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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

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Aims The 2010 European Society of Cardiology (ESC) guidelines for electrocardiogram (ECG) interpretation in athletes are associated with a relatively high false positive rate and warrant modification to improve the specificity without compromising sensitivity. The aim of this study was to investigate whether non-specific anomalies such as axis deviation and atrial enlargement in isolation require further assessment in highly trained young athletes.

Method and results Between 2003 and 2011, 2533 athletes aged 14–35 years were investigated with 12-lead ECG and echocardiography. Electrocardiograms were analysed for non-training-related (Group 2) changes according to the 2010 ESC guidelines. Results were compared with 9997 asymptomatic controls. Of the 2533 athletes, 329 (13%) showed Group 2 ECG changes. Isolated axis deviation and isolated atrial enlargement comprised 42.6% of all Group 2 changes. Athletes revealed a slightly higher prevalence of these anomalies compared with controls (5.5 vs. 4.4%; $P = 0.023$). Echocardiographic evaluation of athletes and controls with isolated axis deviation or atrial enlargement ($n = 579$) failed to identify any major structural or functional abnormalities. Exclusion of axis deviation or atrial enlargement reduced the false positive rate from 13 to 7.5% and improved specificity from 90 to 94% with a minimal reduction in sensitivity (91–89.5%).

Conclusion Isolated axis deviation and atrial enlargement comprise a high burden of Group 2 changes in athletes and do not predict underlying structural cardiac disease. Exclusion of these anomalies from current ESC guidelines would improve specificity and cost-effectiveness of pre-participation screening with ECG.

Keywords Athlete's Heart • Electrocardiogram • Pre-participation screening

Introduction

Pre-participation cardiac evaluation with 12-lead electrocardiogram (ECG) is efficacious in identifying athletes with cardiomyopathies and electrical disorders.[1] Indeed, there are data suggesting that ECG screening in athletes may reduce the incidence of sudden cardiac death (SCD).[2] Although nationwide cardiac screening of athletes with ECG is considered impractical in most countries,[3] the majority of elite sporting organizations recommend or mandate such practice. High false positive rates are an important limitation of ECG screening in young athletes since electrical manifestations of athletic training may overlap with those observed in disorders implicated in exercise-related SCD, particularly the cardiomyopathies.

In 2010, the ESC published updated recommendations for ECG interpretation in athletes.[4] However, despite these modifications, the burden of high false positive rates persists[5,6] and warrants the need for more specific criteria without compromising sensitivity. Some ECG anomalies that are currently considered abnormal in exercising individuals may be markers of cardiac enlargement or represent normal benign variants that are present with similar

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frequency within the general population. Our preliminary observations show no evidence to support that axis deviation and voltage criteria for atrial enlargement in isolation signify serious cardiac disease. However, both are considered abnormal according to ESC 2010 recommendations for ECG interpretation in young athletes[4] and more recently published Seattle criteria.[7,8] Both recommendations rely on consensus opinion that is largely derived from small or ill-defined populations of young exercising individuals rather than large cohorts of highly trained young athletes.

The aim of this study was to investigate whether axis deviation or atrial enlargement in isolation of other non-training-related ECG changes[4] represents manifestations of cardiac pathology.

Methods

Athletes

Between 2003 and 2011, 2550 athletes aged 14–35 years underwent cardiac evaluation as part of a pre-participation screening program. All athletes competed at either regional or national level. Cardiac evaluation consisted of a self-reported health questionnaire, physical examination, 12-lead ECG, and two-dimensional echocardiography. The athlete's ethnicity was self-assigned. Of the 2550 athletes, 17 were excluded from the analysis due to a BP >140/90 mmHg ($n = 12$), a history of repaired ($n = 2$), or on-going surveillance for established congenital heart disease ($n = 3$). The final population comprised of 2533 athletes.

Controls

The control population consisted of 9997 sedentary volunteers. Controls were recruited from a population screening program offered by the charity Cardiac Risk in the Young, which is aimed at identifying potentially sinister cardiac disorders in young individuals (both athletes and non-athletes). Evaluations were performed at large secondary schools and community centres throughout the UK and consisted of a health questionnaire, physical examination, and 12-lead ECG. Individuals with non-training-related (Group 2) ECG changes (*Figure 1*), which also include those with axis deviation or criteria for atrial enlargement, underwent assessment with echocardiography.

