

Results of a nationally implemented de novo cardiac screening programme in elite rugby players in the UK

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ABSTRACT

Objectives: To investigate the feasibility and cost-effectiveness of a de novo, ECG-based cardiac screening programme of elite rugby players.

Design: Prospective, cohort study.

Participants: 1191 rugby players aged ≥ 16 years who were selected for the England teams or registered with the regional academies and Premiership clubs who underwent cardiac screening between 2010 and 2012.

Setting: Screening of young competitive athletes for conditions predisposing to sudden cardiac death (SCD) remains a contentious issue, primarily due to concerns relating to the false positive rate of the 12-lead ECG and the cost of subsequent investigations. In 2010 the Rugby Football Union (RFU), Premiership Rugby and the Rugby Players Association made an executive decision to offer cardiac screening to all professional and aspiring professional players in England. The cardiac screening included a health questionnaire, a 12-lead ECG and a cardiology consultation. Players with concerning findings on initial evaluation were offered on-site transthoracic echocardiogram (TTE). Athletes were referred for further investigations as deemed necessary. The overall cost of the screening program was estimated.

Results: After initial evaluation 9.7% of athletes underwent on-site TTE: 8.2% due to ECG anomalies and 1.4% due to concerns on the questionnaire. After TTE, only 2.9% of the players were referred for further evaluation. Two players were considered to harbour potentially serious conditions, of which 1 exhibited the Wolff-Parkinson-White phenotype and resumed competition after catheter ablation and the other was diagnosed with

hypertrophic cardiomyopathy and withdrew from competition. During a mean follow-up of 52.8 ± 5.5 months, none of the players who were cleared experienced any adverse cardiac events. The total cost of the screening programme was £59875, which averaged to a cost of £50 per player or £29,938 per condition identified. The application of the latest refined ECG criteria would have reduced the ECG false positive rate to $< 5\%$.

Conclusion: Preparticipation cardiovascular screening with 12-lead ECG is feasible. Refinement of the ECG criteria, the utility of on-site TTE and expert setting ensure a low false positive rate, minimise the burden of unnecessary investigations and reduce costs.

INTRODUCTION

Sudden cardiac death (SCD) is one of the leading causes of death in young (≤ 35 years) athletes. The majority of deaths are attributed to inherited or congenital cardiac disorders,¹ some of which can be detected during life. A mandatory, ECG-based, pre-participation cardiovascular screening (PPS) program in the Veneto region of Italy reported a reduction in the incidence of SCD in young athletes through the early identification of cardiomyopathies.² Based on these results, a number of International sporting and scientific organisations advocate cardiac screening of young competitive athletes for the prevention of such tragedies.³ Intense debate, however, remains over the feasibility of such an endeavour, relating primarily to the potential large number of false positive tests and the associated costs. In the current era, where national health systems are strapped of financial resources, a large number of specialist investigations aiming to reassure athletes with an abnormal ECG are likely to be cost-prohibitive. In an attempt to improve the ECG's specificity the ESC and other expert consortiums have revised the original 2005 ESC criteria relating to which ECG changes should raise suspicion of underlying heart disease.^{4,5} Preliminary studies suggest that adoption of more refined ECG criteria results in a significant reduction of the false positive rates.^{6,7}

In the United Kingdom there is no state-sponsored cardiac screening program. In 2010, the Rugby Football Union (RFU), Premiership Rugby and the Rugby Players Association introduced a nationwide, voluntary, cardiac screening for all eligible rugby players ≥ 16 years of age. This provided a unique opportunity to assess the feasibility and cost-effectiveness of

an ECG-based cardiac screening programme in a real life model. In addition this study offers a unique perspective as it describes ECG characteristics in a group of elite, male athletes typically with a high body mass, competing in a single discipline. We also compared the performance of newly proposed criteria to those currently recommended by the ESC.^{4,5,8}

METHODS

Setting:

In 1995 the charitable organisation Cardiac Risk in the Young (CRY) established a cardiac screening program, aimed at young (14-35 years) individuals at risk of SCD. Screening is offered to any young individual, athlete and non-athlete, who wish to be screened for self-protection. Through its mobile units, CRY is able to offer screening throughout the UK.

Cardiac screening of elite rugby players selected to represent England has been in place since 2005, in an attempt to identify those with quiescent cardiac disease and safeguard their safety. The small but constant trickle of SCDs in young sportsmen, led the Rugby Football Union, Premiership Rugby and the Rugby Players Association to expand the existing programme. Since 2010, cardiac screening is offered to all players aged ≥ 16 years who represent England and those playing at the highest level of domestic competition, including players registered with the Premiership clubs (n=12) and the Regional Academies. The cardiac screening events required the co-ordinated efforts of respective team

administrators and medical staff and CRY in identifying the athletes eligible for screening, planning and implementing the screening, delivering the results and arranging appropriate follow-up. All screenings were conducted by CRY. The CRY screening team consisted of a screening manager, a team of accredited cardiac physiologists and a cardiology fellow with experience in sports cardiology and inherited cardiac diseases. The cardiac screening included a detailed health questionnaire, a 12-lead ECG and a consultation with the cardiologist. On-site transthoracic echocardiogram (TTE) was performed on individuals who exhibited an abnormality during the initial evaluation. Athletes were referred for further comprehensive evaluation or regular follow-up, as deemed necessary.

