballers and 2) compare ECG data at three different stages of a competitive season.

METHODS: Eighty-one elite female footballers (21±4 yr) completed a medical assessment, anthropometrics, resting blood pressure and a resting 12-lead ECG. Each 12-lead ECG was interpreted in accordance with 1) ESC; 2) Seattle; 3) International Criteria to determine training-related and non-training related ECG changes. A subset of thirteen (26±4 yr) footballers had repeated resting ECG tests at three time points across the competitive season.

RESULTS: Eighty percent of females had training-related ECG patterns. Sinus bradycardia (65%) and early repolarization (42%) were the most common. Using the ESC Criteria 25% (20/81) of the athletes were considered to have an abnormal ECG, compared to 0% using the Seattle and International Criteria, mainly due to alterations in QT length criteria. There were no clinically significant differences in ECG data across a competitive season.

CONCLUSION: The Seattle and International ECG Criteria significantly reduced the number of ECG false-positives in elite female footballers and the time point of PPS within a competitive season is unlikely to alter the PPS outcome.

Keywords: Electrocardiogram, athletes, female, pre-participation screening, sudden cardiac death

Abbreviations

AMSSM	American Medical Society for Sports Medicine
ANOVA	Analysis of Variance
BSA	Body Surface Area
ECG	Electrocardiogram

ESC	European Society of Cardiology
FA	Football Association
FIFA	The Fédération Internationale de Football Association (FIFA)
F-MARC	FIFA Medical Assessment and Research Centre
LAE	Left atrial enlargement
LVH	Left ventricular hypertrophy
PPS	Pre-participation screening
SCD	Sudden cardiac death

INTRODUCTION

A growing number of global sporting governing bodies, including The Fédération Internationale de Football Association (FIFA) and the English FA (Football Association) mandate cardiac pre-participation screening (PPS) for the early identification of young athletes at risk of exercise-related sudden cardiac death (SCD).^{1, 2} Despite widespread recognition of the value of PPS there is concern regarding the potential rate of false-positive ECG outcomes.³ Errors can occur when differentiating between physiological and pathological ECG changes, that may cause unnecessary referrals and anxiety in athletes as they undergo additional, expensive and time-consuming diagnostic tests. In some instances, athletes may even be disqualified from competition.⁴

In an effort to address these concerns, the European Society of Cardiology (ESC) provided recommendations specific to the interpretation of ECG in athletes.⁵ The ESC recommendations divided findings into 2 groups: 1) common and training-related ECG changes that do not require follow-up; and 2) uncommon and training-unrelated ECG changes that require follow-up. The reclassification of ECGs into these two groups decreased the number of abnormal ECGs from 40% to 11%.^{6, 7} Subsequently, the American Medical Society for Sports Medicine (AMSSM), co-sponsored by the FIFA Medical Assessment and Research Centre (F-MARC), held a “summit” on ECG interpretation in athletes in Seattle, Washington to further revise the ECG recommendations and reduce the false-positive rate.^{4, 8} The ‘Seattle Criteria’ demonstrated favourable outcomes over the ESC Criteria with a reduction in the number of ECGs previously considered abnormal from 17% to 4% in a predominately male group (82%), while still identifying all athletes with cardiac pathology.⁹ Despite the Seattle Criteria modifications, the burden of false-positives was still a concern and on-going research has

helped to refine the criteria relative to ethnicity, age and isolated indices of chamber enlargement^{3, 10, 11, 12-17} allowing the development of the International Recommendations for Interpretation of the 12-lead ECG in Athletes.¹⁸

Women's participation in competitive sport has increased significantly over the past few years however studies assessing gender differences in cardiac adaptation have been scarce. Of those studies that have focussed on the female athlete it is apparent that the nature of adaptation is gender specific with less left ventricular hypertrophy (LVH) and reduced magnitude of right sided and atrial enlargement in female athletes.^{16, 19, 20} Our understanding of ECG adaptation in female athletes is even less developed with fewer studies focusing on gender-specific electrical remodelling.^{3, 7, 21-24} A possible explanation for these inequalities may be driven by female athletes' lower risk of SCD compared to their male counterparts.²⁵⁻²⁷ That aside, FIFA and the FA still recommend that female footballers undergo PPS with a 12-lead ECG used as the primary screening tool. In view of the differences in cardiac adaptation between genders and the lower incidence of SCD it is possible that current criteria may put female athletes more at risk of false-positive findings, generating unnecessary anxiety and onward referral costs. Two studies comparing the ESC, Seattle and International Criteria included females (18-20%) in the total athlete cohort but did not report sex-specific differences.^{9, 28} It is apparent therefore that clarity is required to demonstrate the impact of these criteria on the female athlete ECG.