Selection criteria for inclusion in the study comprised age 14–35 years, sedentary life style defined as physical activity ≤3 h per week, absence of a family history of inherited cardiac disorder or premature (≤40 years old) SCD and free of regular medication.

Hypertrophic cardiomyopathy patients

We studied 171 individuals with hypertrophic cardiomyopathy (HCM). Of these, 140 attended two specialist cardiomyopathy clinic and were diagnosed either following referral for symptoms, detection of a cardiac murmur, an abnormal 12-lead ECG, or during cardiovascular assessment of an individual in the context of a family history of cardiomyopathy or SCD. The remainder comprised of 31 young asymptomatic athletes consecutively diagnosed with HCM as part of the Cardiac Risk in the Young pre-participation athlete screening program between 1997 and 2012.

Hypertrophic cardiomyopathy was defined as a maximal left ventricular wall thickness of ≥15 mm in end-diastole in the absence of a cardiac or systemic cause, or a maximal wall thickness <15 mm in the context of electrocardiographic repolarization anomalies and a family history of HCM in a first-degree relative or positive genotype.[9,10]

Twelve-lead electrocardiography

A standard 12-lead ECG was performed using a GE Marquette Hellige (Milwaukee, WI, USA) or Philips Pagewriter Trim III (Bothel, WA, USA) with a paper speed of 25 mm/s and amplification of 0.1 mV/mm as previously described.[11] Heart rate and QRS axis were calculated. Intervals, durations, and voltages were measured in each lead. Electrocardiograms in athletes were interpreted in accordance with the 2010 ESC recommendations (*Figure 1*).[4] All ECGs were re-analysed by the first author on separate occasions for intra-observer variability and read

ESC classification of ECG abnormalities in athletes

Group 1 (training-related)	Group 2 (training unrelated)
Sinus bradycardia	T-wave inversions
First-degree AV block	ST-segment depression
Incomplete RBBB	Pathological Q-waves
Early repolarization	Left atrial enlargement
Isolated QRS voltage criteria for LVH	Right atrial enlargement
	Left-axis deviation
	Right-axis deviation
	Right ventricular hypertrophy
	Ventricular pre-excitation
	Complete LBBB or RBBB
	Long-QT or short-QT interval
	Brugada-like early repolarization

Figure 1 European Society of Cardiology (ESC) classification of ECG abnormalities in athletes according to training related (Group 1) and non-training-related (Group 2) changes.[4] Abbreviations: ESC, European Society of Cardiology; LVH, left ventricular hypertrophy; LBBB, left bundle branch block; RBBB, right bundle branch block.

independently by two authors (M.P. and S.S.) who are highly experienced in sports cardiology and the diagnosis of cardiomyopathies.

The QT was corrected for heart rate using the Bazett's formula.[12] The Sokolow–Lyon voltage criterion[13] was used to define left ventricular hypertrophy. T-wave inversion in ≥2 contiguous leads, excluding leads V1 aVR and III, was considered significant. Left axis deviation was defined as ≤−30° and right axis deviation (RAD) as ≥+120°. Left atrial enlargement was defined as a negative portion of the *P*-wave in lead V1 ≥ −0.1 mV in depth and ≥0.04 s in duration or ≥120 ms in duration in lead II. Right atrial enlargement was defined as a *P*-wave amplitude ≥0.25 mV in leads II and III or V1.

Electrocardiograms in athletes were broadly categorized as training-related changes (Group 1) or those requiring further investigations (Group 2), in accordance with the 2010 ESC recommendations on ECG interpretation in athletes (*Figure 1*).[4] Athletes with a combination of Group 1 and Group 2 ECG patterns were designated in the Group 2 category.