Participants

Between January 2010 and December 2012, 1191 male rugby players (aged 14 – 37 years; 6 players aged >35 years) underwent PPS. The health questionnaires, including a consent form, were distributed to the players prior to the screening. Questions related to the presence of cardiac symptoms, such as syncope, dizziness, chest pain, palpitations and shortness of breath, family history of inherited cardiac disease or premature SCD (≤ 40 years) and drug history. In addition, data regarding height, weight, ethnicity, and training level and duration were collected.

12-lead electrocardiography:

A standard 12-lead ECG was performed during quiet respiration at rest in all athletes using a Philips Pagewriter Trim III (Bothel, WA, USA) machine with a paper speed of 25mm/s and

amplification of 0.1mV/mm. The ECG was performed in a supine position and electrodes were placed carefully to ensure consistency of precordial lead positions. The ECG trace was stored electronically. The ECG findings were classified as 'Group 1' or 'training-related' and 'Group 2' or 'training-unrelated' changes similar to the ESC 2010 recommendations.⁴ The resting heart rate, PR interval, QRS axis, QRS duration, QT interval, voltage of QRS complex, T-wave abnormalities and ST-segment changes were recorded. The QT interval was corrected for heart rate (QTc) using Bazett's formula. Left ventricular hypertrophy (LVH) was determined by the Sokolow-Lyon voltage criterion (LVH: S-V1 + R-V5/V6 = >3.5mV). Early repolarization (ER) was defined by J-point elevation of ≥1mm in ≥2 continuous leads (anterior, inferior or lateral); and ER in inferior and lateral leads was further characterised based on morphology of QRS complex (notched or slurred) and ST-segment (ascending or descending/horizontal). Table 1 outlines the definitions of all ECG changes that required further evaluation and provides a comparison with the 2010 ESC and "Seattle" criteria.

Trans-thoracic echocardiogram:

Two-dimensional trans-thoracic echocardiogram (TTE) was performed by an accredited cardiologist or a senior cardiac physiologist. A Philips iE33 or Philips CX50 machine with 3 MHz transducer were used. Standard views were obtained and chamber and wall thickness measurements were performed as per current guidelines.⁹ The continuous- and pulsed-Doppler, as well as colour tissue-Doppler images were acquired using standard views.¹⁰

Further Evaluation and Follow-up

Athletes with personal or family history, ECG or echocardiographic anomalies highly suggestive of underlying cardiac pathology were invited for further clinical evaluation. All further evaluations were performed after referral by the relevant player's family physician to a specialist cardiac centre via the National Health Service (NHS). Evaluations were dictated by the local protocols of the relevant specialist centre and included upright exercise stress testing \pm cardiopulmonary testing, 24-48 hour Holter, and cardiac magnetic resonance imaging with gadolinium injection to check for the broader phenotypic manifestations of underlying cardiac disease. Athletes with phenotypes outside the conventional limits but considered most likely to represent cardiac adaptation to exercise were invited to repeat cardiac evaluations on an annual or two-yearly basis.

Cost analysis:

The cost of the initial cardiac screening, including: questionnaire, 12-lead ECG, consultation and possible on-site ECHO, was set at £35 per athlete. This rate is based on the subsidised rate charged by CRY for every young individual (14-35 years) in the UK who wishes to be screened. The additional costs of the screening programme are funded by the charity's fundraising activities and voluntary work by participating individuals. Costs resulting from referral to specialist centres and further diagnostic tests were derived from the NHS payment-by-result tariff 2012-2013; and are expressed in GBP (£).

Ethical Approval/Consent

Ethical approval was granted by the National Research Ethics Service, Essex 2 Research Ethics Committee. Written consent was obtained from individuals aged ≥ 16 years and from a parent/guardian for those aged < 16 years.

Statistical analysis:

Data manipulation and analysis were performed using SPSS software, version 20 (Chicago, IL, USA). Variables were tested for normality using the Kolmogorov-Smirnov test. Values were expressed as mean \pm standard deviation (SD) or percentages, as appropriate.

RESULTS

Clinical characteristics:

Detailed demographics of the rugby players are reported in table 2. The mean body surface area of the cohort was $2.25 \pm 0.2 \text{ m}^2$ reflecting the large body habitus of the athletes. Consistent with their professional status, the athletes exercised for an average of 17.2 ± 5.4 hours per week.

Health questionnaire:

A significant proportion of athletes ($n=179$; 15%) reported one or more symptoms in their health questionnaire including: dizziness ($n=101$; 8.5%), chest pain or tightness ($n=71$; 5.9%),

syncope (n=48; 4%), palpitations (n=47; 3.9%) and shortness of breath (n=31; 2.6%). Only 18 athletes (1.5%) reported symptoms related to exertion. After consultation with the cardiologist, only 6 (0.5%) athletes were considered to exhibit clinically significant symptoms requiring further evaluation. Ten (0.8%) athletes reported a history of premature SCD or an inherited cardiac disease. One athlete had a history of dilated aortic root based on previous screening. Thirty-six (3%) athletes reported a history of asthma.

12-lead Electrocardiogram:

Group 1 ECG changes were detected in 691 (58%) athletes, whereas 107 (9%) athletes exhibited at least one group 2 change (Figure 1). The most common group 1 changes included the early repolarization (ER) pattern (48.2%), sinus bradycardia (42.6%) and voltage criterion for LVH (22.9%) (Table 3). The ER pattern was confined to inferior and/or lateral leads in 20.5% athletes and comprised predominantly of a notched QRS (70.2%) with an associated ascending ST-segment (82%). A slurred QRS with associated descending or horizontal ST-segment confined in the inferior leads was present in 6.6% of rugby players.

