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Title: Electrocardiographic changes following six months of long-distance triathlon training in previously recreationally active individuals

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☆ All authors mentioned above take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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Electrocardiographic Changes Following Six Months of Long-Distance Triathlon Training in Previously Recreationally Active Individuals

ABSTRACT

Background: Clinical electrocardiographic (ECG) guidelines for athlete's heart are based upon cross-sectional data. We aimed to longitudinally evaluate the influence of endurance training on the ECG and compare the prevalence of ECG abnormalities defined by contemporary criteria.

Methods: A group of 66 training-naïve individuals completed a six-month training programme with resting ECGs and cardiopulmonary exercise tests performed at baseline, two and six months. Data were analysed using repeated measures analysis of variance and the prevalence of ECG abnormalities compared between proposed criteria.

Results: Maximal oxygen consumption increased from 45.4 ± 7.1 to 50.3 ± 7.1 ml·kg⁻¹·min⁻¹ ($p < 0.05$) pre-to-post training. ECG changes included, bradycardia (60 ± 12 vs. 53 ± 8 beats·min⁻¹; $p < 0.05$), shorter P wave duration (106 ± 10 vs. 103 ± 11 ms; $p < 0.05$), reduced QTc (413 ± 27 vs. 405 ± 22 ms; $p < 0.05$), and increased left ventricular Sokolow-Lyon index (2.45 ± 0.66 vs. 2.62 ± 0.78 mV; $p < 0.05$). 85% of individuals showed ≥ 1 'training-related' ECG finding at six months vs. 68% at baseline. Using the 2013 Seattle Criteria, 4 ECGs were 'abnormal' at baseline and 3 at month six vs. 2 at baseline and 1 at month six, using the 2017 International Consensus. Prevalence of 'borderline' findings did not increase with training (11% at baseline and six months).

Conclusion: Six-months endurance training leads to a greater prevalence of 'training-related' but not 'borderline' or 'training-unrelated' ECGs. 'Borderline findings' may not necessarily represent training-related cardiac remodelling in novice athletes following a six-month training intervention.

Keywords: Electrocardiography, endurance, exercise, athlete's heart.

Introduction

Cardiovascular-related sudden death is the leading cause of death in athletes during sport and exercise (Harmon et al., 2015; Maron, Doerer, Haas, Tierney, & Mueller, 2009). Most cardiovascular disorders responsible for sudden cardiac death during sport may be identified using electrocardiogram (ECG)

screening criteria (Harmon et al., 2015; Maron et al., 2009). These criteria separate findings into ‘training-related’ or ‘training-unrelated’ categories, with the latter considered abnormal. Athletes with underlying cardiac pathology, reflected in the ECG by ‘training-unrelated’ abnormalities, are at an increased risk of sudden death and of developing life threatening conditions such as arrhythmias and fibrillations (Corrado et al., 2010). The ECG, therefore, is an important screening tool for sports pre-participation (Corrado et al., 2005; Harmon et al., 2015).

Cardiac remodelling in response to repetitive exercise training includes adaptive changes in chamber dimensions (Arbab-Zadeh et al., 2014; Baggish, Wang, et al., 2008), wall thickness (Arbab-Zadeh et al., 2014) and function (Baggish, Wang, et al., 2008; Baggish, Yared, et al., 2008; Weiner et al., 2010) to accommodate the associated haemodynamic alterations that accompany exercise. Furthermore, several large cross-sectional studies have also described ECG findings in endurance athletes, which are different to untrained counterparts (Brosnan et al., 2014; Pelliccia et al., 2007; Pelliccia et al., 2000) and, importantly, can resemble pathology (Kim & Baggish, 2016). In 2017, an international consensus document was published, detailing updated criteria for ECG interpretation in athletes (Drezner et al., 2017), which aimed to improve the specificity and sensitivity of previous guidelines, such as the 2013 Seattle criteria (Drezner et al., 2013). The principal changes implemented in the new criteria include the re-categorisation of several findings from ‘training-unrelated’ abnormalities to ‘borderline’. According to the guidelines, ‘borderline’ findings in isolation do not require further investigation and include left axis deviation, left atrial enlargement, right axis deviation, right atrial enlargement, and complete right bundle branch block (RBBB). The data used to formulate the new consensus statement and previous criteria, however, rely heavily on cross-sectional investigation, since there are few longitudinal studies of ECG changes in response to training. Thus, we performed an explorative longitudinal investigation of ECG changes over a period of high-volume endurance training in a previously training-naïve cohort with two aims. Firstly, to assess longitudinal changes to the ECG in response to endurance training, and secondly, to determine whether these ECG changes support the revisions made to the recent 2017 international consensus criteria (Drezner et al., 2017). It was hypothesised that ‘training-related’ ECG changes would increase following six months