Definition of isolated axis deviation/atrial enlargement

Isolated left axis deviation (LAD) or RAD was considered to be present when it occurred as a single anomaly in an otherwise normal ECG or, in addition to recognized Group 1 ECG changes.[4] Similarly, voltage criterion for left atrial enlargement (LAE) or right atrial enlargement (RAE) was considered to be present when it occurred as a single anomaly, or, in addition to recognized Group 1 ECG changes.[4]

Echocardiography

Two-dimensional echocardiography was performed using either the Philips Sonos 7500, Philips iE33, or Philips CPX50 (Bothel, WA, USA) and Accuson Computed Sono-graph 128XP/10c (San Jose, CA, USA). Standard views were obtained and analysed according to the protocols specified by European Society of Echocardiography.[14] Left ventricular wall thickness was measured in 2-D parasternal short axis, at the level of the mitral valve and papillary muscles, the greatest measurement being defined as the maximal left ventricular wall thickness. Left atrial and ventricular diameters were measured from the parasternal long-axis view using the 2-D images. Right ventricular dimensions were measured in apical four-chamber views. Right ventricular outflow tract dimensions were measured in parasternal short-axis view at the aortic valve level. Assessment of diastolic function included pulsed wave Doppler across the mitral valve and tissue Doppler velocity imaging of the septal and lateral mitral valve annulus and free wall tricuspid valve annulus.

Echocardiographic studies were saved to compact discs as numeric files to generate anonymity. All cardiac measurements were repeated on a separate occasion by the first author (S.G.) and independently by an experienced cardiac physiologist (M.R.).

Ethical approval/consent

The National Research Ethics Service, Essex 2 Research Ethics Committee, granted ethical approval in the UK. Written consent was obtained from individuals aged 16 years or over and from a parent/guardian for those aged <16 years.

Statistical analysis

Statistical analyses were performed using STATA version 11.1. (StataCorps, TX, USA). Variables were tested for normality using the Kolmorgorov–Smirnov test. Values are expressed as either mean ± standard deviation (SD) or percentages, as appropriate. Differences between group means were compared using independent *t*-tests or Mann–Whitney *U*-tests (for normally and non-normally distributed variables, respectively). Analysis of variance (with Bonferroni *post hoc* correction) was used to compare multiple groups. The χ^2 test or Fisher's exact test was used as appropriate to test group differences of proportions.

Logistic regression was used to determine which of the following variables were associated with isolated axis deviation/atrial enlargement: ethnicity; age, intensity (hours of duration) of exercise, and body surface area. A multivariate model was fitted which included variables identified as statistically significant in the univariate analyses. Statistical significance was defined as a two-tailed *P*-value of <0.05 throughout.

Results

Athletes

The athletes were aged 21.8 ± 5.7 years. The majority were male and of Caucasian ethnicity (*Table 1*). Athletes participated in 31 different sporting disciplines and trained for an average period of 18.7 h per week. None reported symptoms indicative of cardiovascular disease. None took regular medications or reported a family history of cardiomyopathy or premature SCD. All athletes had a blood pressure of ≤140 mmHg systolic and ≤90 mmHg diastolic.

Controls

The control group was younger than the athletes with a smaller body surface area (*Table 1*). As with the athletic group, the majority was male, Caucasian, and none exhibited symptoms suggestive of cardiovascular disease.

Prevalence of Group 1 and Group 2 electrocardiogram changes

Almost three quarters (72.9%) of athletes exhibited Group 1 ECG changes and 14.1% demonstrated normal ECGs. Group 2 ECG changes were identified in 13% of athletes (*Figure 2*). A combination of Group 1 and 2 changes was observed in 9.5% of the athlete. A significant proportion of controls (7.6%) revealed Group 2 changes.

The combination of LAD, RAD, LAE, and RAE accounted for 42.6% of all Group 2 changes in athletes. Athletes displayed a higher prevalence of isolated axis deviation and criteria for atrial enlargement compared with controls when the four anomalies were combined ($n = 139$; 5.5% vs. $n = 440$; 4.4%; $P = 0.023$). Specifically, athletes showed a higher prevalence of LAD and LAE compared with controls (1.46 vs. 0.96%; $P = 0.028$ and 2.13 vs. 1.37% respectively), whereas there were no significant differences in the prevalence of RAD and RAE between the groups (1.11 vs. 1.10%; $P = 0.983$ and 0.83 vs. 0.92%; $P = 0.664$, respectively).

Echocardiographic comparison of athletes with and without left or right-axis deviation or atrial enlargement

Athletes with LAD or LAE exhibited larger left atrial and ventricular dimensions compared with athletes with a normal ECG and those with Group 1 changes. There were no appreciable differences in the number of athletes with cardiac dimensions exceeding predicted upper limits between the two groups. In contrast, there were no differences in cardiac dimensions between athletes with RAD or RAE compared with athletes with normal or Group 1 ECG changes.