The effect of seasonal variance in training load on PPS outcomes, and its associated tests (ECG), have received little attention in female athletes. Athletes with high training volumes have demonstrated an increase in left ventricular mass, volume and wall thickness with increased training load.²⁹⁻³¹ Whether variations in training exposure cause alterations in the

athlete's ECG is not clear and could have implications for PPS outcomes. To date, there is no data describing ECG changes across a competitive season in elite female footballers. Within female football there is currently no mandate for the specific timing of PPS and annual screening and/or signing medicals can occur at any point across the competitive season. Based on this, an understanding of the impact of training load and seasonal adaptation on the 12-lead ECG is important for correct interpretation and to further reduce the risk of false-positive findings.

The aims of this study were to 1) compare the ECG outcomes using three published criteria (ESC, Seattle and International) in elite female footballers, and 2) assess 12-lead ECG parameters in elite female footballers at three different stages of a competitive season.

METHODS

Participants

Elite female footballers from three British professional football clubs were included in the study. Players were excluded if they had; known cardiovascular disease, current symptoms suggestive of cardiovascular disease, family history of cardiovascular disease in first degree relative (<50 years), current illness, current cardiovascular medication intake, and an injury within the previous month that lasted greater than seven days that disrupted training and competition.

All participants refrained from exercise training or recreational activity for at least six hours prior to each data collection session. Alcohol and caffeine consumption were restricted for the

12 hours prior to testing. The study was approved by Liverpool John Moores University Ethics Committee with all participants providing written informed consent.

Study Design

All players presented either at the Cardiovascular Laboratories at Liverpool John Moores University or at their club facility for a single session as part of their annual cardiac pre-participation screening. A subset of these players underwent additional testing at three time points within the competitive season. In the sub-study, every effort was made to schedule repeat tests at the same time of day.

Participant Evaluation

All players completed the FA pre-participation medical assessment form (personal symptoms and family history of cardiovascular disease and SCD) and underwent a standard anthropometric assessment including height (Seca 217, Hannover, Germany), body mass (Seca supra 719, Hannover, Germany) and calculated body surface area.³² After five minutes of seated rest brachial artery blood pressure was measured, a minimum of two times, using an automatic sphygmomanometer (Dinamap 300, GE Medical systems, USA). A standard 12-lead ECG was performed by a trained cardiac physiologist using commercially available equipment (CardioExpress SL6, Spacelabs Healthcare, Washington US). All athletes underwent echocardiography excluding the presence of any structural or functional anomaly.

ECG assessment criteria

Each 12-lead ECG was interpreted in accordance with 1) 2010 ESC; 2) Seattle and; 3) International Criteria to determine training-related and non-training related ECG changes (Supplementary Material).^{5, 8, 18} Electrocardiographic parameters including heart rate, P wave duration, PR interval, QRS duration, corrected (Bazett) QTc duration, QRS axis, and voltage criteria for left and right ventricular hypertrophy were reported and defined relative to normative values³³ as well as whether specific ECG criteria were met.

ECG evaluation across a competitive season

Participants' ECGs were interpreted using the International Criteria¹⁸ at the following time points; 1) start of pre-season training (PRE), after an off-season period, 2) after half of the competitive league games were completed (MID), and 3) at the end of the competitive season (END). The timeline and associated training exposure is detailed in Figure 1.

There was no structured training during the four weeks prior to the PRE testing point and they trained *ad libitum*. During the competitive season, athletes underwent five 60-minute field sessions, two 90- minute strength and conditioning sessions, and one competitive match per week. This weekly exposure did not change significantly between MID and END, however, END represented an accumulation of more training/game exposure. Match exposure differed across participants due to selection and/or substitution into games. Those not selected to play in any given fixture performed an extra training (field) session lasting up to 60 minutes.

Statistical Analysis

Continuous variables were reported as mean \pm SD. Frequency tables were generated for all categorical data and reported as number of participants (n) and percentage (%). The three ECG criteria were compared by frequency of occurrence of specific patterns and the false positive rate. In the sub-study, continuous ECG data were analysed using a repeated ANOVA with post-hoc Bonferroni assessment. Statistical analyses were performed using commercially available software package SPSS Version 23.0 for Windows (SPSS, Illinois, USA). A $P < 0.05$ was considered statistically significant.

