Comparison of ECG Criteria for the Detection of Cardiac Abnormalities in Elite Black and White Athletes

Running title: Sheikh et al.; Refining ECG Interpretation Criteria in Athletes

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Abstract

Background—Recent efforts have focused on improving specificity of the European Society of Cardiology (ESC) criteria for ECG interpretation in athletes. These criteria are derived predominantly from white athletes (WAs) and do not account for the effect of Afro-Caribbean ethnicity or novel research questioning the relevance of several isolated ECG patterns. We assessed the impact of the ESC criteria, newly published Seattle criteria and a group of proposed refined criteria in a large cohort of black athletes (BAs) and WAs.

Methods and Results—Between 2000-2012, 1208 BAs were evaluated with history, examination, 12-lead ECG and further investigations as appropriate. Electrocardiograms were retrospectively analysed according to the ESC recommendations, Seattle criteria, and proposed refined criteria which exclude several specific ECG patterns when present in isolation. All 3 criteria were also applied to 4297 WAs and 103 young athletes with hypertrophic cardiomyopathy (HCM). The ESC recommendations raised suspicion of a cardiac abnormality in 40.4% of BAs and 16.2% of WAs. The Seattle criteria reduced abnormal ECGs to 18.4% in BAs and 7.1% in WAs. The refined criteria further reduced abnormal ECGs to 11.5% in BAs and 5.3% in WAs. All 3 criteria identified 98.1% of athletes with HCM. Compared to ESC recommendations, the refined criteria improved specificity from 40.3% to 84.2% in BAs and from 73.8% to 94.1% in WAs without compromising the sensitivity of the ECG in detecting pathology.

Conclusions—Refinement of current ECG screening criteria has the potential to significantly reduce the burden of false-positive ECGs in athletes, particularly BAs.

Key words: screening, ethnicity, exercise, electrocardiography, cardiomyopathy, echocardiography, hypertrophy
Introduction

Pre-participation screening for early identification of young athletes at risk of exercise-related sudden cardiac death (SCD) is recommended by a growing number of sporting bodies and scientific organisations worldwide.\textsuperscript{1–3} Whereas evidence from Italy suggests that ECG-based screening is effective at detecting athletes with potentially serious cardiac disorders,\textsuperscript{4,5} there remain justifiable concerns relating to high false-positive rates arising from the overlap between physiological ECG patterns and those reflecting cardiac pathology.\textsuperscript{6–8}

The 2010 European Society of Cardiology (ESC) recommendations for ECG interpretation in athletes have attempted to facilitate the differentiation between physiological ECG patterns ("Group 1") and those indicative of cardiac disease ("Group 2").\textsuperscript{9} Although such categorisation has improved specificity,\textsuperscript{8,10} false-positive rates of between 10-20% have invariably prompted calls for further refinement.\textsuperscript{11} A recent collaboration between international experts culminated in the "Seattle criteria"\textsuperscript{12} which have improved specificity in some populations.\textsuperscript{13}

New data based on large athlete cohorts from our group has revealed several isolated ECG patterns to have a low diagnostic yield for cardiac disease, questioning their relevance as markers of pathology in elite athletes.\textsuperscript{14,15} Current guidelines in practice are consensus based and do not fully incorporate such scientific observations in their recommendations. Furthermore, they are derived almost exclusively from unselected Caucasian athletes (white athletes; WAs)\textsuperscript{16} and have not been evaluated in large cohorts of elite athletes of African/Afro-Caribbean origin (black athletes; BAs). The paucity of ECG interpretation criteria in BAs is of concern given that they most frequently exhibit profound ECG alterations,\textsuperscript{7,17–21} which overlap with primary cardiomyopathies and magnifies their risk of an erroneous diagnosis.
This study assessed the performance of the ESC and Seattle criteria in large cohorts of highly trained BAs and WAs compared to proposed "refined criteria" (Figure 1A) which incorporate new research findings and the effect of Afro-Caribbean ethnicity. In order to determine their sensitivity for the detection of hypertrophic cardiomyopathy (HCM), all 3 criteria were applied to a well characterised cohort of young, asymptomatic athletes with HCM.

Methods

Setting

The UK does not support a state sponsored cardiac screening program in athletes. However, the charitable organisation Cardiac Risk in the Young (CRY) has an established cardiac screening program for young individuals that also serves many professional sporting organisations including the English Institute of Sport, Lawn Tennis Association, Aviva Premiership Rugby and Football Association. Up to 1000 athletes from numerous regional or national sporting squads are assessed annually. Most preliminary evaluations, including ECG and echocardiography, are performed at training centres through a mobile investigations unit and supervised by the principal investigator (SS). Most athletes with abnormal findings are investigated further in a dedicated Inherited Cardiac Diseases and Sports Cardiology Unit at St. George’s Hospital.

Elite Athletes

Between 2000 and 2012, 1208 elite BAs and 4297 elite WAs aged 14-35 years were evaluated with a health questionnaire, cardiovascular examination, and 12-lead ECG. An athlete was considered “elite” if competing regularly at regional, national or international level and exercising for ≥6 hours per week. Ethnicity was self-assigned. Individuals with concerning symptoms, family history of cardiomyopathy or premature (≤40 years) SCD, a cardiac murmur
or an abnormal ECG were assessed with 2D-echocardiography and further investigations as necessary. A large proportion of elite athletes competing at national or international level underwent 2D-echocardiography as standard in accordance with the screening protocol required by their sporting organisations.