Table 1 Comparison of demographics between athletes and controls

Parameters	Athletes ($n = 2533$)	Controls ($n = 9997$)	Patients with hypertrophic cardiomyopathy ($n = 171$)	*P*-value
Age (years)	21.8 ± 5.7*	18.5 ± 5.5	46.3 ± 17.6	<0.001
BSA (m^2)	2.0 ± 0.3*	1.8 ± 0.2	–	<0.001
Systolic BP (mmHg)	120 ± 12.6*	115 ± 14.9	132 ± 10.2	<0.001
Sex, *n* (%)				
Males	72.2	70.9	68.4	<0.001
Females	27.8	29.0	31.6	
Ethnicity, *n* (%)				
Caucasian	82.4	96.3**	83.6	<0.001
African/Afro-Caribbean	4.3	0.8	16.4	
Asian	4.0	1.1	–	
Mixed race	4.7	1.0	–	
Other ethnicity	4.4	0.7	–	
Hours of training (per week)	18.7 ± 7.7*	2.8 ± 1.27	–	<0.001

Data expressed as mean ± SD.
BP, blood pressure; BSA, body surface area.
*Greater than in controls ($P < 0.05$).
**Greater than in athletes ($P < 0.05$).

Diagnosis of cardiac abnormalities in athletes and controls

We identified 3 (0.1%) cases of HCM in the entire athlete cohort and 1(0.13%) out of 760 controls investigated with echocardiography. Three cases revealed ST segment depression and T wave inversion in the lateral leads and one case showed a plethora of abnormalities including bi-atrial enlargement, left axis deviation, and ST segment depression in the inferior leads.

None of the athletes with isolated axis deviation or voltage criteria for atrial enlargement showed features consistent with cardiomyopathy. The prevalence of commonly recognized congenital cardiac abnormalities, notably patent foramen ovale, bicuspid aortic valve, and mitral valve prolapse in this group was 3, 0, and 1%, respectively, and similar to that observed in athletes with Group 1 ECG changes or a normal ECG (4, 1, and 1.3%, respectively). The total prevalence of minor congenital abnormalities in controls with isolated axis deviation or voltage criteria for atrial enlargement was 4%. Indices of LV systolic and diastolic function were normal in all subjects with isolated axis deviation or voltage criteria for atrial enlargement (*Table 2*).

Prevalence of axis deviation or voltage criteria for atrial enlargement in patients with hypertrophic cardiomyopathy

Patients with HCM were older than the athletes and controls and 65% were symptomatic (*Table 1*). Seventy-one patients (41.5%) were aged <40 years old (29.4 ± 6.2; range 14–39 years old) of which 31 (43.7%) played regular competitive sport at the regional level at the time of diagnosis. A normal ECG or Group 1 changes in isolation were rare (3.5%) in HCM patients.

Of the total HCM cohort, 85 (49.7%) patients displayed either axis deviation and/or voltage criterion of atrial enlargement. Specifically, 63 (36.8%) revealed LAE, 22 (12.9%) LAD, 15 (8.8%) RAE, and 8 (4.7%) showed RAD. Of these, 76 (89%) demonstrated co-existing electrocardiographic changes that are usually considered pathological,[15] notably T wave inversion (68%), ST-segment depression (33%), pathological q waves (31.8%), and complete bundle branch block (15.5%).

Only nine patients (5.3%) exhibited either isolated axis deviation ($n = 3$) or voltage criterion for atrial enlargement ($n = 6$). Of these, six patients (66.7%) expressed mild morphology with a maximal wall thickness ≤16 mm (*Table 3*). The prevalence of isolated axis deviation or atrial enlargement was similar in HCM patients aged <40 years old and those who were older ($n = 3$, 4.2% vs. $n = 6$, 6%; $P = 0.87$). Although a significant proportion of the asymptomatic young athletes with HCM revealed one or more of T wave inversion (90%), ST segment depression (46%), and pathological q waves (18%), none exhibited isolated axis deviation or atrial enlargement.