The most prevalent group 2 ECG anomaly was the presence of T-wave inversions, which was present in 42 (3.5%) athletes. Of the T-wave inversions, 12 (1%) were confined in the anterior leads (V1-V4), but only 4 (0.3%) extended beyond lead V2 and overlapped with the arrhythmogenic right ventricular cardiomyopathy (ARVC) phenotype. In 27 (2.3%) athletes the inferior leads were involved (II, III, aVF), and 3 (0.25%) athletes exhibited T-wave inversion extending in the lateral leads (I, aVL, V5-V6). Bundle branch block was rare (0.7%),

with RBBB being the predominant pattern. Pathological Q-waves were present in only 1 athlete, while ST-segment depression was not identified in any athlete. Patterns considered to represent softer ECG indicators of underlying heart disease such as voltage criterion for axis deviation, atrial enlargement and RVH were present in 3.7%, 1.1% and 1% of athletes, respectively (Table 2). A prolonged QTc ($>470\text{ms}$) was seen in 3 (0.25%) athletes, none of who had symptoms or relevant family history of note. One athlete had pre-excitation on his ECG suggestive of an accessory pathway.

Echocardiographic findings:

After initial evaluation, 115 athletes underwent on-site TTE; 98 (8.2%) as a result of a Group 2 anomaly on the ECG and 17 (1.4%) due to concerning findings in the questionnaire (Figure 2). The attending physician considered that on-site TTE was not essential in nine individuals with borderline voltage criteria for axis deviation, RVH and RBBB, in the absence of symptoms or family history of note. Three athletes had absolute LVEDD of $>65\text{mm}$ (69mm, 70mm and 72mm) and only 2 athletes had an absolute LV wall thickness of $>12\text{mm}$ (13mm and 14mm). None of the athletes, however, demonstrated associated echocardiographic features suggestive of cardiomyopathy. Other minor cardiac abnormalities detected on TTE included 1 bicuspid aortic valve, 1 mild mitral regurgitation, 1 mild pulmonary stenosis, 1 mildly dilated aortic root (44mm) and 1 patent ductus arteriosus.

Further diagnostic evaluation and follow-up:

Of the 115 athletes requiring TTE: 81 (70%) were cleared after a normal scan; 8 players (5 with minor cardiac abnormalities and 3 with absolute LVEDD of >65mm) were referred for surveillance TTE, organised during the time of future screenings; and 26 players were referred for further comprehensive evaluation. Further diagnostic evaluation included cardiac magnetic resonance imaging (CMR) (n=13), exercise treadmill/cardiopulmonary test (ETT) (n=18) and 24-hour ECG monitoring (n=20). Two athletes were diagnosed with a condition predisposing to exercise related SCD. An athlete with the Wolf-Parkinson-White phenotype (Figure 3A) was referred for an electrophysiological study and underwent radiofrequency catheter ablation of the accessory pathway. Following successful recovery he returned to competition. One athlete with T-wave inversion extending to the lateral leads (Figure 3B) but normal TTE indices at initial screening (maximal wall thickness of 11mm, LVEDd of 58mm, LA of 43mm, normal systolic & diastolic indices) was diagnosed with hypertrophic cardiomyopathy (HCM) during subsequent evaluation and follow-up.

Another athlete with T-wave inversions in anterior and lateral leads (I, aVL, V2-V4) did not demonstrate any abnormality on extensive evaluation. He was offered re-evaluation after a period of de-training, and this resulted in resolution of ECG changes. He continues to compete but remains under follow-up on an annual basis. One player exhibited a small PDA with little hemodynamic significance and was asymptomatic; therefore as per Bethesda and ESC recommendations, he was allowed to compete in professional sports with repeat TTE every 2 years. Those with dilated LV cavity underwent surveillance TTE which did not show any significant change; their LV dimensions were deemed acceptable when indexed to body surface area, and other echocardiographic parameters including systolic and diastolic

functions were normal. The remaining players with minor valve abnormalities undergo annual or 2-yearly TTE and continue to play professional rugby.

The 3 athletes with prolonged QTc underwent Holter monitor and exercise stress testing. They did not demonstrate any features to suggest underlying long QT syndrome and were therefore allowed to compete.

During a mean follow-up period of 52.8 ± 5.5 months, none of the players who were cleared after ECG have experienced any adverse cardiac events. Three players were lost to follow-up during the study: 1 with family history of sudden death, asymptomatic, with normal ECG who was referred for comprehensive evaluation for potentially inherited cardiac conditions; 1 with infero-lateral T-wave inversion; and 1 with inferior T-wave inversion.

Cost of screening program and further investigations:

The cardiac screening program was organised and supported by Cardiac Risk in the Young (CRY) at a nominal rate of £35 per player. This included initial evaluation with health questionnaire, 12-lead ECG, consultation with a cardiologist and on-site TTE as required. Thus the cost of initial screening for all 1191 rugby players was £41,685. All further evaluations were performed after referral by the relevant player's family physician to a specialist cardiac centre via the NHS. Cost estimates for the referrals and further tests were based on published NHS payment-by-result tariffs as follows: cardiology consultation (£210),

TTE (£57), 24-hour ECG (£145) and ETT (£145), CMR with reporting (£229), and catheter ablation (£2164). The cost of all these additional tests and follow-up was £18,190. The total cost for the 1191 athletes was £59875, averaging a cost of approximately £50 per player screened. A total of 7 players were identified with an abnormality; of these 2 had serious and potentially life-threatening cardiac disease (1 HCM, 1 WPW) and another 5 needed long-term surveillance. Thus the cost of identifying a player with any cardiac abnormality was £8,554, while the cost of identifying one player with potentially life-threatening condition was £29,938.