of endurance training. Secondly, it was expected that quantitative 'borderline' ECG parameters would be increased, including QRS duration, QRS axis, P-wave duration and P-wave amplitude, together with an increased prevalence of 'borderline' criteria comprising QRS axis deviation, atrial enlargement and complete RBBB as defined by the international consensus (Drezner et al., 2017).

Methods

Study Design

Recreationally active participants (n = 110) with no previous exposure to ultra-endurance exercise or high-level competitive sport were recruited, with a target of completing a long-distance triathlon (3.86 km swim, 180.25 km cycle and 42.20 km run). Individuals with a maximal volume of oxygen consumption ($\dot{V}O_{2\max}$) outside the range of 30 to 50 ml·kg⁻¹·min⁻¹ (female) and 35 to 55 ml·kg⁻¹·min⁻¹ (male) were excluded as well as those with known cardiovascular abnormality, determined from participant history and 12-lead ECG screening, assessed by a clinical cardiologist. None of the participants smoked, used recreational drugs, or had significant medical problems. Sixty-six (53 males, 13 females) participants were included in the final analysis after accounting for apparently abnormal ECG at pre-screen (n = 2), insufficient data (n = 9) and participant withdrawal (n = 33). All, individuals provided written informed consent together with a letter from their respective general practitioners. On all testing days, participants arrived at least 2 hours fasted and abstained from caffeine (≥12h), alcohol (≥12h) and exercise (24h) prior to assessment. All testing took place within the Human Performance Laboratory at the University of Hertfordshire. Demographic information including age, sex, height and body mass were obtained for each participant and body composition was estimated using bioelectrical impedance scales (Tanita BC418MA, Tokyo, Japan).

Anthropometry, resting 12-lead ECG, automated blood pressure, cycling lactate threshold and $\dot{V}O_{2\max}$ tests were performed sequentially. This protocol was approved by the University of Hertfordshire Life and Medical Sciences Ethics Committee (LMS/SF/UH/00011) and conforms to the ethical guidelines of the 1975 Declaration of Helsinki.

Training Programme

Participants were provided with a structured training programme by an accredited physiologist, which contained daily instructions detailing exercise type, duration and intensity, based on rating of perceived exertion (RPE) or heart rate (Borg, 1982). Initially, participants trained 7 to 8 times per week for 45 to 90 mins per session; training volume was incrementally progressed, such that by six months participants were completing ~13 hours per week with at least one “long” session. For all long sessions, heart rate (HR) was maintained at ~65% of maximal HR (HR_{max}). The intensity of training sessions was quantified as either easy (60 to 70% HR_{max}), moderate (70 to 80% HR_{max}) or hard (80 to 90% HR_{max}), allowing periods of stress to be followed by periods of recovery. Participants reported completion of at least 80% of the prescribed training load, quantified as the sum of session RPE and volume (in minutes).

To determine the specific training zone intensities, heart rate and RPE at lactate threshold (Biosen C line analyser, EKF-diagnostic GmbH, Barleben, Germany) and $\dot{V}O_{2max}$ (MetaLyzer 3B, Cortex Biophysik GmbH Leipzig, Germany) were measured every two months. Lactate threshold was identified as the first ‘break-point’ in blood lactate accumulation during an incremental stepped protocol (Kindermann, Simon, & Keul, 1979; Winter, Jones, Davison, Bromley, & Mercer, 2006), and $\dot{V}O_{2max}$ was defined as the highest oxygen uptake, averaged over 60 s during an incremental ramped cycling test to exhaustion (Astorino, 2009).