Determinants of left axis deviation and left atrial enlargement in athletes

Univariate analysis demonstrated that males were more likely to show LAD or voltage criterion for LAE than females (OR: 1.73 95% CI 1.40, 2.13, $P < 0.001$) as were those who trained for ≥20 h per week compared with those training for ≤5 h per week (OR: 1.31 95% CI 1.01, 1.70, $P = 0.04$). There was no significant association between LAD or LAE and age, body surface area, or ethnicity.

Multivariate analysis revealed that after adjusting for sex, ≥20 h of training per week remained an independent predictor of individuals exhibiting LAD or LAE compared with individuals training for ≤5 h per week (adjusted OR: 1.32 95% CI 1.02, 1.72, $P = 0.04$).

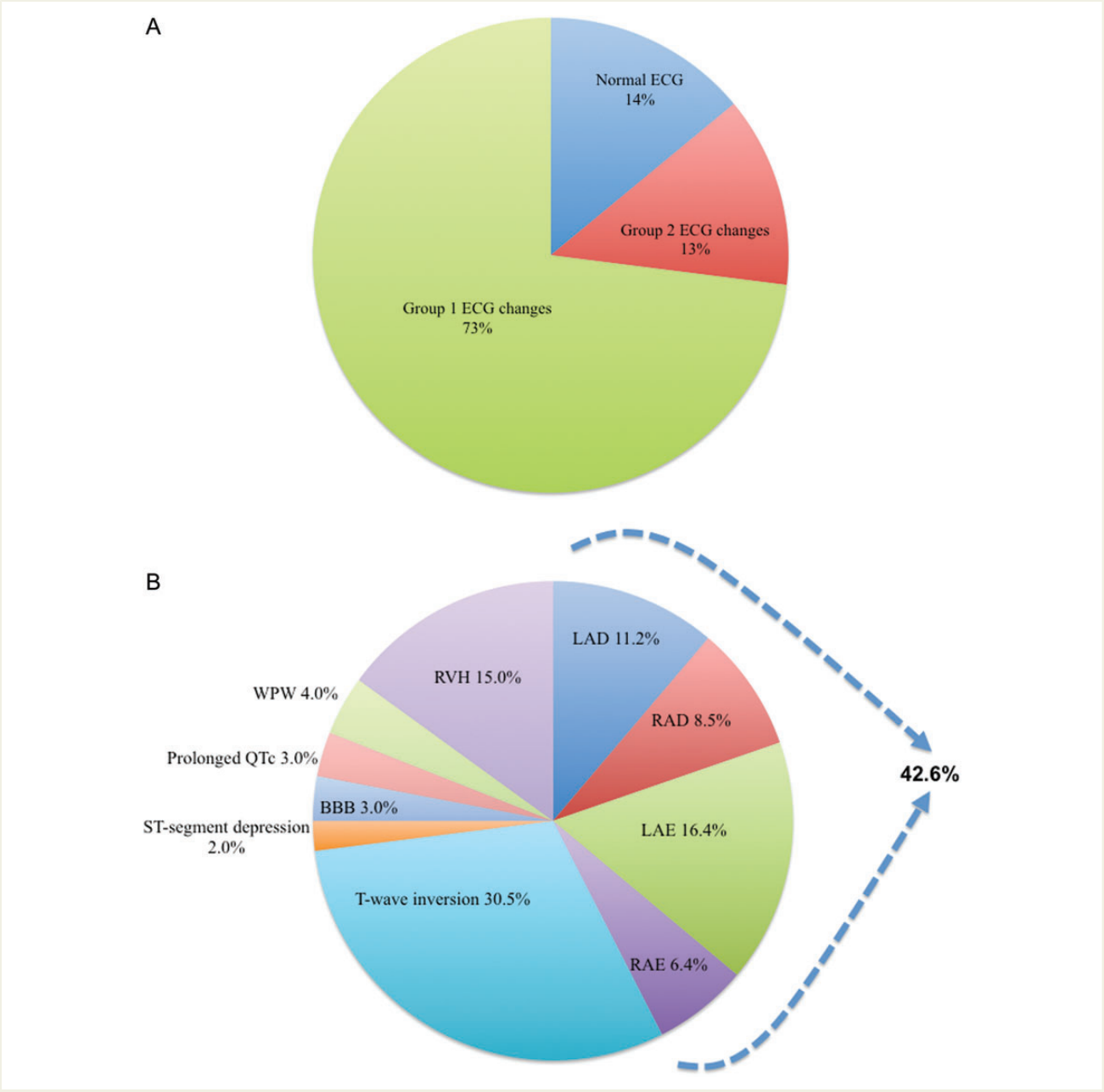

Figure 2 (*A*) Pie chart demonstrating the prevalence of training-related (Group 1) and non-training-related (Group 2) ECG changes in athletes. (*B*) Breakdown of ECG anomalies comprising Group 2 changes. *Abbreviations: BBB, bundle branch block; ECG, electrocardiogram; LAD, left axis deviation, LAE, left atrial enlargement, QTc, corrected QT >470 for males and >480 for females; RAD, right axis deviation; RAE, right atrial enlargement; RVH, right ventricular hypertrophy; WPW, Wolf Parkinson White pattern.

Applications of the revised criteria to the current 2010 ESC guidelines

The positive and negative predictive values for isolated axis deviation or atrial enlargement for significant structural heart disease were 1.4 and 94%, respectively. Omission of these anomalies from the current ESC recommendations in our cohort reduced the false positive rate from 13 to 7.5% and resulted in an increased specificity from 90 to 94% with a minimal reduction in sensitivity from 91 to 89.5%.

Intra-observer variability/inter-observer variability

There were no cases of disparity with respect to isolated axis deviation or atrial enlargement during re-analysis of the ECG by first author (S.G.), and independent reviewer (S.S.) which translated to a kappa (measurement of agreement) = 1.00 ($P < 0.0001$). There were no cases of disparity with respect to echocardiographic evaluation of individuals exhibiting axis deviation or atrial enlargement by the first author (S.G.) and an independent reviewer (M.R.).