Assuming an ECG sensitivity of 100% for potentially life-threatening conditions, the false positive rate for the screening program comprised of health questionnaire, ECG and consultation was 9.5%, the majority (96 of the 115 cases) comprised of a false positive ECG. The inclusion of selective on-site TTE reduced the false positive rate to 2.7% by clearing 81 cases that would otherwise require referral to a cardiology specialist for further evaluation. That would equate to overall savings of £21,627 (£18 per athlete screened) assuming that every individual referred to a specialist would require as a minimum a cardiology consultation and a TTE as part of their evaluation.

Evaluation of different ECG screening criteria:

Utilising the 2010 ESC ECG criteria would have increased the ECG false positive rate from 8.8% to 20.1%, due to the inclusion of a considerable number of athletes outside the conservative QTc intervals (n=58 with QTc <380ms; n=53 with QTc >440ms) and the

presence of a PR interval < 120ms in the absence of pre-excitation (n=23).⁴ Assuming that all individuals would require as a minimum on further evaluation, a cardiology consultation, a 24-hour ECG and ETT, the estimated additional costs would amount to a minimum of £67,000 for the entire cohort or £56 per athlete screened.

Utilising the Seattle criteria would have resulted in an overall reduction of the ECG false positive rate from 8.8% to 6.5%.⁵ The reduction in the false positive rate would be primarily due to differences in the definition of T-wave inversion requiring further evaluation in the anterior (n=8 confined in V1-V2) and inferior (n=15 in leads III and aVF only). In addition, 11 athletes would not require TTE based on the Seattle definitions of RVH (n=5) and non-specific Intraventricular conduction delay (n=1) and the exclusion of RBBB (n=5) from ECG anomalies indicative of cardiac pathology. On the contrary, based on the Seattle criteria definition of pathological Q-waves, 7 additional athletes would have required at least on-site TTE.

Utilising the refined ECG criteria as proposed by Sheikh et al.⁸ would result in significant reduction of the false positive ECG rate from 8.8% to 4.9%. This is due to the inclusion of ECG criteria for atrial enlargement, QRS axis deviation and right ventricular hypertrophy, when present in isolation, to normal variants (Table 3). Such criteria were present in isolation in 49 athletes. Only 1 athlete with isolated right axis deviation (RAD) demonstrated evidence of mild pulmonary stenosis on TTE, requiring regular surveillance.

DISCUSSION:

Concerns relating to the implementation of pre-participation cardiac screening in competitive athletes focus primarily on the low incidence of SCD in young athletes, the associated high false positive rates and prohibitive costs. Published literature relating to the impact and cost-effectiveness of cardiac screening in athletes reports highly variable results, in a non-standardised way, and is commonly based on a number of assumptions and theoretical projections. This is the first large-scale study to report on a real life, nationwide screening program of elite competitive athletes from a single sporting discipline. Importantly, this study involves a cohort of professional athletes of extraordinary size and strength often considered likely to exhibit a higher prevalence of ECG anomalies and extremes of cardiac dimensions, posing a greater challenge in differentiating athlete's heart from quiescent cardiac disease.

Value of individual screening modalities:

In our study, both rugby players with potentially life threatening cardiac abnormalities (HCM and WPW) were detected on the basis of an abnormal ECG. Reassuringly, the ECG would have flagged both players for further evaluation, irrespective of the criteria used. In addition, the ECG prompted further evaluation with TTE in 3 other players with less significant cardiac abnormalities: 1 with RBBB and bi-cuspid aortic valve, 1 with isolated RAD and mild pulmonary stenosis and 1 with inferior T-wave inversion and PDA. It is unclear whether these ECG anomalies relate to the structural findings or are simply a chance finding.

On the contrary, of the 179 (15%) athletes who reported cardiovascular symptoms on the questionnaire, only 0.5% were considered significant after consultation with our cardiologist. This highlights the importance of employing physicians with experience in the field of sports cardiology and inherited cardiac diseases, who are able to distinguish benign symptoms from those indicative of cardiac pathology. None of the players with symptoms demonstrated any significant abnormality on further investigations or follow-up. Similar to existing studies, our study underscores the poor predictive value of symptoms as indicators of cardiac disease in young athletes and reinforces the importance of ECG for the detection of cardiac conditions implicated in SCD.¹¹⁻¹⁴

Utility of different ECG criteria:

Our study demonstrated a false positive ECG rate of 8.8% utilising our group's criteria, which are similar to the criteria proposed by the ESC in 2010 with notable exception the definitions of an abnormal QT interval and pre-excitation. This is very reassuring given the athletic prowess and body habitus of our athletes, as it compares favourably with the false positive rates reported in the literature. Although direct comparisons are difficult due to the different ECG criteria used, our results are similar to the study by Chevalier *et al.*, who evaluated 135, predominantly Caucasian, French, rugby players and identified distinctly abnormal ECGs, comprised of T-wave inversions, extreme axis deviation and pre-excitation, in 4% of the players.¹⁵