**Investigations in Elite Athletes**

**Electrocardiography**

Electrocardiography was performed using standard 12-lead positions using a GE Marquette Hellige (Milwaukee, USA) or Philips Pagewriter Trim III (Bothel, Washington) as described elsewhere.17

**Echocardiography**

Two-dimensional echocardiography was performed using either a GE Vivid I (Tirat, Israel), Philips Sonos 7500, Philips iE33 or Philips CPX50 (Bothel, Washington). Standard views were obtained and cavity and wall thickness measurements were performed using established guidelines.23 Pulsed Doppler recordings were performed at the distal margins of the mitral valve leaflets for early (E) and late (A) diastolic velocities. Tissue Doppler imaging (TDI) of septal and lateral mitral annular movement was recorded from the apical 4-chamber views to obtain early (E') and late (A') diastolic peak velocities.24 The ratio of transmitral flow velocity to the septal (E/E'sept) and lateral (E/E'lat) annular velocities were averaged (E/E'average) to provide an index of diastolic function.25 Left ventricular ejection fraction was calculated from LV volumes using Simpson’s rule.23

**Further investigations**

Requirement for further investigations was determined by symptomatic status, relevant family history, abnormal examination and results of the ECG and/or echocardiogram. These included a
maximal exercise tolerance test, 24-hour Holter monitor and cardiac magnetic resonance imaging scan (CMRI), as described previously.7

**ECG Interpretation**

The first comprehensive recommendations for interpretation of a young athlete’s ECG were published by the ESC in 2005 and modified in 2010 to improve specificity. In 2012, an international panel adjourned in Seattle to provide yet another revision to facilitate the development of universally accepted guidelines for ECG interpretation in young athletes that may also be applicable to BAs. Between 2000 and 2010, our own practice for ECG interpretation in athletes was similar to the 2010 ESC recommendations and was associated with an unacceptably high false-positive rate.

**Refined ECG criteria**

Between 2010 and 2012 we reassessed our practice of ECG interpretation in athletes more critically. Based on our longstanding experience of evaluating several thousand athletes and in line with recent consensus documents and research findings,7,12,14,15,17,19,20,26 we identified certain ECG anomalies (“Borderline Variants”; Figure 1A) currently included in the Group 2 category of the ESC recommendations and some deemed abnormal by the Seattle criteria which we would now consider as normal variants in asymptomatic athletes without a relevant family history or abnormal cardiac examination. Specifically, we would not recommend further investigation of athletes with any one of the following ECG patterns when present either in isolation or in association with recognised training related ECG changes: (1) left atrial enlargement (LAE); (2) right atrial enlargement (RAE); (3) left axis deviation (LAD); (4) right axis deviation (RAD); (5) Sokolow-Lyon voltage criteria for right ventricular hypertrophy (RVH). Based on our experience27 and in conjunction with the Bethesda guidelines28 and more recent Seattle criteria,12
we have increased the cut-off for an abnormal corrected QT interval (QTc) to 470 milliseconds (msec) in males and 480 msec in females. Finally, consistent with previous publications from our group,7,17,19,20 we no longer investigate asymptomatic BAs with T-wave inversion preceded by convex ST-segment elevation confined to V1-V4, a practice also adopted by the recent Seattle criteria.12 Conversely, the presence of 2 or more of these 6 patterns in combination or in association with other Group 2 ESC changes would be a requirement for further investigation. The refined criteria are illustrated in Figure 1A. Definitions of specific ECG patterns used in all 3 criteria are provided in Table 1.

**Retrospective ECG analysis**

The ECG of all 5505 elite athletes were analysed retrospectively using the ESC recommendations and Seattle criteria, with specific attention to the presence of abnormalities necessitating further investigation (ESC “Group 2 changes,”9 Figure 1B; or Seattle criteria “Abnormal ECG findings in athletes,”12 Figure 1C). We also applied the refined criteria retrospectively to our entire cohort of athletes. During ECG analysis, readers were blinded to pathological findings in all athletes.

**Athletes with Hypertrophic Cardiomyopathy**

We applied the ESC recommendations, Seattle criteria and refined criteria to a well characterised cohort of 103 consecutive young athletes with HCM assessed in 4 dedicated cardiomyopathy clinics in London (UK) and the French Institute of Health and Medical Research in Rennes (France). All individuals were aged between 14-35 years, asymptomatic, and exercised for a minimum of 4 hours per week at the time of presentation, enabling the performance of ECG criteria in identifying HCM to be assessed in a group comparable to that encountered during pre-participation evaluation. The initial 12-lead ECG obtained at the time of first evaluation was
used for analysis. Athletes were diagnosed with HCM after either: (1) investigation for abnormalities identified through pre-participation evaluation; (2) cascade screening of family members of an individual affected with HCM; (3) referral for a specialist opinion from another centre.

Hypertrophic cardiomyopathy was diagnosed on the basis of LVH ≥15mm in any myocardial segment, as assessed on echocardiography and/or CMRI, in the presence of a non-dilated left ventricle and the absence of another cardiac disorder or systemic condition capable of producing the same magnitude of LVH. In cases of mild LVH (<15mm), HCM was diagnosed in the context of a combination of features including: (1) electrocardiographic repolarization anomalies, specifically ST-segment depression or marked T-wave inversion; (2) unusual patterns of LVH; (3) presence of a small LV cavity; (4) identification of HCM in a first-degree relative and/or a positive gene test.

**Ethical Approval**

Ethical approval was granted by the National Research Ethics Service, Essex 2 Research Ethics Committee in the UK, the French Ministry of health and youth in France, and Shafallah Medical Genetic Centre in Qatar. Written consent was obtained from athletes aged ≥16 years and from a parent/guardian for those aged <16 years.

**Statistical Analysis**

Data were expressed as mean ± standard deviation or percentages as appropriate and analysed using SPSS software, version 20 (Chicago, IL, USA). Continuous variables were tested for normality using the Kolmogorov-Smirnov test. Group differences were tested using Student’s t-test or Mann-Whitney-U test for normally and non-normally distributed variables, respectively. Chi-squared test were used to compare the number of positive ECGs in BAs versus WAs within
each criteria, with significance defined as p<0.05 throughout. Positive agreement between the 3
criteria was determined using kappa and c-statistic.

The sensitivity and specificity for the screening process and 95% confidence intervals
were calculated from the athletic population who underwent history, examination, ECG and
echocardiography as standard using 2x2 contingency tables in GraphPad Prism software, version
6.01 (La Jolla, CA, USA). Sensitivity was defined as the ability to detect any cardiac disorder in
this cohort (serious or minor) based on the screening procedures performed (history,
examination, ECG and echocardiography). A serious cardiac disorder was defined as one that
has been implicated as a recognized cause of exercise-related SCD in young athletes. Specificity
was defined as the ability to correctly identify athletes without a cardiac disorder in this cohort
based on the screening procedures performed. Echocardiography was used as the gold-standard
test for detection or exclusion of structural disease. Negative and positive predictive values were
calculated based on the definitions above.