Discussion

In this study, 13% of athletes exhibited Group 2 ECG criteria. Of these, over two-fifths (42%) revealed one of either axis deviation or voltage criterion for atrial enlargement in association with normal or Group 1 ECG patterns. Left axis deviation and voltage criterion for LAE were more common in athletes that trained for ≥20 h per week compared with those training ≤5 h per week. Echocardiographic investigation of athletes demonstrating axis deviation or atrial enlargement failed to demonstrate a cardiomyopathy or other major structural abnormalities. Our findings are not dissimilar to those from an American study of 508 university students,[6] which revealed that of the 49 athletes with abnormal ECGs, at least 29 (59%) exhibited either voltage criteria for LAE alone or in combination with large QRS complexes. Subsequent echocardiography revealed a structurally normal heart or findings consistent with athletic training.

Our athletes displayed a slightly higher prevalence of LAD and LAE compared with controls. The atrial and ventricular dimensions in athletes fulfilling criteria for LAD and LAE were greater than those with normal ECG or Group 1 ECG patterns, suggesting that the presence of one of LAD or LAE in association with other Group 1 ECG changes may reflect a measure of physical conditioning. Previous studies in young athletes provide support for our notion; Sharma *et al.*[16] revealed that 46% of adolescent Caucasian athletes (14–18 years old) exhibited axis deviation or atrial enlargement compared with 9.2% of controls and Papadakis *et al.*[10] demonstrated that 16% of adult black athletes exhibited either axis deviation or criteria for atrial enlargement in the absence of a structural cardiac disorder. The differences between these two athletic populations and the cohort under examination, which consisted predominantly of young adult Caucasian athletes, suggest that an athlete's demographics are strong influencing factors on the 12-lead ECG. Nevertheless, the inability to detect a cardiac abnormality in all of these athletes is a common theme.

Comparisons with current recommendations

The 2010 ESC recommendations on ECG interpretation are generally based on consensus panel opinion rather than scientific evidence.

