

Exploratory Insights from the Right-sided Electrocardiogram following Prolonged Endurance

Exercise

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Abstract

Background

Prolonged strenuous exercise has a profound effect on cardiac function. The right heart may be more susceptible to this imposition yet right-sided chest leads have not been utilised in this setting.

Methods

Thirty highly trained athletes at the 2014 Western States 100 mile Endurance Run from Squaw Valley to Auburn, California (Body mass 68 ± 12 kg, age 45 ± 10 years, 57 ± 15 miles per week) were recruited for the study. Pre and post-race, a right-sided 12-lead ECG was obtained and data extracted for P, R and S waves, J point, ST segment and T wave amplitude. Data were compared using Students T-test and statistical significance set as $P < 0.05$.

Results

There was a significant increase in P wave amplitude (29%) and QTc interval (4%) pre to post-race from standard 12-lead ECG. From the right sided 12-lead ECG, a 23% ($P = 0.01$) and 38% ($P = 0.03$) increase in J point amplitude in V1R and V2R and a 22% ($P = 0.05$) increase in ST segment integral in V2R and V3R were evident. T wave inversion was evident in leads V2R-V6R in 50-90% of athletes respectively. Close examination revealed marked heterogeneity in individual ECGs.

Conclusions

Completion of a 100 mile ultra-marathon resulted in significant changes in the right-sided ECG alongside more marked responses in specific individuals. P wave, ST segment and T wave changes post-race are indicative of acute exercise induced right heart electrical adaptation.

Keywords: electrocardiogram, right-sided ECG, prolonged endurance exercise,

Abbreviations

Abbreviation	Definition
ECG	Electrocardiogram
RV	Right Ventricle
LV	Left Ventricle
RBBB	Right Bundle Branch Block

Introduction

Prolonged strenuous exercise appears to have a profound effect on the structure and function of the heart. This includes a change in the electromechanical association in the ventricles suggesting a potential impact upon electrical activation (Chan-Dewar et al., 2010) that was also associated with post-exercise cardiac dysfunction.

The right side of the heart has been suggested to be more susceptible than the left ventricle to the impact of prolonged exercise stress as a result of the relatively higher pulmonary vascular resistance and a higher relative wall stress in the right ventricle (RV) when compared to the left ventricle (LV) (La Gerche et al., 2011). Indeed, early interest in RV adaptation to prolonged endurance exercise stemmed from case studies of pulmonary embolism following an ultra-marathon (McKechnie et al., 1979) and exercising pulmonary artery pressures are reported to reach values of 60-70 mmHg. This is in excess of those clinically diagnosed in pulmonary hypertension (Kovacs et al., 2009).

A recent study by our research group (Lord et al., 2014) assessed 12-lead electrocardiogram (ECG) changes following a 100 mile ultra-marathon and demonstrated an increase in the summated R wave in V1 and S wave in V5, J point elevation in V1, partial right bundle branch block (RBBB), T wave inversion in lead V1 and early repolarisation pre to post-race pointing to changes in the right-sided electrical conduction system. Nevertheless, the use of right-sided ECG leads, focusing on the right

heart, have not been employed in this setting. Given the imposition of a disproportional workload on the RV during exercise and the changes reported in the standard 12-lead ECG, it is pertinent to further investigate the electrical activity in the right side of the heart following prolonged exercise. Right-sided chest leads have been previously utilised in addition to the standard 12-lead ECG to determine RV involvement in myocardial infarctions associated with the right coronary artery (Zehender et al., 1993) and in the diagnosis of pulmonary hypertension and pulmonary embolism (Akula et al., 2003; Chia et al., 1997). In these clinical situations, the right-sided leads demonstrate ST segment changes that are not evident on a standard 12-lead ECG and have an increased sensitivity and specificity in diagnosing conditions affecting the RV.

The R wave in the standard ECG leads represents depolarisation of the main mass of predominantly the left ventricle. The R wave in the right-sided lead represents the depolarisation of both ventricles with a preponderance towards the right ventricle. Without comparison with detailed echocardiographic measurements of the right ventricle we are unsure whether these changes represent right ventricular morphology, function and pulmonary artery pressure, but they are likely to represent right-sided pressure/volume overload.