RESULTS

Subject characteristics

Eighty-one elite female football players (21 ± 4 yr) completed testing (Table 1). The majority of athletes were Caucasian (n=77; Black African- Caribbean, n=1; mixed, n=2; Asian British, n=1). Athletes had participated in football activities for a total of 14 ± 4 years. Thirteen athletes (26 ± 4 y; Caucasian n=13) participated in the sub-study. There were no significant differences in weight, BSA, systolic blood pressure across the testing sessions ($P > 0.05$) (Table 1). Diastolic blood pressure was significantly lower at PRE compared to MID and END time points ($P < 0.01$).

ECG assessment criteria

All ECG parameters were within normal ranges except resting heart rate (lower than normative values) (Table 2). A total of 65 (80%) athletes displayed training-related ECG patterns. The most common training-related patterns were sinus bradycardia (65%), early repolarisation (42%), and/or sinus arrhythmia (15%) (Table 3). Approximately 25% (20/81) of the athletes

were considered to have an abnormal ECG according to the ESC recommendations (Table 4). The ECG abnormalities were long QTc Interval (2.5%), short QTc Interval (16%) and intraventricular conduction delay (1.2%), right ventricular hypertrophy (2.5%) and T wave inversion (2.5%). In comparison to the ESC recommendations, both the Seattle Criteria and International Criteria reduced the number of abnormal ECG from 25% to 0% (Table 4).

ECG evaluation across a competitive season

All ECG parameters were within normal ranges except resting heart rate (lower than normative values). There were no significant differences in ECG patterns across testing sessions ($P > 0.05$; Table 2). Early repolarisation pattern (85% across all time points) and sinus bradycardia (69%, 77% and 54%, at PRE, MID and END, respectively) were the most common training-related ECG patterns. First degree atrioventricular block was observed in one athlete at both PRE and END assessments. One athlete demonstrated a single borderline ECG finding (right axis deviation) observed only at the END testing session. All remaining ECG recordings were considered normal across all seasonal time points.

DISCUSSION

The key findings from the current study were: 1) 25% of elite female footballers presented with a likely training-unrelated ECG according to the ESC criteria, that would require follow-up, whilst the Seattle and International Criteria reduced the false-positive rate to 0%; 2) there were no significant changes in the ECG of elite female footballers assessed across a competitive season.

The ECG of the Female Footballer

There is a scarcity of data pertaining to ECG findings in female athletes and specifically in female footballers. In those studies that have separately analysed genders it is apparent that female athletes present different criteria to their male counterparts.^{20, 21} Female athletes present with a lower prevalence of left atrial enlargement (LAE) and LVH of which is consistent with the findings from the current study. When considering gender-specific cardiac adaptation, it is likely that these findings are in-fitting with the lower absolute values of cardiac remodelling seen in female athletes.³⁴

T-wave inversion constrained to the anterior leads has also been reported to be more prevalent in the female athlete. Malhotra et al demonstrated that 6.5% of female athletes had anterior T-wave inversion compared to 2% of male athletes.²² This can be further complicated with an increased prevalence in females of black ethnicity.¹⁹ Interestingly, we were unable to reproduce these findings in the female footballer with only 2.5% presenting with anterior T-wave inversion. It is difficult to fully elucidate this finding, but it is important to acknowledge that this study is predominantly based on a white Caucasian population and there is an equally low prevalence of other criteria that is more frequently found in black female athletes such as LAE and significant ST elevation.¹⁹

It is also apparent that of those few ECG studies that have reported female athletes have generally categorised them based on sporting disciplines. Football is a team sport, defined as being based upon low static and high dynamic workloads.³⁵ The heart and its chambers are subject to this unique training stimulus and, therefore, it is not unreasonable to expect differences in electrical remodelling when compared to other sporting disciplines and

generalised broad classifications. A recent study clearly highlights that those athletes with high intensity components to their sport are more likely to have an abnormal ECG.³⁶ In corroboration, data has been presented on ECG findings in female collegiate athletes as per sporting classification.³⁷ Those athletes with high dynamic activity presented with the most pronounced changes with 60 athletes of low static / high dynamic sporting discipline having specific adaptation with increased QTc compared to other sporting classifications. Although the athletes in the current study did not demonstrate similar abnormal findings it is clearly relevant that an individualised assessment of the athlete based on their demographics should be undertaken when making any interpretation.