Results

Athlete Demographics

The majority of BAs and WAs were male (85.8% and 76.8% respectively). Overall, WAs were
younger than BAs (19.3±5.4 versus 22.2±5.7 years, p<0.001). White athletes and BAs had
similar body surface areas (1.92±0.26 versus 1.92±0.21m², p=0.7) and all had a blood pressure
of ≤140/90mmHg. Athletes competed in a total of 31 different sporting disciplines; the top 5
sports represented were soccer (26.2%), rugby (11.6%), athletics (11.1%), tennis (9.5%) and
swimming (6.5%). White athletes exercised for slightly more hours per week than BAs (16.3±7.5
versus 15.5±6.1 hours, p<0.001). Of the BAs, 56.4% were of West African origin, 26.5%
Caribbean, 14.9% North African, 4.8% East African, 4.6% mixed ethnicity, and 2.7% from the Americas.

**Characteristics of Athletes with Hypertrophic Cardiomyopathy**

The average age of athletes with HCM was 24.3±6.9 years (range 14-35 years) and the majority (94.2%) were male. Athletes with HCM exercised for an average of 9.7±5.1 hours per week. A significant percentage of the total HCM cohort (n=34, 33.0%) were African/Afro-Caribbean. Further characteristics of athletes with HCM are provided in Table 2.

Nine athletes (15.4%) with HCM showed concentric LVH and wall thicknesses <15mm, placing them in a diagnostic “grey zone” between athlete’s heart and HCM. Of these, 7 were Afro-Caribbean. All 9 exhibited deep T-wave inversion extending into the infero-lateral leads, 2 showed pathological Q-waves and 4 revealed resting ST-segment depression. The mean relative wall thickness in this group was 0.5 ± 0.08. Four athletes exhibited late gadolinium enhancement on CMRI and 3 had a family history of HCM and/or SCD.

**Analysis of ECGs**

**Application of the ESC Recommendations and Seattle criteria**

The number of ECGs deemed abnormal using the ESC recommendations and Seattle criteria are illustrated in Figure 2. Application of the ESC recommendations to our total athlete cohort resulted in 1183 (21.5%) athletes being designated to the abnormal category. Black athletes were 2.5-times more likely to exhibit an abnormal ECG compared to WAs (40.4% versus 16.2%, p<0.0001). The most prevalent ECG abnormalities in BAs were T-wave inversion (19.3%), RVH (10.7%) and LAE or RAE (13.8%) (Figure 3).

The Seattle criteria reduced the number of abnormal ECGs to 9.6% for the total athlete cohort. Using the Seattle criteria, BAs were 2.6-times more likely to exhibit an abnormal ECG
compared to WAs (18.4% versus 7.1%, p<0.0001; Figure 2).

**Refined criteria**

The refined criteria reduced the number of abnormal ECGs to 6.6% for the total athlete cohort. Compared to the ESC and Seattle criteria, the refined criteria were associated with a significant reduction in the number of abnormal ECGs in both BAs and WAs, to 11.5% and 5.3%, respectively (p<0.0001; Figure 2). Relative to the ESC recommendations, the refined criteria offered a 71.5% reduction in abnormal ECGs in BAs and 67.3% reduction in WAs. In absolute terms, this represented an almost 3-fold greater reduction in abnormal ECGs in BAs compared to WAs (28.9% versus 10.9%, respectively). Relative to the Seattle criteria, the refined criteria offered a further 37.5% reduction in abnormal ECGs in BAs and 25.4% reduction in WAs. Based on the refined criteria, the leading cause for an abnormal ECG in BAs remained T-wave inversion (Figure 3), in the inferior and lateral leads.

**Comparison of criteria for agreement**

Comparison of the 3 criteria for positive results in BAs and WAs revealed the strongest agreement between the Seattle and refined criteria, particularly in WAs (Table 3 and Figure 4). There was only fair to moderate agreement between the ESC recommendations and the refined and Seattle criteria for BAs and WAs respectively.

**Application of the ESC, Seattle and Refined criteria to Athletes with HCM**

All 3 ECG criteria detected all but 2 athletes with HCM (1.9%) on the basis of ECG alone. Both athletes exhibited a normal ECG. The first individual was diagnosed after routine echocardiography as part of his pre-participation evaluation and the second after family screening for HCM (Table 4).

None of the athletes with HCM exhibited isolated atrial enlargement, axis deviation or
RVH on their ECG. Similarly, none of the BAs with HCM showed isolated TWI in V1-V4.

Identification of Pathology

3210 athletes (58.3%) underwent echocardiography (2392 WAs [55.7%] and 818 BAs [67.7%]). Of these, 1183 (36.9%) had an ECG deemed abnormal (695 WAs and 488 BAs), 28 (0.9%) had symptoms, 24 (0.7%) had a cardiac murmur, and 20 (0.6%) had a significant family history. The remaining 1955 athletes (60.9%) underwent echocardiography, despite normal preliminary investigations, as a result of their club policy.

Of the 3210 athletes who underwent both ECG and echocardiography, 40 (1.25%) were diagnosed with a cardiac disorder. Specifically, 15 (0.47%) had a serious disorder: HCM (n=5), Wolff-Parkinson-White syndrome (n=5), long-QT syndrome (n=3), Brugada syndrome (n=1) and anomalous coronary artery origin (n=1); and 25 (0.78%) had a minor congenital/valvular abnormality: bicuspid aortic valve (n=10), mitral valve prolapse (n=7), atrial septal defect (n=3), ventricular septal defect (n=2), mild aortic regurgitation (n=1), mild pulmonary stenosis (n=1), and cor triatriatum (n=1) (Figure 5).

Fourteen of the 15 athletes (93.3%) with a potentially serious cardiac disorder (including all 5 cases of HCM) were identified with ECG and only 1 (6.7%) was detected on the basis of symptoms. Of the athletes with minor congenital abnormalities, 10 (40.0%) were identified on the basis of abnormal examination findings and 15 (60.0%) were detected on routine echocardiography in the setting of a normal history, examination and ECG.

In contrast with the ability to detect sinister disorders, the ECG alone failed to identify all 25 individuals with minor congenital or valvular abnormalities, irrespective of the ECG criteria used. The ECGs in these athletes revealed either normal or isolated Group 1 changes.