It is widely accepted that the criteria recommended by the ESC for the interpretation of a young athlete's ECG require refinement in order to incorporate data that have emerged since 2010 and to accommodate for the effect of ethnicity, age, gender and sporting discipline on the athlete's ECG.¹⁶⁻²¹ The Seattle criteria, published in 2013, were an improvement on the ESC criteria resulting in significant reduction of the false positive results and therefore a reduction of athletes subjected to unnecessary and costly investigations. Recent publications, however, investigated a number of ECG indices, considered by both expert consensus documents to represent phenotypes of quiescent cardiac pathology, and demonstrated that in isolation they were unlikely to be associated with disease.^{22,23} This led to a proposal for further refinement of the ECG criteria in athletes, which on initial assessment appear to reduce the number of false positive tests and improve specificity without compromising the sensitivity of the ECG in detecting potentially sinister cardiac disease.^{8,24} In agreement with current literature, the adoption of the 2010 ESC criteria in our cohort would have resulted in an unacceptably high proportion (20%) of athletes requiring further evaluation with escalating costs but no apparent increase in the diagnostic yield. In contrast application of the Seattle or the refined criteria would have reduced the false positive rate to 6.5% and 5%, respectively, while still identifying the two athletes with potentially life threatening conditions.^{8,24,25}

Cost-effectiveness of cardiac screening:

Literature on the cost-effectiveness of cardiac screening is fairly limited and suggests highly variable costs ranging from \$42,900 per life-year saved to \$14.4 million per life saved. These results should be viewed with caution as all existing studies exhibit significant limitations.

First and foremost all studies use theoretical projection models rather than true costs. Secondly, Wheeler et al.²⁶ and Halkin et al.²⁷ extrapolated results from the Italian experience to the US population, predominantly high-school athletes, which may not hold true. Finally, the studies by Wheeler et al.²⁶ and Fuller et al.²⁸ include a number of arbitrary assumptions relating to the life years saved by identifying a potentially life-threatening condition. Our study is the first study to report on a real life, nationwide screening programme of elite, professional athletes competing in a single sporting discipline. The cost of £50 per player or £29,938 (\$44,668 or €41,000 based on exchange rates on the 01/04/2015) per condition identified is well within the conventional cost-effectiveness limits utilised in different health care systems, even if we assume that only 1 life-year was saved for every condition identified.

There is general consensus that routine TTE is not recommended as part of the initial athlete's evaluation, as it only improves marginally the diagnostic yield for conditions predisposing to SCD in young athletes at a high cost.^{28,29} This would have also been true for the CRY screening programme as performing a TTE in every player would have considerably reduced the number of players screened per session and would have had a significant impact on our resources and associated costs. Our model, however, introduces a novel screening protocol, which utilises on-site TTE as a second line investigation. In our cohort only 1 in 10 rugby players required a TTE. The addition of on-site TTE reduced the athletes requiring further evaluation from 9.7% to 2.9%, resulting in considerable savings (£10,814 per potentially life-threatening condition identified and £18 per athlete screened) and minimising the burden and costs of further comprehensive evaluation on the NHS. In

addition, excluding a severe disease phenotype on TTE offers considerable reassurance to the player, team and physician until further investigations are completed.

Finally, the adoption of more refined ECG criteria is likely to result in considerable cost savings as our study indicates that they may potentially reduce the ECG false positive rate to less than 5%. Such a low false positive rate may negate the need for on-site TTE in our programme or at the very least reduce the burden of TTE as only 1 in every 20 athletes will fail initial evaluation.

Limitations:

The lack, of a specific pathway for further evaluation after the initial screening and reliance on primary care physicians to initiate an NHS referral to a centre of their choice meant that athletes with similar phenotypes were investigated in a variable way. In addition, for a significant proportion of the athletes, the authors relied on reports from other centres, though specialist, rather than our own investigative outcomes.

The reported costs were subsidised by the charitable organisation CRY and as such it may be argued that they underestimate the true cost of screening. If we substitute the CRY fee of £35 for the cost of a cardiology consultation and 12-lead ECG (£210) that the NHS charges then the cost per condition identified would raise to £134,150 and the cost per athlete screened to £225. However, the aim of the study was to present a real life, sustainable

screening program. Cardiac risk in the young is a well-established charity in existence since 1995. Since its inception the screening program has expanded year upon year, and the charity currently screens in excess of 17,000 young individuals per annum with the exact screening protocol described in this study. As such, although the costs associated with screening young athletes may seem unattainable for the strained financial resources of many health care systems, our study provides at the very least a convincing argument for alternative funding sources that national health care systems and sporting organisations should consider.

Conclusion:

Our study on a well-characterised cohort of elite professional athletes demonstrates that cardiac screening with 12-lead ECG is feasible and may result in the identification of potentially sinister conditions. The refinement of the ECG criteria and the expert setting safeguard a relatively low false positive rate, minimise the burden of unnecessary investigations and reduce costs.

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COMPETING INTERESTS

S Ghani, A Zaidi, S Gati, M Papadakis were funded by a research grants from the charitable organization Cardiac Risk in the Young (CRY), which supports preparticipation screening of young athletes.

S Sharma has been co-applicant on previous grants from CRY to study athletes.

What is already known on this subject

- Sudden cardiac death is one of the leading causes of death in young (≤ 35 years) athletes.
- ECG-based cardiac screening of athletes can identify conditions predisposing to sudden cardiac death.
- Widespread implementation of cardiac screening in competitive athletes is hindered by concerns relating to the low incidence of SCD in young athletes, the high false positive rate of the 12-lead ECG and prohibitive costs of subsequent investigations.