In the UK, female football is receiving more attention, greater television audiences and wider awareness and hence there is increased frequency for players to have professional contracts and more structured training regimes. This is bound to impact on the nature and magnitude of cardiac adaptation, particularly over the forthcoming years, where the longevity of high levels of training volume will directly influence electrical remodelling and reciprocal ECG changes.^{36,38} We can speculate that the low prevalence of female specific adaptation in the current study reflects the lower life-long volumes of training in this sport. That aside, based on this ongoing transition and evolution of the game, it is important to provide current criteria for this specific demographic particularly in view of mandated screening from the professional sporting bodies including FIFA and the FA.

Comparison of ECG criteria

Similar to previous studies in predominantly male athletes,^{3, 9, 23, 24, 33} the ESC criteria elicited a higher false-positive rate compared to the Seattle and International Criteria in elite female

footballers. Specifically, the refining of the long QTc interval from >440 ms in men and >460 ms in women (ESC) to >470 ms (men) and 480 ms (women) in the Seattle and International Criteria reduced the prevalence of cases from 2.5% to 0%. Further, the 13 (10.5%) athletes with a short QTc (<340 ms; ESC criteria) was reduced to 0% using Seattle and International Criteria (<320 ms). Similarly, the one athlete that displayed intraventricular conduction delay using the ESC Criteria (>110 ms) was not highlighted by the Seattle or International Criteria (>140 ms). A single athlete (1.2%) presented with right ventricular hypertrophy using the ESC Criteria but this occurred in the absence of right axis deviation so was not considered a positive outcome using the Seattle and International Criteria (0%). One (1.2%) athlete presented with a T wave inversion using the ESC Criteria. The T-wave inversion in this athlete did not meet the requirements using the Seattle or International Criteria because the T-wave inversion was present in leads V1 and V2 only. The present study confirms that International and Seattle ECG Criteria improves the false-positive rate in elite female footballers.

The present study did not identify any training-unrelated (abnormal) ECG changes when using the Seattle and International Criteria. Despite seeing a lower false-positive rate when Seattle and International Criteria were compared to ESC in male athletes,³ there were still abnormal ECG patterns in 8.5% and 2.1% Caucasian males, respectively. The absence of abnormal ECG changes in a group of elite female athletes with no overt cardiovascular disease is also congruent with the lower SCD risk observed in female athletes.

The present study supports previous data that training-related ECG changes are common in elite female endurance and team sports athletes'.^{24, 39, 40} Eighty percent of female footballers displayed training-related ECG changes with sinus bradycardia (65%) and early repolarisation

(42%) pattern being the most common. The presence of sinus bradycardia is likely secondary to enhanced vagal tone as a result of endurance training.⁴⁰ Similarly, exercise training has been demonstrated to lead to an increased prevalence of early repolarization although this was not associated with evidence of structural myocardial remodelling.⁴¹

ECG evaluation across a competitive season

There were minimal changes in ECG data and criteria across a competitive season in elite female footballers. This suggests that the training-cycle related changes in exercise frequency, intensity and volume do not represent a large enough physiological stimuli to mediate changes in the ECG of elite female footballers. Conversely, Dawkins et al., reported that in untrained individuals who undertook a rigorous training program, an increase of training-related ECG patterns is observed.²⁸ This is likely due to a drastic change in their exercise frequency, intensity and volume, confirming our suggestion that ECG changes do not occur when exercise levels remain relatively stable. There was no presentation of training-unrelated ECG patterns at any time point in the present study. In contrast, Oxborough et al. demonstrated ECG changes in response to an acute increase in training intensity and volume (increasing from an average of 10 hours per week to 32).⁴² In this case, a boxer demonstrated T wave inversion after the first week of intense physical training but reverted back to normal in week two and for the remainder of the training period. The current data suggest that performing ECGs at different time points in female footballers with a relatively stable training volume should not alter PPS outcomes.

Study limitations

The present study provides novel ECG data in elite female footballers but some limitations should be noted. The overall sample size is relatively small however it is important to acknowledge that this is a homogenous group of athletes of a unique and specific demographic and therefore represents one of the largest populations of elite female footballers in the current literature. In the main cohort, the timing of ECG assessment during the competitive season was not controlled for and this was part of the rationale for the sub-study. The sub-study would suggest this was unlikely to significantly alter the outcome of the first study. It is also important to note, however, that the sub-sample was relatively small (albeit in-fitting with other seasonal studies of this type) and changes in training exposure (intensity, duration and volume) were not sensitively assessed. A non-athletic comparison group may have been beneficial, however, the repeated measures study design provides a framework where each athlete acts as their own control. The present study did not control for menstrual cycle phase or oral contraceptive use at the time of PPS. The sample included an ethnically homogenous group of elite female footballers and the results cannot be extrapolated to other ethnic groups. Finally, this study was cross-sectional in design and hence the lack of any longitudinal follow-up renders it impossible to establish the long-term impact of a 0% false positive rate.