Sensitivity and Specificity of ESC Recommendations, Seattle criteria and Refined Criteria
Of the 3210 athletes who underwent echocardiography, 3087 (96.2%) were required to do so as part of their club’s policy, irrespective of clinical or ECG findings (805 BAs and 2282 WAs). This group contained all athletes diagnosed with pathology and was used to assess the effect of the refined criteria on the sensitivity and specificity of the overall screening process (Table 5A).

Compared with the ESC recommendations, the Seattle criteria were associated with a marked improvement in specificity for both BAs (40.3% to 79.3%) and WAs (73.8% to 92.1%). The refined criteria offered a further improvement in specificity, to 84.2% in BAs and 94.1% in WAs.

Sensitivity for all cardiac diseases remained 70.0% in BAs and 60.0% in WAs for all 3 criteria. Following exclusion of minor congenital and valvular abnormalities, the sensitivity for all 3 criteria improved to 100% in both BAs and WAs without compromising specificity (ESC: 40.1% BAs, 73.5% WAs; Seattle: 79.3% BAs, 92.1% WAs; refined criteria: 84.2% BAs, 93.9% WAs; Table 5B).

Inter-observer Variability between ECG Findings

There was excellent agreement with respect to ECG findings during re-analysis of a random selection of 1000 ECGs by the first and senior authors, translating to a kappa (measurement of agreement) of 0.97 (p<0.0001).

Further Investigations and ECG Predictors of Cardiac Disease

A substantial number of athletes (3210; 58.3%) underwent additional investigations after the ECG (Figure 6). This group included 1955 asymptomatic athletes with normal/training related ECG patterns who would have normally been cleared without additional tests but were required to have echocardiography as part of their club’s policy. None of the 1955 athletes were diagnosed with a serious structural disorder.
519 (9.4%) athletes were recommended for further investigations after ECG and echocardiography. Of these, 466 were advised a CMRI, exercise test and Holter monitor to exclude a cardiomyopathy based on marked ECG repolarisation changes (n=389) or structural changes placing them in the grey zone for cardiomyopathy (n=77), and 53 were advised an exercise test and Holter monitor on the basis of symptoms and/or family history (n=38) or a prolonged QT interval (n=15). Complete data was available in 454 athletes (87.5%), including all those with a prolonged QT interval, infero-lateral T-wave inversion, and a wall thickness ≥13mm (Figure 6).

Exercise testing facilitated the diagnosis of long-QT syndrome in 3/15 (20%) athletes with a prolonged QT interval. All 3 athletes revealed a QTc >500msec. Cardiac MRI after echocardiography aided the diagnosis of a cardiomyopathy in only 2 of 401 (0.5%) athletes with marked repolarisation changes or echocardiographic features of possible cardiomyopathy. Both athletes had HCM and exhibited lateral T-wave inversion. With respect to specific T-wave inversion patterns, lateral T-wave inversion was the only consistent finding in the 5 athletes with cardiomyopathy (all HCM), and had a positive-predictive value of 22.2% in WAs, 8.3% in BAs and 11.1% overall. In contrast, T-wave inversion confined to the inferior leads did not predict any cardiomyopathy.

Discussion

This study compared the performance of current ESC and Seattle criteria for ECG interpretation in athletes with proposed refined criteria in a large cohort of elite black and white athletes. All 3 criteria were also applied to a young group of asymptomatic athletes with HCM to assess their ability to detect a condition that accounts for a significant proportion of SCD in young athletes,
and often forms part of the differential diagnosis in athletic individuals with ECG anomalies or mild LVH on echocardiography.

**ESC recommendations and Seattle criteria versus Refined Criteria**

The results indicate that although current ESC recommendations perform well in detecting HCM and excluding potentially sinister structural disease, they are associated with unacceptably high false-positive rates, particularly in BAs. Based on current ESC recommendations, almost 1 in 2 BAs and almost 1 in 5 WAs exhibit ECG patterns warranting further evaluation (Figure 2). These findings are highly problematic, particularly in countries accommodating large populations of BAs, including the UK and US.

In agreement with a recent analysis,13 the Seattle criteria perform well in identifying sinister disease and are associated with a significant improvement in specificity in WAs. However, despite accounting for anterior T-wave inversion (V1-V4) as a normal ethnic variant,7,14,15,17,19,20 almost one-fifth of BAs continue to exhibit abnormal ECG patterns (Figure 2) after application of the Seattle criteria, primarily due to the presence of isolated voltage criteria for atrial enlargement and left axis deviation. Such ECG patterns also appear highly relevant in WAs and account for a high proportion of abnormal ECGs (Figures 2 and 3). We have recently demonstrated that the presence of any one of these ECG patterns, either in isolation or in combination with recognised training-related ECG patterns, correlates poorly with underlying cardiac disorders in asymptomatic elite athletes.14,15 By excluding these ECG patterns from the abnormal category, the refined criteria result in a significant improvement in specificity in athletes of both ethnicities whilst maintaining sensitivity (Table 5).

The refined criteria impact most impressively on the black athlete population, in whom the false-positive ECG rate is decreased by over 70% compared to current ESC
recommendations. Indeed, application of the refined criteria results in a lower positive ECG rate in BAs (11.5%) than is presently observed in WAs (16.2%) using the ESC recommendations (Figure 2). Importantly, the refined criteria also have a significant impact in WAs, reducing the false-positive ECG rate down to a far more acceptable level of 5.9%. In the current financial climate, the impact on resources and cost savings inherent in such refinement are difficult to ignore.

Clinical Implications

Identification of Pathology
The refined criteria identified all elite athletes with potentially sinister pathology and the majority (98.1%) of athletes with HCM. These observations are particularly important for BAs, who reveal a higher relative risk of exercise-related SCD from HCM.31

Irrespective of the criteria employed, the ECG was poor at identifying minor congenital abnormalities and valvular heart disease, some of which may theoretically degenerate more rapidly in individuals exercising at high intensities. Inclusion of clinical examination, which is usual practice in both the American Heart Association (AHA) and ESC screening protocols, improved the detection rate to over 40%, highlighting the importance of this aspect of pre-participation cardiovascular evaluation.