12-Lead Electrocardiography

12-lead ECGs were collected at baseline, two months and six months into the training programme and were performed in accordance with the Mason-Likar (Mason & Likar, 1966) modified ECG formation using a Custo Med electrocardiogram (GmbH, Munich, Germany), with participants rested in the supine position for at least 10 minutes. Following ECG acquisition, automated blood pressure recordings were obtained in duplicates with the participant in the rested supine position (Omron M2 Basic, Omron Healthcare, Milton Keynes, UK). Each lead of the ECG was analysed with respect to its interval and voltage in line with the current guidelines for athlete’s ECGs, using machine generated metrics. QTc interval was calculated using Bazett’s formula and left ventricular hypertrophy (LVH; S

in V1 + R in V5 or V6) and right ventricular hypertrophy (RVH; R in V1 + S in V5) using Sokolow and Lyon voltage criteria (Sokolow & Lyon, 1949, 2001). The same individual assessed the accuracy of electrode placement at each time point. Subsequent to the initial screening, all ECG analyses were conducted retrospectively in a single batch with the investigator blinded to time point. All ECGs were interpreted using the Seattle criteria (Drezner et al., 2013) and the revised international criteria (Drezner et al., 2017) by TD and checked by AD and AB.

Statistical Methods

Normality of distribution for all variables was assessed using Kolmogorov-Smirnov tests, combined with skewness and visual analysis of Q-Q plots of the expected vs. observed standardised residuals. Slight violations in normality of distribution were deemed tolerable due to the robustness of the statistical procedure used (Field, 2009). A repeated-measures analysis of variance (ANOVA) was performed on all quantifiable ECG variables with Sidak post-hoc pairwise analysis conducted where there was a significant main effect. Data analysis was performed using the Statistical Package for the Social Sciences (SPSS) software version 23 (SPSS Inc., Illinois, United States of America). Significance was determined by an alpha level of $p < 0.05$ and variables reported as mean \pm standard deviation.

Results

Participant Characteristics

The cohort was comprised of predominantly males (80%) aged 36 (range: 18 to 49 years). Prescribed training load and reported average training load increased from baseline (1789 vs 1948 arbitrary units; AU) to month two (2568 vs 2628 AU) and month six (2961 vs 2921 AU). Body mass did not significantly change across time; however, body fat was significantly reduced at month six versus baseline and month two ($p < 0.05$; Table 1). Systolic and diastolic blood pressure decreased at month two in comparison with baseline values ($p < 0.05$) but no significant difference in blood pressure was found between baseline and month six. $\dot{V}O_{2\max}$ was significantly increased across time and was

greatest at month six ($p<0.05$). $\dot{V}O_2$ and heart rate at lactate threshold increased at month two and month six and power output at lactate threshold was significantly increased by month six ($p<0.05$).

[Table 1 near here]

Standard ECG measurements

Group mean values for conventional ECG measurements are shown in (Table 2). Compared to baseline (60 ± 12 beats \cdot min $^{-1}$), heart rate was decreased at two months (57 ± 10 beats \cdot min $^{-1}$; $p<0.001$) and six months (53 ± 8 beats \cdot min $^{-1}$). QRS axis remained unchanged, as did QRS duration. P-wave duration remained unchanged from baseline (106 ± 10 ms) to month two (105 ± 10 ms) but was significantly reduced at six months of training (103 ± 11 ms; $p<0.05$). P-wave amplitude across leads II, III and aVF remained unchanged across time ($p<0.05$). QT interval increased progressively from baseline (417 ± 35 ms) to month two (421 ± 35 ms) and month six (435 ± 32 ms; $p<0.05$), and QTc decreased from 413 ± 27 ms at baseline to 406 ± 22 ms and 405 ± 22 ms at month two and six, respectively ($p<0.05$). Sokolow-Lyon criteria for LVH did not change from baseline (2.45 ± 0.66 mV) to month two (2.55 ± 0.76 mV) but was significantly elevated at month six (2.62 ± 0.78 mV; $p<0.005$; Figure 1). Sokolow-Lyon criteria for RVH was not significantly different from baseline at month two (0.37 ± 0.23 mV vs 0.40 ± 0.25 mV) or month six (0.39 ± 0.26 mV; $p=0.109$). T-wave voltage was greater in leads V3-6 and lead I, II and aVF following six months of endurance training ($p<0.05$).