CONCLUSIONS

Both the Seattle and International Criteria reduced the number of ECG false-positives during PPS from 25% to 0% when compared to the ESC criteria in elite female footballers. This reduction in false-positives in elite female footballers is clinically important in minimising unnecessary referrals for further testing and it is reasonable to consider the use of the International Criteria in female athletes from other similar sporting disciplines. That aside, further work is required to establish validity across other diverse female athlete populations.

The time point of PPS within a competitive season in elite female footballers is unlikely to alter any relevant clinical outcome.

References

1. Corrado D, Pelliccia A, Bjornstad HH, et al. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *European heart journal* 2005;26:516-24.
2. Maron BJ, Hutter JAM, Krauss MD, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. *Circulation* 2007;115:1643-1655.
3. Riding NR, Salah O, Sharma S, et al. ECG and morphologic adaptations in Arabic athletes: are the European Society of Cardiology's recommendations for the interpretation of the 12-lead ECG appropriate for this ethnicity? *British journal of sports medicine* 2014;48:1138-1143.
4. Drezner JA, Asif IM, Owens DS, et al. Accuracy of ECG interpretation in competitive athletes: the impact of using standardised ECG criteria. *British journal of sports medicine* 2012;46:335-340.
5. Corrado D, Pelliccia A, Heidbuchel H, et al. Recommendations for interpretation of 12-lead electrocardiogram in the athlete. *European heart journal* 2010;31:243-59.
6. Pelliccia A, Maron BJ, Culasso F, et al. Clinical significance of abnormal electrocardiographic patterns in trained athletes. *Circulation* 2000;102:278-84.
7. Riding NR, Sheikh N, Adamuz C, et al. Comparison of three current sets of electrocardiographic interpretation criteria for use in screening athletes. *Heart* 2015;101:384-90.
8. Drezner JA, DiFiori JP, Fischbach P, et al. Electrocardiographic interpretation in athletes: the 'Seattle criteria'. *British journal of sports medicine* 2013;47:122.

9. Brosnan M, La Gerche A, Kalman J, et al. The Seattle Criteria increase the specificity of preparticipation ECG screening among elite athletes. *British journal of sports medicine* 2014;48:1144-50.
10. Gati S, Sheikh N, Ghani S, et al. Should axis deviation or atrial enlargement be categorised as abnormal in young athletes? The athlete's electrocardiogram: time for re-appraisal of markers of pathology. *European heart journal* 2013;34:3641-8.
11. Wilson MG, Chatard JC, Carre F, et al. Prevalence of electrocardiographic abnormalities in West-Asian and African male athletes. *British journal of sports medicine* 2012;46:341-347.
12. McClean G, Riding NR, Ardern CL, et al. Electrical and structural adaptations of the paediatric athlete's heart: a systematic review with meta-analysis. *British journal of sports medicine* 2018;52:230.
13. 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. *European heart journal* 2011;32:2304-13.
14. Calore C, Melacini P, Pelliccia A, et al. Prevalence and clinical meaning of isolated increase of QRS voltages in hypertrophic cardiomyopathy versus athlete's heart: relevance to athletic screening. *International journal of cardiology* 2013;168:4494-7.
15. Lakdawala NK, Thune JJ, Maron BJ, et al. Electrocardiographic features of sarcomere mutation carriers with and without clinically overt hypertrophic cardiomyopathy. *The American journal of cardiology* 2011;108:1606-13.
16. Zaidi A, Ghani S, Sheikh N, et al. Clinical significance of electrocardiographic right ventricular hypertrophy in athletes: comparison with arrhythmogenic right ventricular cardiomyopathy and pulmonary hypertension. *European heart journal* 2013;34:3649-56.
17. Zaidi A, Sheikh N, Jongman JK, et al. Clinical Differentiation Between Physiological Remodeling and Arrhythmogenic Right Ventricular Cardiomyopathy in Athletes With Marked Electrocardiographic Repolarization Anomalies. *Journal of the American College of Cardiology* 2015;65:2702-11.
18. Sharma S, Drezner JA, Baggish A, et al. International Recommendations for Electrocardiographic Interpretation in Athletes. *Journal of the American College of Cardiology* 2017;69:1057-1075.