Future Directions
Despite the on-going debate between the AHA and ESC concerning routine use of 12-lead ECG, the vast majority of professional sporting organisations in the US32 and Europe incorporate an ECG in their screening protocols. Therefore a significant number of athletes, including BAs who comprise up to 70% of individuals participating in certain sports in the US, continue to be evaluated using electrocardiography prior to clearance to compete. The high false-positive rates
observed in BAs using current ECG screening criteria support concerns raised by the AHA. With this consideration in mind, the best alternative is to strive towards an improvement in screening specificity through better understanding of benign versus abnormal ECG patterns, coupled with appropriate training and education of physicians in the correct interpretation of an athlete’s ECG.\textsuperscript{12,33}

The ESC recommendations are unfavourable to BAs. The recently published Seattle criteria\textsuperscript{12} perform better by incorporating a growing body of scientific evidence\textsuperscript{7,17,19,20} relating to electrical remodelling in athletes of Afro-Caribbean ethnicity. Further refinement of current ECG criteria as demonstrated above improves the unfavourable situation in BAs without compromising detection of HCM. We have previously reported that T-wave inversion confined to V1-V4 in BAs is a normal variant.\textsuperscript{7,17,19,20} This study revealed that T-wave inversion confined to the inferior leads failed to predict cardiomyopathy in BAs (Figure 7). Therefore, it is possible that exclusion of this particular repolarisation pattern in BAs in the future may reduce the false positive rate to \(<10\%\).

\textbf{Study Limitations}

In this study, echocardiographic data was not available in all individuals and therefore we may have underestimated the prevalence of some minor abnormalities. However a large proportion of athletes (3210) underwent both ECG and echocardiography, which enabled robust conclusions on the role of ECG in identifying diseases implicated in exercise-related SCD. Given that many athletes with a normal ECG received only one echocardiogram, we cannot comment accurately on the false-negative results since some individuals may develop HCM at a later date. Although 98\% of our athletes with HCM exhibited an abnormal ECG, the authors recognise the heterogeneity of HCM and that a small proportion may reveal normal ECGs or one of the
The aforementioned isolated ECG patterns that we would now consider as normal variants. Finally, the study was conducted in elite athletes; therefore the applicability and comparisons of the refined criteria with the ESC recommendations and the Seattle criteria in non-elite athletes should be an area for further study.

**Conclusion**

Application of the proposed refined criteria significantly reduces the number of false-positive ECGs in both elite black and white athletes without compromising sensitivity. Coupled with appropriate training of physicians in ECG interpretation, such refinement of ECG screening criteria would minimise the risk of an erroneous diagnosis in BAs and lead to substantial savings from unnecessary investigations in both cohorts. The results from this preliminary study require further evaluation and confirmation by other centres. It is our aspiration that the data will provide an important evidence base for revising existing guidelines in the future.

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**Conflict of Interest Disclosures:** Drs Sheikh, Papadakis, Ghani, Zaidi and Gati were funded by research grants from CRY. Professor Sharma has been co-applicant on previous grants from CRY to study BAs.

**References:**


21. Weinstock J, Estes N a M. The heart of an athlete: black, white, and shades of grey with no


Table 1. Electrocardiographic parameters used to define various ECG abnormalities in the European Society of Cardiology recommendations, Seattle criteria and refined criteria.

<table>
<thead>
<tr>
<th>ECG Abnormality</th>
<th>European Society of Cardiology Recommendations⁹</th>
<th>Seattle Criteria¹²</th>
<th>Refined Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left atrial enlargement</td>
<td>Negative portion of the P wave in lead V1 ≥0.1mV in depth and ≥40msec in duration</td>
<td>Prolonged P wave duration of &gt;120msec in leads I or II with negative portion of the P wave ≥1mm in depth and ≥40msec in duration in lead V1</td>
<td>As ESC</td>
</tr>
<tr>
<td>Right atrial enlargement</td>
<td>P-wave amplitude ≥2.5mm in leads II, III or aVF</td>
<td>As ESC</td>
<td>As ESC</td>
</tr>
<tr>
<td>Left QRS-axis deviation</td>
<td>-30⁰ to -90⁰</td>
<td>As ESC</td>
<td>As ESC</td>
</tr>
<tr>
<td>Right QRS-axis deviation</td>
<td>&gt;115⁰</td>
<td>&gt;120⁰</td>
<td>As ESC</td>
</tr>
<tr>
<td>Right ventricular hypertrophy</td>
<td>Sum of R wave in V1 and S wave in V5 or V6 ≥10.5mm</td>
<td>Sum of R wave in V1 and S wave in V5 &gt;10.5mm and right axis deviation &gt;120⁰</td>
<td>As ESC</td>
</tr>
<tr>
<td>Complete left bundle branch block</td>
<td>QRS ≥120msec predominantly negative QRS complex in lead V1 (QS or rS), and upright monophasic R wave in leads I &amp; V6</td>
<td>As ESC</td>
<td>As ESC</td>
</tr>
<tr>
<td>Complete right bundle branch block</td>
<td>RSR’ pattern in anterior precordial leads with QRS duration ≥120msec</td>
<td>Not relevant</td>
<td>As ESC</td>
</tr>
<tr>
<td>Intraventricular conduction delay</td>
<td>Any QRS duration &gt;120msec including RBBB and LBBB</td>
<td>Any QRS duration ≥140msec or complete LBBB</td>
<td>As ESC</td>
</tr>
<tr>
<td>Pathological Q-wave</td>
<td>&gt;4mm deep in any lead except III, aVR</td>
<td>&gt;3mm deep and/or &gt;40msec duration in ≥2 leads except III and aVR ≥40msec in duration or ≥25% of the height of the ensuing R-wave</td>
<td>≥40msec in duration or ≥25% of the height of the ensuing R-wave</td>
</tr>
<tr>
<td>Significant T-wave inversion</td>
<td>≥2mm in ≥2 adjacent leads (deep) or ‘minor’ in ≥2 leads</td>
<td>≥1mm in depth in two or more leads V2–6, II and aVF or I and aVL (excludes III, aVR and V1)</td>
<td>As Seattle</td>
</tr>
<tr>
<td>ST-segment depression</td>
<td>≥0.5mm deep in ≥2 leads</td>
<td>As ESC</td>
<td>As ESC</td>
</tr>
<tr>
<td>Ventricular pre-excitation</td>
<td>PR interval &lt;120msec with or without delta wave</td>
<td>PR interval &lt;120msec with delta wave</td>
<td>As Seattle</td>
</tr>
</tbody>
</table>