[Table 2 near here]

[Figure 1 near here]

ECG Interpretation Criteria

Based on the 2013 Seattle criteria and 2017 international guidelines, most individuals presented at least one ‘training-related’ ECG finding at month six (85%; $n = 56$), an increase of 17% from baseline (Table 3). The number of males with one or more ‘training-related’ ECG finding increased throughout the six months of training; however, the number of females presenting one or more ‘training-related’

finding did not change. There was no difference in ‘training-related’ findings between the 2013 and 2017 guidelines across time points, due to the similarities between criteria. Using the 2013 guidelines, four ECGs were identified as ‘training-unrelated’ at baseline vs. three ECGs at month six, all of which reflected either left axis deviation (QRS axis between -30° to -90°) or long QT interval (QTc >470 ms males and >480 ms females). In contrast, using the 2017 guidelines the number of ‘training-unrelated’ findings were fewer (two ECGs at baseline and one ECG at month six). Following six months of training, there was no increase in the incidence of ‘borderline findings’ (i.e. left axis deviation, left atrial enlargement, right axis deviation, right atrial enlargement or complete RBBB) introduced in the 2017 guidelines (Table 3).

[Table 3 near here]

Discussion

To our knowledge, this is the first study to longitudinally investigate exercise-induced ECG changes in a previously recreationally active cohort. The key findings were; first, a six-month endurance exercise programme lead to increased ‘training-related’ electrocardiographic changes, including sinus bradycardia, voltage criteria for LVH and early repolarisation. Secondly, the prevalence of revised ‘borderline’ criteria, according to the 2017 international consensus, did not increase and the associated quantitative ECG data (e.g. P-wave amplitude, QRS axis and QRS duration) remained unchanged.

Cardiac remodelling in response to exercise training has been well documented in cross-sectional studies of elite level endurance athletes (Arbab-Zadeh et al., 2014; D'Andrea et al., 2013; Pluim, Zwinderman, van der Laarse, & van der Wall, 2000) and few longitudinal studies (Arbab-Zadeh et al., 2014; Weiner et al., 2015). Although the ECG can be reflective of the underlying cardiac structure, the ECG criteria used to screen athletes is exclusively based on large-scale cross-sectional studies (Corrado, Biffi, Basso, Pelliccia, & Thiene, 2009; Corrado et al., 2010; Drezner et al., 2013). Furthermore, longitudinal studies investigating the influence of endurance exercise on the ECG are limited. One ECG variant that has been demonstrated longitudinally in athletes is the early repolarisation pattern (Noseworthy et al., 2011). Consequently, the magnitude of change (i.e. voltage

amplitude and interval duration) and the time course across which these adaptations occur, remains unknown. Our data show that ECG characteristics of the athlete's heart are not confined to elite athletes and can appear as normal variants in active individuals, particularly those initiating a high-volume training programme.

Cardiac Hypertrophy

The ECG changes associated with cardiac hypertrophy, such as increased R-wave and S-wave amplitude in V5 and V6, and resultant increase in Sokolow-Lyon index for LVH, likely reflect structural adaptations observed previously in response to endurance training (Arbab-Zadeh et al., 2014; Howden et al., 2015; Weiner et al., 2015). In a cardiac magnetic resonance imaging study, left and right ventricular mass was shown to increase progressively from 3 to 12 months of endurance training (Arbab-Zadeh et al., 2014). Previous findings from Weiner et al. (2015) suggest that the LV predominantly undergoes eccentric remodelling (increased chamber dimension) within the acute phase of training, followed by increases in wall thickness in the chronic phase. The delay in the changes to the LVH criteria in our data may be explained by the transition from acute to chronic phase remodelling.

Conduction Intervals

A reduction in heart rate of seven beats·min⁻¹ following six months of endurance training is consistent with previous studies finding training induced bradycardia (Scharhag-Rosenberger, Walitzek, Kindermann, & Meyer, 2012; Wilmore et al., 1996). The prolonged QT interval observed in this study likely represent the effect of prolonged depolarisation and repolarisation due to increased ventricular mass and stroke volume. Additionally, the reduction in heart rate likely contributed to the prolonged QT interval in our data (Malik, Farbom, Batchvarov, Hnatkova, & Camm, 2002). The concomitant decrease in QTc likely reflects the inadequacies of the Bazett's formula at low HR, such as that observed in endurance athletes (Basavarajaiah et al., 2007). ECGs with slow heart rates (<50 beats·min⁻¹) should therefore be repeated following a mild exercise stimulus as suggested in the international consensus guidelines (Drezner et al., 2017), to achieve a heart rate closer to 60

beats·min⁻¹. However, ECG's were not collected during exercise in the current study but should be in future investigations.