What this study adds

- Nationwide cardiac screening with 12-lead ECG is feasible and can identify potentially sinister conditions.
- Adoption of refined ECG criteria and the expert setting safeguard a relatively low false positive rate, minimise the burden of unnecessary investigations and reduce costs.
- On-site, second line echocardiography reduces referral rates for further investigations and may limit costs.
- Although the costs associated with screening young athletes may seem unattainable for the strained financial resources of most national health care systems, screening could be implemented by utilising alternative funding sources such as sporting federations, clubs and charitable organisations.

TABLES

Table 1: ECG criteria considered abnormal during CRY screenings leading to further evaluation. Comparison is presented with the 2010 ESC criteria and the 2013 Seattle criteria.

ECG Abnormality	ESC Recommendation	Seattle Recommendation	CRY ECG criteria
Right atrial enlargement	P-wave amplitude \geq 2.5mm in lead II, III, or aVF	As ESC	As ESC
Left atrial enlargement	Negative portion of the P wave in lead V ₁ \geq 1mm in depth and \geq 40ms in duration.	Prolonged P wave duration of > 120 ms in lead I or II with negative portion of the P wave \geq 1mm in depth and \geq 40ms in duration in lead V ₁	As ESC
Right QRS axis deviation	$>115^\circ$	$>120^\circ$	As ESC
Left QRS axis deviation	-30° to -90°	As ESC	As ESC
Right ventricular hypertrophy	Sum of R wave in V ₁ and S wave in V ₅ or V ₆ ≥ 10.5 mm	Sum of R wave in V ₁ and S wave in V ₅ > 10.5 mm and right axis deviation $> 120^\circ$	As ESC
Complete LBBB	QRS ≥ 120 ms, predominantly negative QRS complex in lead V ₁ (QS or rS), and upright monophasic R wave in leads I and V ₆	As ESC	As ESC
Complete RBBB	RSR' pattern in anterior precordial	Not relevant	As ESC

	leads with QRS duration ≥ 120 ms		
Non-specific Intraventricular conduction delay	Any QRS duration > 110 ms of non RBBB or LBBB morphology	Any QRS duration ≥ 140 ms	Any QRS duration ≥ 120 ms of non RBBB or LBBB morphology
Pathological Q-wave	> 4 mm deep in any lead except III, aVR	> 3 mm deep 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 ≥ 2 leads V_2 – V_6 , II and aVF, or I and aVL (excludes III, aVR, and V_1)	≥ 1 mm deep in ≥ 2 adjacent leads
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	PR interval < 120 ms with delta wave
Atrial arrhythmias	Not stated	Supraventricular tachycardia, atrial-fibrillation, atrial-flutter	Supraventricular tachycardia, atrial-fibrillation, atrial-flutter
Ventricular arrhythmias	Not stated	≥ 2 PVCs per 10sec tracing and couplets, triplets and non-sustained ventricular tachycardia	≥ 1 PVC per 10sec tracing and any other ventricular arrhythmias
Prolonged QTc	> 440 ms	≥ 470 ms	≥ 470 ms
Short QTc	< 380 ms	≤ 320 ms	≤ 320 ms
Brugada-like pattern	Brugada-like early repolarisation pattern	High take-off and downsloping ST segment elevation followed by a negative T wave in ≥ 2 leads in V_1 – V_3	Type-1 pattern OR type-2 that converts to type-1 by raising leads V_1 and V_2 to 3 rd and 2 nd intercostal space

Table 2: Demographic characteristics of rugby players

	Mean \pm SD
Age (years)	22.5 \pm 5.1
Height (cm)	185.4 \pm 7.3
Weight (kg)	99.1 \pm 12.8
Body Mass Index (kg/m ²)	28.8 \pm 3.1
Body Surface Area (m ²)	2.25 \pm 0.2
ETHNICITIES	Percentage
Caucasian	1019 (85.6%)
Afro-Caribbean	66 (5.5%)
Other*	106 (8.9%)

(*) = Players from Argentina, New Zealand, Pacific Islands and South Africa

Table 3: ECG characteristics of rugby players

ECG Parameter	Numbers (%) n=1191
GROUP 1 (training-related) ECG findings	
Sinus Bradycardia (HR < 60)	507 (42.6%)
1 st Degree AV Block (PR > 120ms)	99 (8.3%)
Partial Right Bundle Branch Block	92 (7.7%)
Left Ventricular Hypertrophy	275 (23.1%)
Early Repolarization – overall prevalence	578 (48.5%)
- ER in anterior leads	468 (39.9%)
- ER in inferior and/or lateral leads	244 (20.5%)
GROUP 2 (training-unrelated) ECG findings	
Abnormal T-wave inversions (TWI)	42 (3.5%)
- TWI confined in anterior leads	12 (1%)
- TWI in inferior leads	27 (2.3%)
- TWI in lateral leads	3 (0.25%)
Abnormal Q-waves	1 (0.08%)

Right Bundle Branch Block	7 (0.6%)
Left Bundle Branch Block	1 (0.08%)
Right Atrial enlargement	4 (0.3%) [3 in isolation]
Left Atrial Enlargement	9 (0.8%) [7 in isolation]
Right Axis Deviation	13 (1.1%) [10 in isolation]
Left Axis Deviation	31 (2.6%) [24 in isolation]
Right Ventricular Hypertrophy	12 (1%) [5 in isolation]
Ventricular Ectopics	6 (0.5%)
2 nd Degree (Wenckebach) AV Block	1 (0.08%)
Inter-ventricular Conduction Delay	1 (0.08%)
Wolff-Parkinson-White pattern	1 (0.08%)
Prolonged QTc interval ($\geq 470\text{ms}$)	3 (0.25%)