LBBB, left bundle branch block; mm, millimetres; msec, milliseconds; RBBB, right bundle branch block.
Table 2. Characteristics of 103 athletes with hypertrophic cardiomyopathy.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at diagnosis (years)</td>
<td>24.3±6.9</td>
</tr>
<tr>
<td>Male Gender (%)</td>
<td>94.2</td>
</tr>
<tr>
<td>African/Afro-Caribbean Ethnicity (%)</td>
<td>33.0</td>
</tr>
<tr>
<td>Blood Pressure (mmHg)</td>
<td>123±12/73±11</td>
</tr>
<tr>
<td>Family history of hypertrophic cardiomyopathy/sudden cardiac death (%)</td>
<td>29.2</td>
</tr>
<tr>
<td>Mode of diagnosis (%)</td>
<td></td>
</tr>
<tr>
<td>Pre-participation screening abnormal ECG</td>
<td>81.3</td>
</tr>
<tr>
<td>Family screening</td>
<td>16.7</td>
</tr>
<tr>
<td>Other</td>
<td>2.0</td>
</tr>
<tr>
<td><strong>Echocardiographic characteristics</strong></td>
<td></td>
</tr>
<tr>
<td>Left atrial dimension (mm)</td>
<td>38.1±6.6</td>
</tr>
<tr>
<td>Left ventricular cavity dimension in diastole (mm)</td>
<td>47.3±5.9</td>
</tr>
<tr>
<td>Maximal left ventricular wall thickness (mm)</td>
<td>15.5±2.9</td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.61±0.23</td>
</tr>
<tr>
<td>Left ventricular mass (g)</td>
<td>255.1±76.9</td>
</tr>
<tr>
<td>Mitral Inflow E-wave (m/s)</td>
<td>0.77±0.17</td>
</tr>
<tr>
<td>Mitral Inflow A-wave (m/s)</td>
<td>0.47±0.11</td>
</tr>
<tr>
<td>E/A</td>
<td>1.78±0.55</td>
</tr>
<tr>
<td>E’ lateral (m/s)</td>
<td>0.12±0.03</td>
</tr>
<tr>
<td>E’ septal (m/s)</td>
<td>0.08±0.02</td>
</tr>
<tr>
<td>E/E’ average</td>
<td>8.2±2.8</td>
</tr>
<tr>
<td>Resting systolic anterior motion mitral valve leaflets (%)</td>
<td>8.6</td>
</tr>
<tr>
<td>Resting left ventricular outflow tract gradient ≥30mmHg (%)</td>
<td>3.4</td>
</tr>
<tr>
<td>Left ventricular ejection fraction (%)</td>
<td>68.2 ± 7.2</td>
</tr>
<tr>
<td>**LVH pattern (%)</td>
<td></td>
</tr>
<tr>
<td>Apical</td>
<td>35.7</td>
</tr>
<tr>
<td>Septal</td>
<td>44.0</td>
</tr>
<tr>
<td>Concentric</td>
<td>15.5</td>
</tr>
<tr>
<td>Other</td>
<td>4.8</td>
</tr>
<tr>
<td><strong>Electrocardiographic characteristics (%)</strong></td>
<td></td>
</tr>
<tr>
<td>Sinus bradycardia (heart rate &lt;60 beats per minute)</td>
<td>52.4</td>
</tr>
<tr>
<td>LVH (Sokolow-Lyon criteria)</td>
<td>60.2</td>
</tr>
<tr>
<td>Romhilt-Estes score ≥4/≥5</td>
<td>88.3/68.9</td>
</tr>
<tr>
<td>Right ventricular hypertrophy (Sokolow-Lyon criteria) (isolated)</td>
<td>10.7</td>
</tr>
<tr>
<td>Left atrial enlargement</td>
<td>37.9</td>
</tr>
<tr>
<td>Right atrial enlargement</td>
<td>18.4</td>
</tr>
<tr>
<td>Left-axis deviation</td>
<td>7.8</td>
</tr>
<tr>
<td>Right-axis deviation</td>
<td>1.9</td>
</tr>
<tr>
<td>Pathological Q-waves</td>
<td>25.2</td>
</tr>
<tr>
<td>Inverted T-waves</td>
<td>97.1</td>
</tr>
<tr>
<td>Deep</td>
<td>87.4</td>
</tr>
<tr>
<td>V1–V4</td>
<td>2.0%</td>
</tr>
<tr>
<td>Inferior leads</td>
<td>11.0%</td>
</tr>
<tr>
<td>Lateral leads</td>
<td>87.0%</td>
</tr>
<tr>
<td>ST-segment elevation</td>
<td>63.1</td>
</tr>
<tr>
<td>ST-segment depression</td>
<td>54.4</td>
</tr>
</tbody>
</table>

E/A, ratio of mitral inflow E and A waves; E/E’, ratio of mitral inflow E-wave to mitral annular tissue Doppler E’; LVH, left ventricular hypertrophy.
Table 3. Agreement between 3 criteria.

<table>
<thead>
<tr>
<th></th>
<th>Number of positive ECGs</th>
<th>Kappa</th>
<th>95% confidence interval</th>
<th>c-statistic</th>
<th>95% confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Black Athletes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESC positive, Seattle positive</td>
<td>222</td>
<td>0.50</td>
<td>0.45-0.55</td>
<td>0.78</td>
<td>0.76-0.80</td>
</tr>
<tr>
<td>Seattle positive, refined positive</td>
<td>139</td>
<td>0.73</td>
<td>0.68-0.79</td>
<td>0.93</td>
<td>0.92-0.95</td>
</tr>
<tr>
<td>ESC positive, refined positive</td>
<td>139</td>
<td>0.32</td>
<td>0.26-0.38</td>
<td>0.71</td>
<td>0.69-0.74</td>
</tr>
<tr>
<td><strong>White Athletes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESC positive, Seattle positive</td>
<td>305</td>
<td>0.57</td>
<td>0.53-0.61</td>
<td>0.91</td>
<td>0.90-0.92</td>
</tr>
<tr>
<td>Seattle positive, refined positive</td>
<td>228</td>
<td>0.85</td>
<td>0.81-0.88</td>
<td>0.98</td>
<td>0.98-0.99</td>
</tr>
<tr>
<td>ESC positive, refined positive</td>
<td>228</td>
<td>0.45</td>
<td>0.40-0.50</td>
<td>0.89</td>
<td>0.88-0.90</td>
</tr>
</tbody>
</table>

ESC, European Society of Cardiology.