Atrial Conduction

The reduction in P-wave duration observed in the current study conflicts with cross-sectional investigations, suggesting that athletes have significantly prolonged P-wave duration (Wilhelm et al., 2011). Wilhelm et al. (2011) showed a greater P-wave duration (131 ± 6 ms vs. 142 ± 13 ms) in low compared to high trained groups, which is likely reflective of atrial enlargement, as has been reported with endurance training and the athlete's heart (D'Andrea et al., 2010; Hauser et al., 1985). Indeed, left atrial dilation and P-wave duration have been linked to the volume and intensity of training (D'Ascenzi et al., 2012; Pelliccia et al., 2005; Wilhelm et al., 2012). As previously mentioned, evidence suggests that the LV undergoes phasic remodelling (Arbab-Zadeh et al., 2014; Weiner et al., 2015) and it is possible the left atrium follows a similar phasic remodelling process. The reduction in P-wave duration indicating faster atrial conduction, however, is contrary to expected findings. Speculatively, this small but statistically significant change could be related to the exercise mediated cardiac fibroblast response (Burgess, Terracio, Hirozane, & Borg, 2002), or the electrical coupling of fibroblasts and myocytes (Kohl, Camelliti, Burton, & Smith, 2005). However, further investigation is warranted to examine the longitudinal P-wave response to endurance exercise and the potential of phasic remodelling.

ECG Interpretation Criteria

Screening athletes for cardiac pathology requires an understanding of the normal and abnormal ECG. The updated consensus for interpretation of an athlete's ECG has resulted in the re-categorisation of certain 'abnormal' criteria, to 'normal' when found in isolation. Because of this re-categorisation, the application of the 2017 international criteria to the current dataset, resulted in a lower prevalence of 'abnormal' ECGs in comparison to the Seattle 2013 criteria. Specifically, re-categorisation of isolated left axis deviation in the 2017 guidelines resulted in a reduction in 'training-unrelated' findings. Importantly, all previous ECG criteria have been based on cross-sectional investigation, due to a lack

of longitudinal studies investigating exercise-induced ECG changes. Although these cross-sectional studies are useful in understanding the impact of training on the ECG, they do not help us understand the progressive change in the ECG associated with exercise. Our exploratory study supports the contention that sinus bradycardia, early repolarisation and isolated voltage criteria for LVH, are indeed 'training-related'. In contrast, incomplete RBBB and first-degree atrioventricular (AV) block did not increase in incidence and there were no other 'training-related' findings across time; including junctional escape rhythm, ectopic atrial rhythm and Mobitz type I Wenckebach 2° AV block. It is possible, however, that six months of training was insufficient for several 'training-related' morphologies to manifest. Contrary to 'training-related' findings, there was no increase in the prevalence of the 2017 'borderline' ECG findings (i.e. left axis deviation, left atrial enlargement, right axis deviation, right atrial enlargement, and complete RBBB). Furthermore, we found no change in QRS axis, QRS duration or P wave amplitude (in lead II, III and aVF), and a small but significant decrease in P wave duration (in lead II). It is possible that the lack of increase in prevalence of borderline findings is also due to a phasic remodelling response, and such ECG abnormalities may develop with training durations greater than six months. As such, the findings from this exploratory investigation suggest that 'borderline' ECG findings may not necessarily represent training-related adaptation in novice athletes within the first six months of training, and therefore may warrant further clinical consideration. However, further investigation with larger sample sizes and greater training duration are necessary to ascertain the potential temporal development of these ECG variants and may be a future consideration of sports cardiologists when investigating novice athletes.

Study Limitations

The major limitation of this study is the absence of cardiac imaging, which restricts any direct association between ECG changes and structural or functional remodelling. Future longitudinal investigation should combine ECG measurements with imaging techniques such as echocardiography and magnetic resonance imaging, ensuring assessment of the right side of the heart. Doing so would contribute to our understanding of the relationship between the ECG and structural and functional adaptations and give insight into the time course of adaptive remodelling.