Table 2 Comparison of echocardiographic parameters in athletes with Group 1 plus left axis deviation or left atrial enlargement and athletes with Group 1 or normal electrocardiograms

Parameters	Athletes with Group 1 plus LAD or LAE ($n = 91$)	Athletes with Group 1 or normal ECG ($n = 1067$)	*P*-value
Ao (mm)	30.6 ± 4.3	27.9 ± 4.3	<0.001
LA (mm)	34.2 ± 4.9	30.6 ± 4.3	<0.001
LVED (mm)	54.0 ± 5.5	51.0 ± 5.4	<0.001
Max-LVWT (mm)	10.0 ± 1.2	9.4 ± 1.4	<0.001
LV FS (%)	52.4 ± 5.3	50.6 ± 5.1	0.001
E/A	2.1 ± 0.7	2.2 ± 0.7	0.191
E′ septal wall (cm/s)	12.9 ± 2.7	12.9 ± 2.5	1.000
E′ lateral wall (cm/s)	18.6 ± 4.8	18.9 ± 4.2	0.518
E/E′ (septal wall)	7.5 ± 2.1	7.2 ± 1.8	0.133
E/E′ (lateral wall)	5.2 ± 1.5	4.9 ± 1.2	0.025
RA (cm^2)	15.9 ± 1.6	14.9 ± 4.0	0.018
RVED1 (mm)	40.1 ± 5.4	37.9 ± 6.3	0.001
RVOT1 (mm)	33.5 ± 3.8	30.8 ± 5.5	<0.001

Data are expressed as mean ± SD.
Ao, aortic annulus diameter; E/A, ratio of early diastolic mitral valve peak inflow velocity to late diastolic mitral valve inflow velocity; E′, early diastolic annular peak velocity (septal and mitral annulus); LA, left atrial diameter; LAD, left axis deviation; LAE, left atrial enlargement; LVED, LV end-diastolic diameter; LV FS, LV fractional shortening; LVH, left ventricular hypertrophy; Max-LVWT, maximal left ventricular wall thickness in end-diastole; RA, right atrial area; RVED, right ventricular end-diastolic dimension; RVOT, right ventricular outflow tract.

Table 3 Demographic data on the nine patients with hypertrophic cardiomyopathy and isolated axis deviation or atrial enlargement

HCM patient	Age (years)	Sex	Family history of SCD/HCM	Isolated ECG change	Max-LVWT (mm)	Pattern of LVH	LVED (mm)	LA (mm)	LVM (g)	E/A	LVOT gradient >30 mmHg
1	19	M	Yes	RAE	15	Septal	49	40	243	1.4	No
2	66	F	No	LAE	13	Concentric	49	49	243	1.2	No
3	43	M	No	LAE	15	Concentric	40	37	188	1.3	No
4	76	M	No	LAE	16	Septal	42	37	189	1.4	No
5	32	M	No	LAE	21	Septal	50	46	332	1.3	Yes
6	81	M	No	LAE	19	Septal	31	38	227	0.8	No
7	41	F	No	LAD	15	Septal	50	36	224	1.1	No
8	51	M	No	LAD	15	Septal	52	44	282	1.6	No
9	37	M	Yes	RAD	23	Septal	49	40	–	1.7	No

E/A, ratio of early diastolic mitral valve peak inflow velocity to late diastolic mitral valve inflow velocity; ECG, electrocardiogram; LA, left atrial diameter; LAD, left axis deviation; LAE, left atrial enlargement; LVED, LV end-diastolic diameter; LVH, left ventricular hypertrophy; LVM, left ventricular mass; LVOT, left ventricular outflow tract; Max-LVWT, maximal left ventricular wall thickness in end diastole; SAM, systolic anterior motion of the mitral valve.

Although the newly published Seattle recommendations are a laudable and innovative incentive for the education of sports physicians,[7,8] they are almost identical to the ESC recommendations and classify axis deviation or voltage criteria for atrial enlargement in the abnormal category. In the context of this study, application of the Seattle criteria[7,8] would lead to a marginally lower false positive rate (0.4%) than the 2010 ESC recommendation based on the slight differences in the definition of a RAD ($>+115^{0\ 8}$ vs. $>+110^{0\ 4}$, respectively).