19. Rawlins J, Carre F, Kervio G, et al. Ethnic differences in physiological cardiac adaptation to intense physical exercise in highly trained female athletes. *Circulation* 2010;121:1078-1085
20. Finocchiaro G, Dhutia H, D'Silva A, et al. Effect of sex and sporting discipline on LV adaptation to exercise. *JACC Cardiovasc Imaging*. 2017;10(9):965–7.
21. Storstein L, Bjornstad H, Hals O, et al. Electrocardiographic Findings according to Sex in Athletes and Controls. *Cardiology* 1991;79:227–236
22. Malhotra A, Dhutia H, Gati S, et al. Anterior T-wave inversion in young white athletes and nonathletes. *J Am Coll Cardiol*. 2017;69(1):1–9
23. Berge HM, Gjesdal K, Andersen TE, Solberg EE, Steine K. Prevalence of abnormal ECGs in male soccer players decreases with the Seattle criteria, but is still high. *Scandinavian journal of medicine & science in sports* 2015;25:501-8.
24. Bessem B, de Bruijn MC, Nieuwland W. The ECG of high-level junior soccer players: comparing the ESC vs. the Seattle criteria. *British journal of sports medicine* 2015;49:1000-6.
25. 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;119:1085-1092.
26. Finocchiaro G, Papadakis M, Robertus JL, et al. Etiology of Sudden Death in Sports: Insights From a United Kingdom Regional Registry. *Journal of the American College of Cardiology* 2016;67:2108-15.
27. Landry CH, Allan KS, Connelly KA, Cunningham K, Morrison LJ, Dorian P. Sudden Cardiac Arrest during Participation in Competitive Sports. *N Engl J Med*. 2017;377:1943-1953.
28. Dawkins TG, Shave RE, Baggish AL, et al. Electrocardiographic changes following six months of long-distance triathlon training in previously recreationally active individuals. *Eur J Sport Sci* 2020;20:4:553-562.
29. Weiner RB, Baggish AL. Accuracy of ECG-inclusive preparticipation screening in athletes: more work to be done. *Expert review of cardiovascular therapy* 2012;10:671-673
30. Cabanelas N, Freitas S, Gonçalves L. Morphological and functional changes in athletes' hearts during the competitive season. *Rev Port Cardiol* 2013;32:291-6.

31. D'Ascenzi F, Pelliccia A, Cameli M, et al. Dynamic changes in left ventricular mass and in fat-free mass in top-level athletes during the competitive season. *European journal of preventive cardiology* 2015;22:127-34.
32. Mosteller RD. Simplified calculation of body-surface area. *N Engl J Med.* 1987;317:1098.
33. Wagner GS, Strauss DG. *Marriot's Practical Electrocardiography.* Baltimore: Williams and Wilkins; 1994.
34. Foryst-Ludwig A, Kintscher U. Sex differences in exercise-induced cardiac hypertrophy. *Eur J Physiol.* 2013;465(5):731–7.
35. Mitchell JH, Haskell W, Snell P, et al. Task Force 8: classification of sports. *J Am Coll Cardiol* 2005;45:1364-7
36. Does H, Malhotra A, Sheikh N, et al. Abnormal electrocardiographic findings in athletes: Correlation with intensity of sport and level of competition. *Rev Port Cardiol* 2016 Nov;35(11):593-600
37. Rambarat C, Reifsteck F, Clugston J, et al. Preparticipation Cardiac Evaluation Findings in a Cohort of Collegiate Female Athletes. *Am J Cardiol* 2021;140:134-139
38. 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;113:1567-73
39. Sheikh N, Papadakis M, Ghani S, et al. Comparison of electrocardiographic criteria for the detection of cardiac abnormalities in elite black and white athletes. *Circulation* 2014;129:1637-49.
40. Sharma S, Whyte G, Elliott P, et al. Electrocardiographic changes in 1000 highly trained junior elite athletes. *British journal of sports medicine.* 1999;33:319-24.
41. Noseworthy PA, Weiner R, Kim J, et al. Early repolarization pattern in competitive athletes: clinical correlates and the effects of exercise training. *Circulation Arrhythmia and electrophysiology* 2011;4:432-440.

42. Oxborough D, George K, Utomi V, et al. Acute response and chronic stimulus for cardiac structural and functional adaptation in a professional boxer. *Oxf Med Case Reports* 2014;2014:65-68.