Table 4. Characteristics of athletes with hypertrophic cardiomyopathy and a normal ECG.

<table>
<thead>
<tr>
<th>Age of Presentation</th>
<th>Sex</th>
<th>Ethnicity</th>
<th>Mode of Identification</th>
<th>Symptoms</th>
<th>Family history of hypertrophic cardiomyopathy</th>
<th>Examination Findings</th>
<th>ECG Findings</th>
<th>max-LVWT (pattern)</th>
<th>Relative Wall Thickness</th>
<th>LGE on CMRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>18</td>
<td>Male</td>
<td>Caucasian</td>
<td>Pre-participation screening*</td>
<td>None</td>
<td>No</td>
<td>Nil</td>
<td>Nil</td>
<td>14† (asymmetric septal)</td>
<td>0.53</td>
<td>No</td>
</tr>
<tr>
<td>23</td>
<td>Male</td>
<td>Caucasian</td>
<td>Familial screening</td>
<td>None</td>
<td>Yes</td>
<td>Nil</td>
<td>Nil</td>
<td>14 (asymmetric septal)</td>
<td>0.42</td>
<td>No</td>
</tr>
</tbody>
</table>

CMRI, cardiac magnetic resonance imaging; LGE, late gadolinium enhancement; max-LVWT, maximal left ventricular wall thickness. *On routine echocardiography. †No regression of left ventricular hypertrophy after detraining.
**Table 5.** Sensitivity and specificity of the screening process using different ECG criteria to detect: A. both major and minor cardiac abnormalities; B. major cardiac abnormalities only (95% confidence intervals in parentheses).

<table>
<thead>
<tr>
<th></th>
<th>European Society of Cardiology</th>
<th>Seattle Criteria</th>
<th>Refined Criteria</th>
<th>European Society of Cardiology</th>
<th>Seattle Criteria</th>
<th>Refined Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Black Athletes (n=805)</td>
<td>White Athletes (n=2282)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sensitivity</strong></td>
<td>70.0% (34.8%-93.3%)</td>
<td>60.0% (40.6%-77.3%)</td>
<td>60.0% (40.6%-77.3%)</td>
<td>70.0% (34.8%-93.3%)</td>
<td>60.0% (40.6%-77.3%)</td>
<td>60.0% (40.6%-77.3%)</td>
</tr>
<tr>
<td><strong>Specificity</strong></td>
<td>40.3%</td>
<td>84.2%</td>
<td>73.8%</td>
<td>79.3%</td>
<td>92.1%</td>
<td>94.1%</td>
</tr>
<tr>
<td><strong>Positive predictive value</strong></td>
<td>1.5% (0.6%-3.0%)</td>
<td>5.3% (2.1%-10.5%)</td>
<td>3.0% (1.8%-4.6%)</td>
<td>4.1% (1.7%-8.2%)</td>
<td>9.2% (5.6%-14.2%)</td>
<td>12.0% (7.3%-18.3%)</td>
</tr>
<tr>
<td><strong>Negative predictive value</strong></td>
<td>99.1% (97.3%-99.8%)</td>
<td>99.6% (98.7%-99.9%)</td>
<td>99.3% (98.8%-99.6%)</td>
<td>99.5% (98.7%-99.9%)</td>
<td>99.4% (99.0%-99.7%)</td>
<td>99.4% (99.0%-99.7%)</td>
</tr>
<tr>
<td><strong>False-positive rate</strong></td>
<td>59.7%</td>
<td>20.7%</td>
<td>15.8%</td>
<td>26.2%</td>
<td>7.9%</td>
<td>5.9%</td>
</tr>
<tr>
<td><strong>False-negative rate</strong></td>
<td>30.0%</td>
<td>30.0%</td>
<td>30.0%</td>
<td>40.0%</td>
<td>40%</td>
<td>40.0%</td>
</tr>
</tbody>
</table>

**B. Major Cardiac Abnormalities Only**

<table>
<thead>
<tr>
<th></th>
<th>European Society of Cardiology</th>
<th>Seattle Criteria</th>
<th>Refined Criteria</th>
<th>European Society of Cardiology</th>
<th>Seattle Criteria</th>
<th>Refined Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Black Athletes (n=805)</td>
<td>White Athletes (n=2282)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sensitivity</strong></td>
<td>100% (39.8%-100%)</td>
<td>100% (39.8%-100%)</td>
<td>100% (39.8%-100%)</td>
<td>100% (39.8%-100%)</td>
<td>100% (39.8%-100%)</td>
<td>100% (39.8%-100%)</td>
</tr>
<tr>
<td><strong>Specificity</strong></td>
<td>40.1%</td>
<td>84.2%</td>
<td>73.5%</td>
<td>79.3%</td>
<td>92.1%</td>
<td>93.9%</td>
</tr>
<tr>
<td><strong>Positive predictive value</strong></td>
<td>0.8%</td>
<td>3.1%</td>
<td>1.8%</td>
<td>2.4%</td>
<td>5.9%</td>
<td>7.4%</td>
</tr>
<tr>
<td><strong>Negative predictive value</strong></td>
<td>100% (0.2%-2.1%)</td>
<td>100% (0.6%-5.9%)</td>
<td>100% (0.8%-7.7%)</td>
<td>100% (0.2%-2.1%)</td>
<td>100% (0.6%-5.9%)</td>
<td>100% (0.8%-7.7%)</td>
</tr>
<tr>
<td><strong>False-positive rate</strong></td>
<td>59.9%</td>
<td>20.7%</td>
<td>15.8%</td>
<td>26.5%</td>
<td>7.9%</td>
<td>6.1%</td>
</tr>
<tr>
<td><strong>False-negative rate</strong></td>
<td>0.0%</td>
<td>0.0%</td>
<td>0.0%</td>
<td>0.0%</td>
<td>0.0%</td>
<td>0.0%</td>
</tr>
</tbody>
</table>
Figure Legends:

**Figure 1.** The definition of an abnormal ECG using (A) refined criteria; (B) European Society of Cardiology (ESC) recommendations;\(^9\) (C) Seattle criteria.\(^{12}\)

**Figure 2.** The number of positive ECGs produced by the 3 different ECG screening criteria.

**Figure 3.** Prevalence of the seven commonest abnormal ECG patterns in athletes, defined by the European Society of Cardiology recommendations and refined criteria.