Conversely, an inquisitive analysis of ECG interpretation in young athletes by Uberoi *et al.*[17] led to the recommendation that isolated axis deviation does not warrant further assessment in the absence of hypertension or pulmonary heart disease. In contrast, isolated criteria for atrial enlargement in asymptomatic junior athletes only warrant further investigation in the presence of abnormal physical examination, whereas the same anomaly in adult counterparts is an indication for investigation irrespective of symptoms or physical signs. The report was informed by a respectable consensus panel in circumstances where evidence was sparse and carries a more pragmatic flavour than the 2010 ESC[4] and Seattle recommendations in some respects.[7,8] The discrepancies between these recommendations are an understandable source of confusion particularly as several members of the expert panel are common to all three. In this regard, our findings from a large cohort of regional and national athletes provides a more concrete scientific basis for future updates on the interpretation of the athlete's ECG. Furthermore, our concept is associated with a 42% reduction in the false positive rate in athletes without jeopardizing specificity, which is a welcome initiative among physicians.

Association with disease

We investigated 579 athletes and controls with isolated axis deviation or atrial enlargement and failed to detect a major structural abnormality in any individual. The main concern with these anomalies is the presence of HCM. Whereas these anomalies, particularly LAE, were common in HCM, they co-existed with a plethora of other pathological ECG patterns.

Our reliance on ECG patterns in predominantly middle aged patients with HCM may be considered to be inapplicable to young competitive athletes harbouring HCM. Intuitively, such individuals would be expected to exhibit a milder phenotype including a higher prevalence of minor ECG anomalies such as isolated axis deviation and atrial enlargement than observed in our HCM cohort. However, there is no association between functional capacity and ECG patterns. Indeed, it is our experience that most athletes with HCM exhibit the apical variant of the disease, which may be considered a milder form but is associated with profound repolarization changes. In this study, the prevalence of isolated axis deviation and atrial enlargement did not differ in young patients with HCM compared with those aged >40 years old (4.2 vs. 6%) and paradoxically, none of the 31 competitive athletes with HCM showed axis deviation or atrial enlargement in isolation.

Cost issues

On the basis that only 3 (4.2%) HCM patients aged <40 years old showed isolated axis deviation or atrial enlargement, we calculated that exclusion of these anomalies from the abnormal category would fail to detect just 1 case of HCM for almost 12 000 young athletes screened. Although the failure to detect even a single case of HCM has potentially serious implications, we noted that most HCM patients with these anomalies expressed morphologically mild disease, which is generally associated with a favourable prognosis. In the current financial climate, it is difficult to ignore the issue of cost.[18] The minimal cost of echocardiography following an abnormal ECG in most Western European countries is €295 (€410 in the UK). Based on our data, an additional 660 athletes would require investigation with echocardiography for isolated axis deviation or voltage criterion for atrial enlargement resulting in a substantial increase in costs per case of HCM identified.

There is a theoretical possibility that axis deviation may be a harbinger of cardiac conduction tissue disease; however, the Framingham study demonstrated that inclusion of ECG variables in the prediction of SCD had little effect on identifying those individuals at risk of sudden unexpected death; in particular, the QRS axis was unrelated to SCD.[19]

Conclusion

The presence of axis deviation or voltage criteria for atrial enlargement on the 12-lead ECG rarely predicts pathology in young athletes. Exclusion of these anomalies from the abnormal category in asymptomatic athletes without a family history of premature cardiac disease, or abnormal physical findings, would increase specificity from 90 to 94% with a minimal sacrifice in ECG sensitivity from 91 to 89.5%. The substantial reduction in the false positive rate from 13 to 7.5% has the potential for huge cost savings, which bodes well for financially constrained sporting organizations. The data have important implications for future recommendations on interpretation of the athlete's ECG, particularly if they can be replicated in different large cohorts of athletes by other reputable organizations.

Limitations

Our conclusions are based on an observational cross-sectional study; therefore, it is impossible to exclude with certainty that a proportion of young individuals with isolated axis deviation or voltage criterion for atrial enlargement may develop cardiac disease in later life. Nevertheless, neither the authors nor any of the major scientific bodies would consider repeat assessments or long-term follow-up of asymptomatic individuals with such anomalies in the absence of symptoms, physical signs, or a relevant family history. For practical purposes, we relied on echocardiography rather than cardiac MRI to investigate structural heart disease; therefore, we may have failed to detect subtle forms of cardiomyopathy in a small proportion of individuals. Finally, the prevalence of axis deviation and atrial enlargement is predominantly derived from the young adult Caucasian population and the results of this study should be applied with caution to veteran (>40 years old) athletes and non-Caucasian athletes.

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