Table 1: Participant demographics for total population and three seasonal assessment points

	Total Population (n=81)	PRE (n=13)	MID (n=13)	END (n=13)
Age (years)	21±4 (15-39)	25±4 (19-33)	25±4 (19-33)	25±4 (19-33)
Height (cm)	170±10 (154-179)	170±6 (156-179)	170±6 (156-179)	170±6 (156-179)
Body mass (kg)	63±7 (52-88)	61±9 (43-77)	63±5 (51-75)	64±6 (54-75)
BSA (m²)	1.70±0.11 (1.51-1.94)	1.69±0.15 (1.43-1.92)	1.72±0.09 (1.53-1.90)	1.73±0.10 (1.53-1.90)
Systolic BP (mmHg)	117±13 (90-147)	109±12 (88-126)	117±11 (103-135)	123±13 (93-142)
Diastolic BP (mmHg)	67±8 (53-85)	60±7 * (48-71)	67±5 (55-73)	67±7 (48-74)
BSA- Body Surface Area; BP- Blood Pressure; PRE - Pre-season; MID- Mid-season, END- End season. *PRE lower than MID and END (P<0.01)				

Table 2: Continuous data for total population and at three seasonal assessment time points

	Total Population (n=81)	PRE (n=13)	MID (n=13)	END (n=13)	Normal population range²⁹
Heart rate (beats. min⁻¹)	57±10 (38-82)	54±7 (43-61)	53±10 (40-71)	57±9 (49-71)	60 to 100
P Duration (ms)	94 ± 9 (73-112)	100±8 (90-112)	99±10 (76-118)	99±19 (80-154)	<120
PR Interval (ms)	153±21 (115-206)	164±29 (128-186)	149±18 (120-178)	157±37 (118-266)	120 to 200
QRS Duration (ms)	90±10 (72-114)	87±6 (82-96)	89±6 (80-98)	89±6 (82-94)	80 to 110
QTc (Bazett) (ms)	396±20 (346-462)	420±22 (386-459)	407±27 (375-459)	420±24 (373-457)	350 to 440
QRS Axis (°)	69±20 (6-111)	66±12 (31-96)	67±17 (39-99)	47±51 (-45-129)	-30 to 120
Voltage: RV₁ + SV₅ (mv)	0.40±0.24 (0.1-1.2)	0.21±0.07 (0.1-0.3)	0.23±0.07 (0.1-0.3)	0.18±0.06 (0.1-0.3)	< 1.05
Voltage: SV₁ + RV₅ (mV)	2.5±0.7 (1.0-4.4)	2.0±0.6 (1.3.-2.9)	2.0±0.5 (1.4-3.0)	2.0±0.6 (1.1-3.2)	< 3.5

Table 3: Training and borderline - related ECG patterns

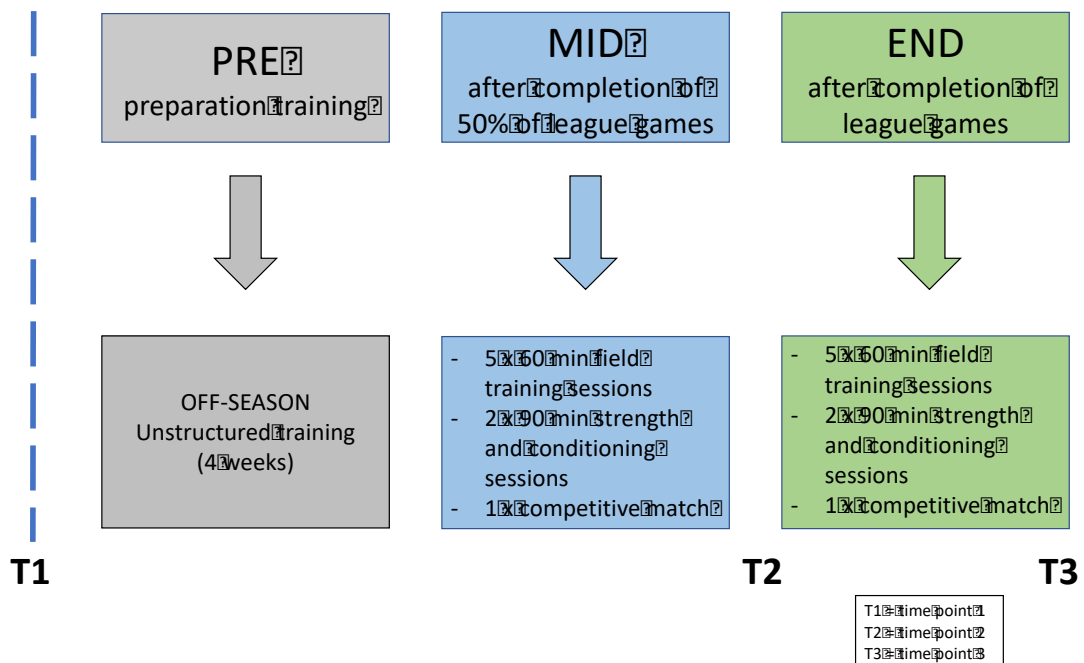
Training-Related ECG Changes	Entire population (n=81)	PRE (n=13)	MID (n=13)	END (n=13)
Increased QRS voltage for RVH	2 (2)	0	0	0
Increased QRS voltage for LVH	5 (6)	0	0	0
Incomplete right bundle branch block	7 (9)	0	0	0
Early repolarisation	34 (42)	11(85)	11(85)	11(85)
Black athlete repolarisation variant	0	0	0	0
Juvenile T wave pattern	0	0	0	0
Sinus arrhythmia	12 (15)	0	0	0
Sinus bradycardia	53 (65)	9 (69)	10 (77)	7(54)
Junctional rhythm	0	0	0	0
Ectopic atrial rhythm	0	0	0	0
1° atrioventricular block	1 (1)	1(8)	0	1(8)
Mobitz type 1 (Wenkebach) 2° atrioventricular	0	0	0	0
2 or more training-related changes	44 (54)	8 (62)	7 (54)	6 (46)
Borderline ECG Changes				
Right axis deviation	0	0	0	1 (8)
Left axis deviation	0	0	0	0
Left atrial enlargement	0	0	0	0
Right atrial enlargement	0	0	0	0
Complete right bundle branch block	0	0	0	0