Whilst the primary aim of this study was to assess longitudinal changes to the ECG in response to endurance training, we secondly sought to assess the revisions made to the recent 2017 international consensus criteria. Therefore, it is important to note that the age of participants in the current study ranged between 18 and 49 years, in comparison to the international criteria, which was developed in the context of athletes aged 12-35 years. In addition, while the number of individuals with ECG changes associated with training increased over the programme, the number of females presenting 'training-related' ECG findings did not increase. This may represent a blunted cardiovascular response to endurance exercise in females, as has been previously observed (Howden et al., 2015), although more likely is a consequence of an underpowered sample size. It is also possible that the longitudinal design of the study did not allow sufficient time to fully characterise the training adaptation process. Indeed, Arbab Zadeh et al. (2014) and Weiner et al. (2015) recently demonstrated the complexities of the phasic response of exercise-induced cardiac remodelling.

Conclusion

Six months of endurance training in previously training-naïve individuals leads to ECG changes typically associated with ventricular hypertrophy, and a greater prevalence of 'training-related' ECG changes. Over a relatively modest time-period, ECG characteristics of the athlete's heart are likely to develop in those participating in high volume endurance exercise. Whilst the revised 'borderline findings' in the 2017 international consensus may reflect normal physiological adaptations in competitive athletes, these ECG variants may not necessarily represent training-related adaptation in novice athletes following a six-month training intervention. However, further investigation with larger sample sizes and greater training duration are required before explicit and broad conclusions can be made.

KEY MESSAGES

- This study aimed to assess the longitudinal ECG changes following six months of endurance training, in training-naïve individuals, and whether these ECG changes support the revisions made to the recent 2017 international consensus criteria.
- The prevalence of 'training-related' findings were increased with six months of endurance training, however the prevalence of the revised 'borderline' criteria, according to the 2017 international consensus, did not increase and the associated quantitative ECG data (e.g. P-wave amplitude, QRS axis and QRS duration) remained unchanged.
- Further clinical consideration may be warranted for individuals within the early phase of exercise engagement presenting with 'borderline' ECG abnormalities, defined by the International criteria.

Competing interests None declared.

Ethics approval This study was approved by the University of Hertfordshire Life and Medical Sciences Ethics Committee (LMS/SF/UH/00011) and conforms to the ethical guidelines of the 1975 Declaration of Helsinki.

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Table I. Participant characteristics displayed as mean \pm standard deviation (n = 66; F = 13, M = 53).

	Baseline	Month two	Month six
Age (years)	36 \pm 8		
Height (cm)	175.7 \pm 8.3		
Body mass (kg)	75.3 \pm 15.3	76.1 \pm 11.6	75.5 \pm 10.9
Body fat (%)	19.7 \pm 7.1	19.4 \pm 6.6	17.9 \pm 6.5*†

Systolic blood pressure (mmHg)	130 ± 10	124 ± 16*	133 ± 15†
Diastolic blood pressure (mmHg)	80 ± 7	75 ± 12*	78 ± 10
$\dot{V}O_{2\max}$ (l·min ⁻¹)	3.427 ± 0.653	3.688 ± 0.661*	3.773 ± 0.656*†
$\dot{V}O_{2\max}$ (ml·kg ⁻¹ ·min ⁻¹)	45.4 ± 7.1	47.8 ± 8.9*	50.3 ± 7.1*†
$\dot{V}O_2$ at LT (l·min ⁻¹)	2.181 ± 0.468	2.330 ± 0.461*	2.416 ± 0.545*
HR at LT (beats·min ⁻¹)	148 ± 13	139 ± 20*	139 ± 12*
Power output at LT (watts)	151 ± 27	157 ± 30	166 ± 34*†

HR, heart rate; LT, lactate threshold; $\dot{V}O_2$, volume of oxygen consumption over time. *Significantly different from baseline ($p < 0.05$); † significantly different from month two ($p < 0.05$).

Table 2. Selected quantitative 12-Lead ECG measurements at baseline, two and six months of a progressive triathlon-training programme (n = 66; F = 13, M = 53).