Brugada pattern	0
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FIGURES

Figure 1: Prevalence of ECG abnormalities in rugby players according to ESC classification

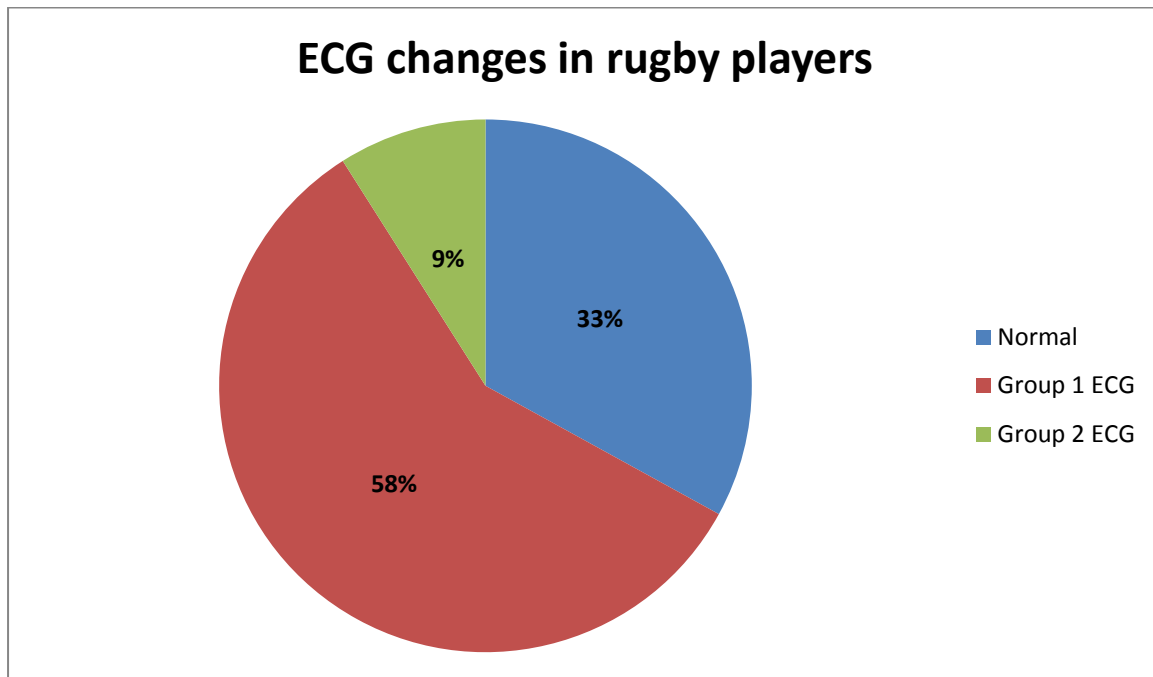
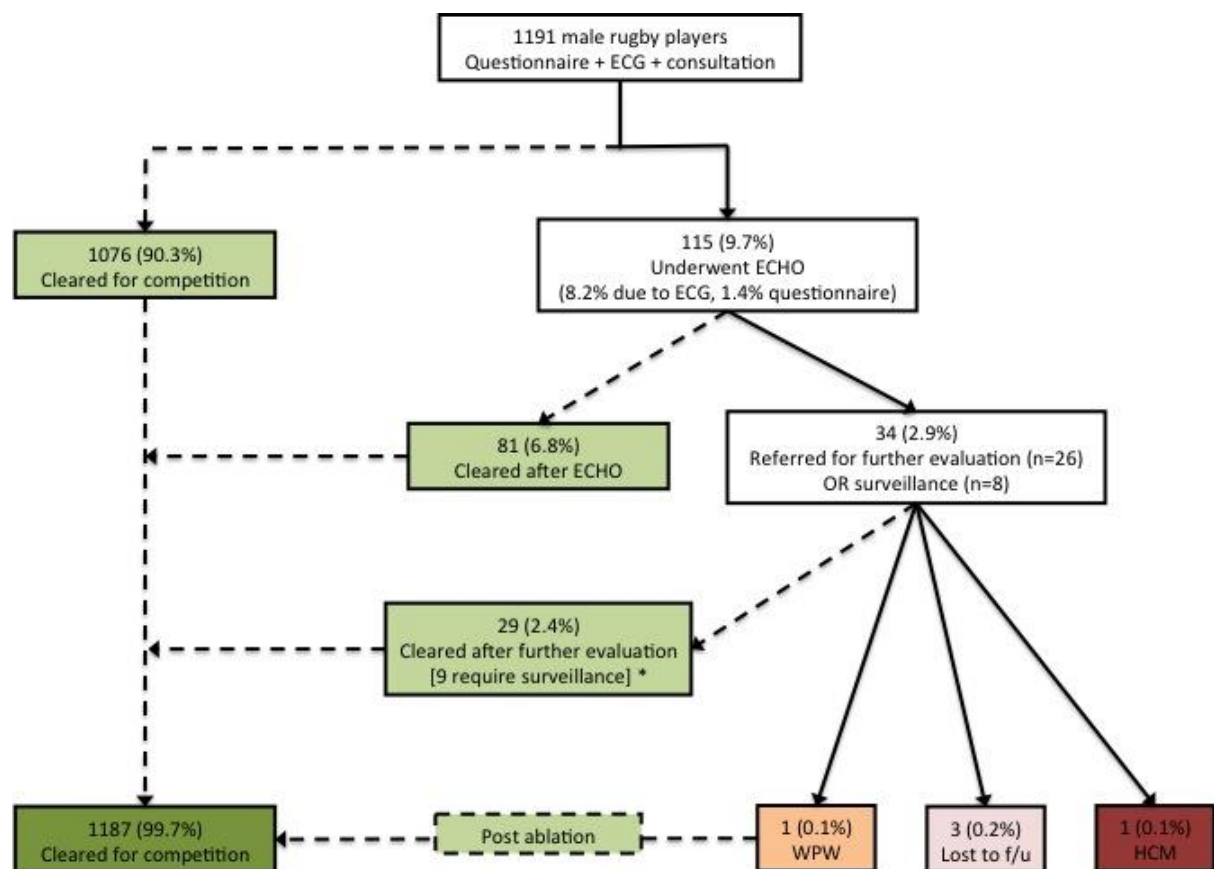
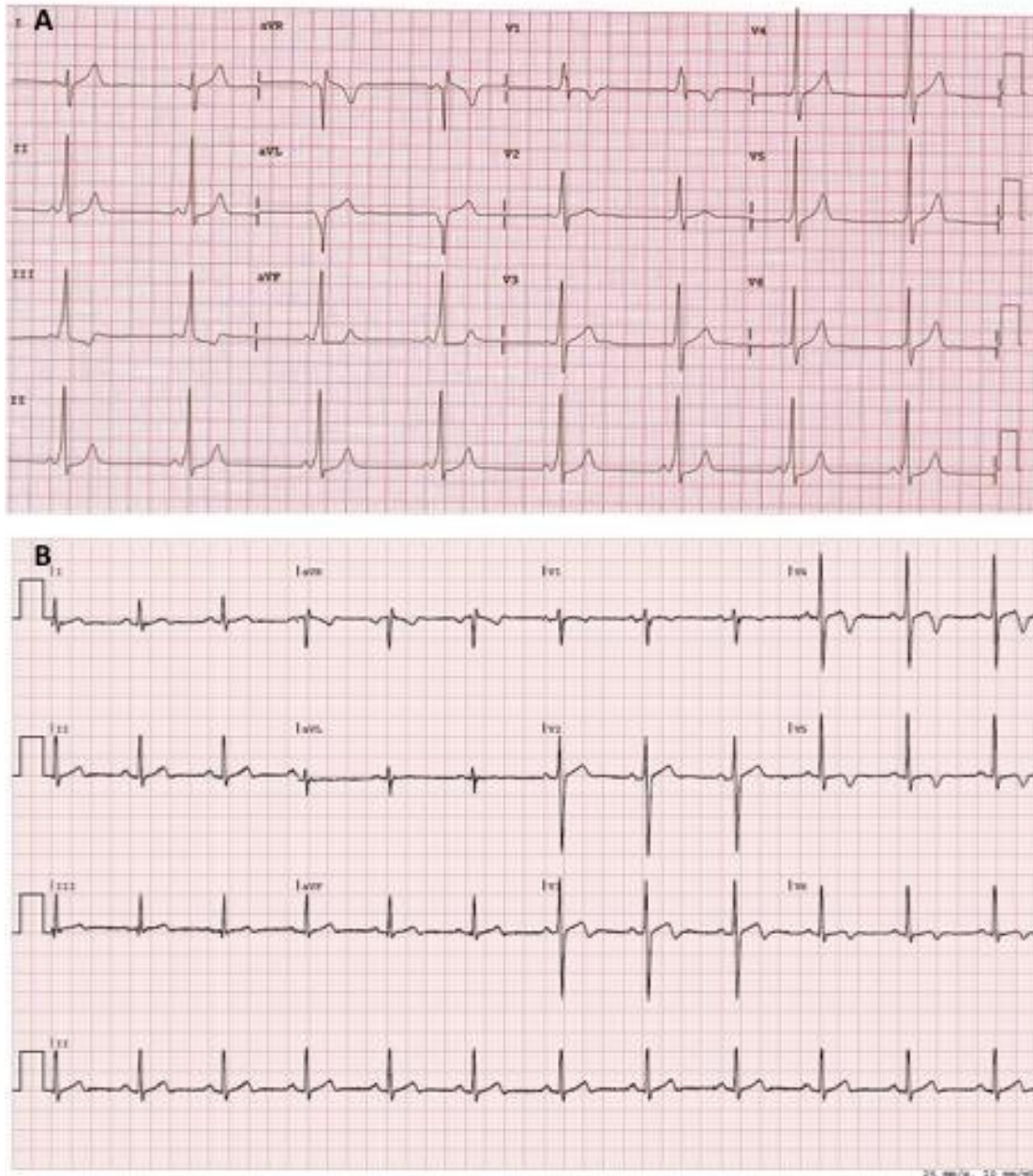


Figure 2: Flow chart of preparticipation screening of 1191 elite rugby players



* Dilated left ventricular cavity (n=3), T-wave inversions in anterior and lateral leads (n=1), bicuspid aortic valve (n=1), mild mitral regurgitation (n=1), mild pulmonary stenosis (n=1), mildly dilated aortic root (n=1), patent ductus arteriosus (n=1).

Figure 3: ECG of athletes with major cardiac abnormality; (a) ECG showing T-wave inversions in athlete with HCM; (b) Pre-excitation and delta wave suggestive of WPW syndrome



HCM, Hypertrophic Cardiomyopathy; WPW, Wolff-Parkinson-White

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