**Figure 4.** Correlation of Kappa and c-statistic for each pair of criteria in white and black athletes (line represents trend of how these correlations are located).

**Figure 5.** Number of athletes with pathology, characterised by ethnicity and screening modality triggering diagnosis. Green font: minor congenital/valvular defects; red font: serious pathology.

**Figure 6.** Diagnostic algorithm for further investigations after initial screening which lead to an ultimate cardiac diagnosis in our athlete cohort. The total numbers of athletes cleared are shown on the right (green box) and the total numbers of athletes diagnosed with a cardiac condition are shown at the bottom (purple box). Sixty-five athletes requiring 2\(^{nd}\) tier investigations failed to attend (orange box).

**Figure 7.** Sensitivity, specificity, and predictive values of T-wave inversion for cardiomyopathy in black athletes. *The only cardiomyopathy diagnosed in our athlete cohort was hypertrophic cardiomyopathy.*
Figure 1

A. Refined Criteria Training Related Normal Variants
Not Warranting Further Investigation*

- Sinus bradycardia
- First degree AV block
- Incomplete RBBB
- Early repolarization
- Isolated QRS voltage criteria for left ventricular hypertrophy

B. Refined Criteria Borderline Variants
Potentially Warranting Further Investigation

- Left atrial enlargement
- Right atrial enlargement
- Left axis deviation
- Right axis deviation
- Right ventricular hypertrophy
- TWI up to V4 in BAS

C. Refined Criteria Training Unrelated Changes
Warranting Further Investigation

- ST-segment depression
- Pathological Q-waves
- Ventricular pre-excitation
- TWI beyond V1 in WAS beyond V4 in BAS
- Complete LBBB or RBBB
- QTC 460 msec in males
- 480 msec in females
- Brugada-like ER
- Atrial or vent. arrhythmias
- ≥2 PVCs per 10s tracing

If present in ISOLATION* If TWO OR MORE present

---

B. ESC Group 1 Training Related Changes

- Sinus bradycardia
- First-degree AV block
- Incomplete RBBB
- Early repolarization
- Isolated QRS voltage criteria for left ventricular hypertrophy

ESC Group 2 Training Unrelated Changes

- T-wave inversion
- ST-segment depression
- Pathological Q-waves
- Ventricular pre-excitation
- Complete LBBB or RBBB
- QTC >440 msec males
- QTC >460 msec females
- Left or right atrial enlargement
- Left or right atrial enlargement
- Left axis deviation / left anterior hemiblock
- Right axis deviation / left posterior hemiblock
- Right ventricular hypertrophy
- Ventricular arrhythmias
- Atrial/ventricular arrhythmias

---

C. Seattle Criteria Abnormal Findings in Athletes

- T-wave inversion beyond V2 in WAS and V4 in BAS
- ST-segment depression
- Pathological Q-waves
- Complete left bundle branch block
- Intraventricular conduction delay (any QRS ≥100 msec)
- Left axis deviation
- Short-QT interval ≤300 ms
- Right atrial enlargement
- Right ventricular hypertrophy pattern
- Ventricular pre-excitation
- Left atrial tachyarrhythmias
- Long-QT interval 460 ms in males
- Premature ventricular contractions
- Long-QT interval 480 ms in females
- Ventricular arrhythmias

---

KEY:

AV: Atrioventricular
LBBB: Left bundle branch block
WAS: White athletes
BAS: Black athletes
PVCs: Premature ventricular complexes
ER: Early repolarization
RBBB: Right bundle branch block
TMI: T-wave Inversion

*In otherwise asymptomatic athletes with no family history or abnormal examination findings. *When preceded by characteristic convex ST-segment elevation.
Figure 2
Figure 3

ECG Abnormalities by European Society of Cardiology (ESC) Recommendations and Refined Criteria (RC)
Figure 4
Figure 5

40 athletes with pathology

10 BAs

30 WAs

Screening Modality Triggering Diagnosis

4 on Hx/Ex

1 ACAO (symptoms)
1 BAV (murmur)
1 ASD (murmur)
1 VSD (murmur)

3 on Echo

2 MVP
1 BAV

3 on ECG

3 HCM (all with lateral TWI)

11 on ECG

1 BrS (Type 1 pattern)
2 HCM (lateral TWI)
3 LQTS (QTc >500 msec)
5 WPW (short PR, δ-wave)

12 on Echo

1 ASD
1 AR
1 Cor triatriatum
1 Pulmonary stenosis
4 MVP
4 BAV

7 on Hx/Ex

1 ASD (murmur)
1 VSD (murmur)
1 MVP (murmur)
1 WPW (short PR, δ-wave)

KEY.

AR: Aortic regurgitation
ASD: Atrial septal defect
BAs: Black athletes
BAV: Bicuspid aortic valve
BrS: Brugada syndrome
ACAO: Anomalous coronary artery origin
ECG: 12-lead electrocardiography
Echo: 2D-transthoracic echocardiography
Ex: Physical examination
HCM: Hypertrophic cardiomyopathy
Hx: History
LQTS: Long-QT syndrome
MVP: Mitral valve prolapse
TWI: T-wave inversion
VSD: Ventricular septal defect
WAs: White athletes
WPW: Wolff-Parkinson-White syndrome
Figure 7

T-wave Inversion in Black Athletes as markers of cardiomyopathy

Sensitivity: 100%
Specificity: 94.1%

Non-anterior

Sensitivity: 100%
Specificity: 100%

Inferior
Sensitivity: 0.0%
Specificity: 96.7%

Lateral
Sensitivity: 100%
Specificity: 97.4%

Positive-predictive value: 4.1%
Negative-predictive value: 100%
Positive-predictive value: 9.3%
Negative-predictive value: 100%
Comparison of ECG Criteria for the Detection of Cardiac Abnormalities in Elite Black and White Athletes
Nabeel Sheikh, Michael Papadakis, Saqib Ghani, Abbas Zaidi, Sabiha Gati, Paolo Adami, François Carré, Frédéric Schnell, Paloma Avila, Mathew Wilson, William McKenna and Sanjay Sharma

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