LVH – left ventricular hypertrophy; RVH – right ventricular hypertrophy

Table 4: Absolute number of training-unrelated ECG changes using the ESC, Seattle and International Criteria

ECG ABNORMALITY	Players with Abnormality, n (%)		
	ESC	Seattle	International
Long QTc Interval	2 (2.5)	0	0
Short QTc Interval	13 (16.0)	0	0
Intraventricular conduction delay	1 (1.2)	0	0
Right ventricular hypertrophy	2 (2.5)	0	0
T wave Inversion	2 (2.5)	0	0
Overall false positives	20 (25.0)	0	0

Fig. 1 - Summary of testing time points



SUPPLEMENTARY MATERIAL

Comparison of ESC, Seattle and International ECG criteria for athlete pre-participation

screening

ECG abnormality	ESC Criteria	Seattle Criteria	International Criteria
Left atrial enlargement	Negative portion of the P-wave in lead V1 ≥ 0.1 mV in depth and ≥ 40 ms in duration	Prolonged P-wave of >120 ms in leads I or II with negative portion of the P-wave ≥ 0.1 mV and ≥ 40 ms in lead V1	As ESC
Right atrial enlargement	P-wave amplitude ≥ 2.5 mm in leads II, III or aVF	As ESC	As ESC
Left QRS-axis deviation	-30° to -90°	As ESC	As ESC
Right QRS-axis deviation	$>115^\circ$	$>120^\circ$	As ESC
Right ventricular hypertrophy	Sum of R-wave in V1 and S-wave in V5 or V6 ≥ 1.05 mV	Sum of R-wave in V1 and S-wave in V5 >1.05 mV and right axis deviation $>120^\circ$	As ESC
Corrected QT interval	>440 ms (men) and >460 ms (women)	>470 ms (men) and 480 ms (women)	As Seattle
Complete left bundle branch block	QRS ≥ 120 ms predominantly negative QRS complex in lead V1 (QS or rS), and upright monophasic R-wave in leads I and V6	As ESC	As ESC
Complete right bundle branch block	RSR pattern in anterior precordial leads with QRS duration ≥ 120 ms	Not relevant	As ESC
Intraventricular conduction delay	Any QRS duration >120 ms including RBBB and LBBB	Any QRS duration ≥ 140 ms or complete LBBB	As ESC
Pathological Q-waves	>0.4 mV deep in any lead except III, aVR	>0.3 mV deep and/or >40 ms duration in ≥ 2 leads except III and aVR	≥ 40 ms in duration or $\geq 25\%$ of the

			height of the ensuing R-wave
Significant T-wave inversion	≥ 2 mm in ≥ 2 adjacent leads (deep) or 'minor' in ≥ 2 leads	> 1 mm in depth in two or more leads V2–6, II and aVF or I and aVL (excludes III, aVR and V1)	As Seattle
ST-segment depression	≥ 0.5 mm deep in ≥ 2 leads	As ESC	As ESC
Ventricular pre-excitation	PR interval < 120 ms with or without delta wave	PR interval < 120 ms with delta wave	As Seattle