ECG Parameter	Baseline			Month two			Month six			ANOVA
HR (beats·min ⁻¹)	60	±	12	57	±	10*	53	±	8*†	$p < 0.001$
PR interval (ms)	164	±	22	166	±	23	167	±	26	$p = 0.223$
P-wave duration (ms)	106	±	10	105	±	10	103	±	11*	$p < 0.05$
QRS Duration (ms)	100	±	8	101	±	8	101	±	8	$p = 0.375$
QT interval (ms)	417	±	35	421	±	35	435	±	32*†	$p < 0.001$
QTc (ms)	413	±	27	406	±	22*	405	±	22*	$p < 0.001$
P Axis (°)	51	±	28	52	±	28	48	±	41	$p = 0.471$
QRS Axis (°)	66	±	29	67	±	31	66	±	35	$p = 0.846$
T axis (°)	51	±	15	50	±	17	53	±	25	$p = 0.332$
P-wave voltage										
II (mV)	0.15	±	0.06	0.15	±	0.06	0.16	±	0.07	$p = 0.851$
III (mV)	0.09	±	0.05	0.09	±	0.06	0.10	±	0.06	$p = 0.358$
aVF (mV)	0.12	±	0.05	0.12	±	0.06	0.12	±	0.06	$p = 0.884$
T-wave voltage										
V3 (mV)	0.72	±	0.33	0.77	±	0.32*	0.84	±	0.39*†	$p < 0.001$
V4 (mV)	0.74	±	0.34	0.83	±	0.34*	0.88	±	0.41*	$p < 0.001$
V5 (mV)	0.61	±	0.26	0.74	±	0.32*	0.74	±	0.33*	$p < 0.001$
V6 (mV)	0.45	±	0.18	0.58	±	0.26*	0.56	±	0.26*	$p < 0.001$

II (mV)	0.46	±	0.18	0.49	±	0.18*	0.48	±	0.20	$p<0.05$
III (mV)	0.19	±	0.15	0.21	±	0.17	0.23	±	0.20*	$p<0.05$
avF T (mV)	0.32	±	0.15	0.34	±	0.18	0.36	±	0.17*	$p<0.05$
QRS voltage										
V5 R (mV)	1.71	±	0.50	1.81	±	0.61*	1.83	±	0.62*	$p<0.05$
V5 S (mV)	-0.17	±	0.19	-0.21	±	0.22*	-0.19	±	0.21	$p<0.05$
V6 R (mV)	1.33	±	0.39	1.52	±	0.62*	1.42	±	0.46*	$p<0.05$
V6 S (mV)	-0.07	±	0.08	-0.10	±	0.13*	-0.08	±	0.11	$p<0.05$

Results are mean ± SD. HR, heart rate. *Significantly different from baseline ($p<0.05$); † significantly different from month two ($p<0.05$).

Table 3. Frequency and distribution of ‘training-related’ and ‘borderline’ ECG criteria (n = 66; M = 53, F = 13).

	Baseline	Month two	Month six
‘Training-Related’ ECG Finding	Frequency		
Sinus bradycardia	37	42	46
First degree AV block	7	8	6
Early Repolarisation	10	14	19
Incomplete RBBB	6	6	6
Isolated voltage criteria for LVH	3	6	10
Isolated voltage criteria for RVH	2	1	0
Junctional escape rhythm	0	0	0
Ectopic atrial rhythm	0	0	0
Mobitz type I Wenckebach 2° AV block	0	0	0
No. of participants showing ≥ 1 ‘training-related’ findings	45	52	56
Frequency of ‘training-related’ findings: TOTAL	65	77	87
No. of males showing ≥ 1 ‘training-related’ findings	37	49	49
Frequency of ‘training-related’ findings: Male	55	72	84
No. of females showing ≥ 1 ‘training-related’ findings	7	7	7
Frequency of ‘training-related’ findings: Female	8	7	8
‘Borderline’ ECG Finding	Frequency		

Left axis deviation	2	1	2
Left atrial enlargement	0	0	0
Right axis deviation	0	0	0
Right atrial enlargement	5	5	5
Complete RBBB	0	0	0

AV, atrioventricular; RBBB, right bundle branch block; LVH, left ventricular hypertrophy; RVH, right ventricular hypertrophy.

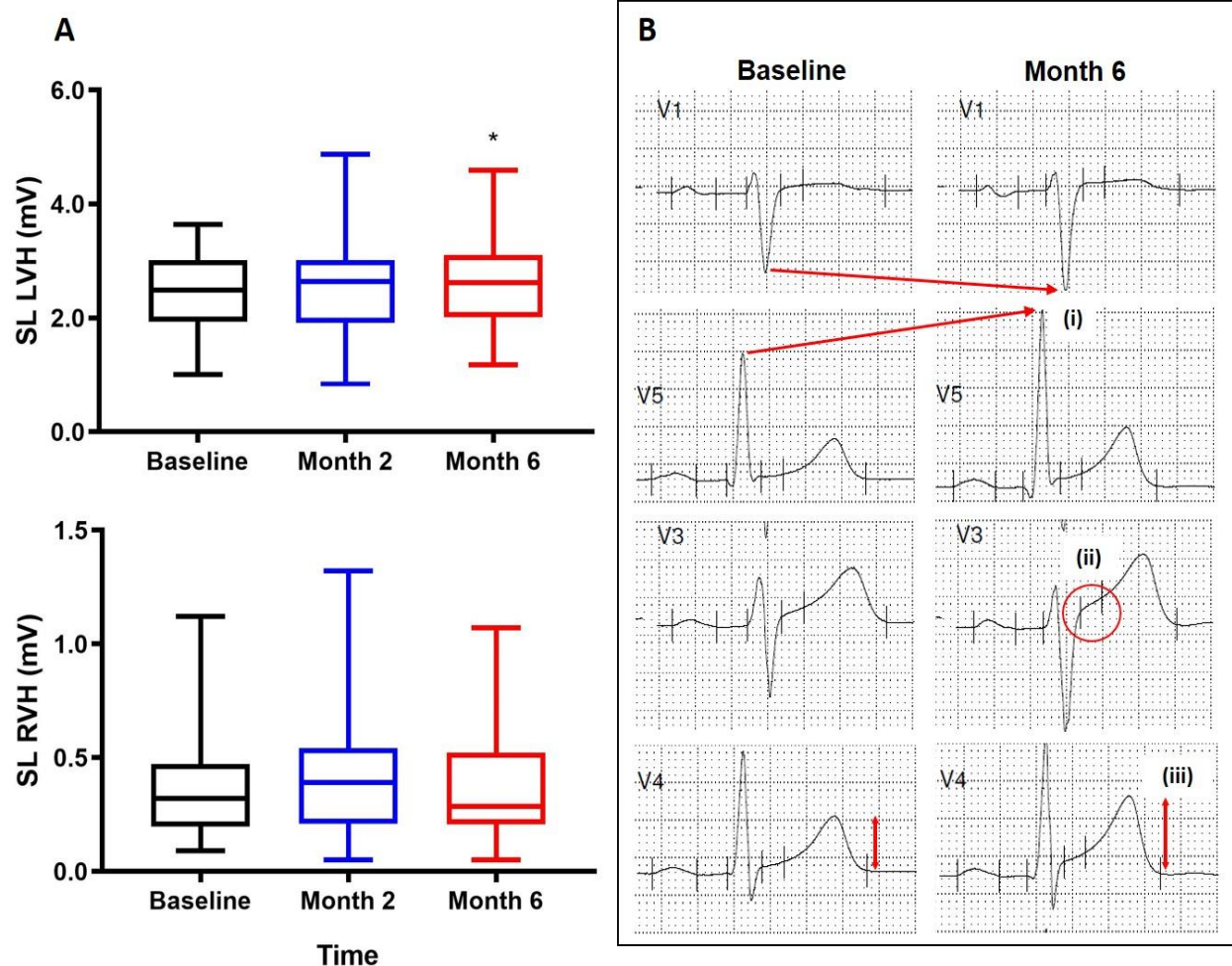


Figure 1. (A) Group data showing Sokolow Lyon voltage criteria for left ventricular hypertrophy (SL LVH) and right ventricular hypertrophy (SL RVH) over a six-month training programme and (B) individual ECG trace at baseline and month six of training. At month six, this individual's ECG

presented 'training related' findings including (i) isolated voltage criteria for LVH (3.69 mV) but not RVH (0.24 mV) and (ii) early repolarisation (ST segment elevation 0.2mV above isoelectric line in precordial lead V3). Also shown is the progressive increase in T wave amplitude from V4 (iii). * represents a significant difference from baseline ($p < 0.05$).