The S wave in the standard leads represents the final depolarisation of the ventricles at the base of the heart. The S wave in the right-sided leads represents the final depolarisation with a preponderance towards the right ventricle. Changes in the right-sided S wave amplitude are likely to reflect changes in the right ventricle with right-sided pressure/volume overload.

The ST segment is the time between the end of the QRS complex and the start of the T-wave and reflects the period of zero potential between ventricular depolarisation and repolarisation. The J point is the onset of the ST segment. Changes in the ST segment and J point in the standard leads (whether elevated or depressed) can be associated with many conditions such as coronary ischaemia, myocardial infarction, myocarditis, pericarditis, electrolyte imbalance and certain drugs. ST segment changes are also considered a normal variant especially in highly-trained athletes. Changes in the ST segment in the right-sided leads may reflect similar conditions affecting the right ventricle, especially conditions resulting in right-sided pressure/volume overload.

Consequently, the aim of the current study was to determine whether the right-sided ECG demonstrates changes indicative of right-sided adaptation following prolonged endurance exercise. This broad aim allows for the generation of the following hypothesis: the right-sided 12-lead ECG will indicate ST segment elevation and T wave changes indicative of right-sided pressure/volume overload.

Methods

Sample Population

Thirty athletes (20 male, 10 female) at the 2014 Western States Endurance Run (28th June, Squaw Valley to Auburn, CA) (Body mass 68 ± 12 kg, height 173 ± 11 cm, age 45 ± 10 years) were recruited and volunteered to take part in the study. Recruitment took place through the official race research committee email correspondence to all athletes taking part in the race. Participants self-reported that they had no family history or diagnosis of cardiovascular disease or co-morbidities for cardiovascular disease, were not taking any prescribed medication and had no musculoskeletal injuries. The current training status for the cohort was 6 ± 1 days per week, 57 ± 15 miles per week, 14 ± 5 training hours per week and 35 ± 26 previously completed ultra-marathons. Participants completed the race in 26.29 ± 2.95 hours. Written informed consent was obtained and Ethics approval was granted by the Liverpool John Moores University Ethics Committee.

Study Design

The 30 participants were assessed pre-race (24 - 48 hours prior to the race) and immediately post-race (within 30 minutes of race completion). Height, body mass, resting blood pressure and right-sided 12-lead ECGs were recorded at baseline and immediately post-race. Participants were requested to avoid vigorous training and alcohol for the 24 hours prior to and caffeine for the 6 hours prior to the pre-race assessments. Participants were permitted to consume food and fluid *ad libitum* during the race. The Western States Endurance Run encompasses 100.2 miles, with a starting

elevation of 1,899 m climbing to a maximum of 2,656 m and a minimum of 166 m above sea level. The race includes 5514 m of ascent and 7001 m of descent over the duration of the race. The ambient temperature ranged from 0 to 31.7 °C.

Standard 12-lead Electrocardiogram and Posterior Leads

The 12-lead ECG was undertaken in accordance with recommended guidelines on standard limb and precordial electrode positions (Kossmann et al. 1967) using CardeaScreen (DETAILS) with the subject in a supine position. All standard 12 lead ECGs were interpreted using the European Society guidelines (REF). Extracted data included P wave amplitude in lead II, T wave amplitude in lead V1, J point elevation in lead V1, ST segment elevation in lead V1, QRS axis, QTc interval (Bazetts formula) and the summation of the R wave in lead V1 and S wave in lead V5 (RV1SV5). The incidence of the following findings were recorded; J point elevation in lead V1 (>1mm), ST segment elevation (>1mm), partial or complete RBBB, T wave inversion (>1mm) in lead V1, right axis deviation (RAD, >90°), right ventricular hypertrophy (RVH, R wave amplitude in V1 plus S wave amplitude in V5 >10.5mm) and early repolarisation.

All participants underwent a repeat 12-lead ECG with V4-6 relocated to posterior chest leads V7-9 prior to and on completion of the race. These ECGs were analysed for any change pre to post-race indicative of coronary artery involvement.

Right-sided 12-lead Electrocardiogram

Limb leads were placed in the same position as the standard 12-lead ECG, the precordial leads were mirrored from the standard 12-lead ECG onto the right side of the chest and positioned following the recommended guidelines for the standard 12-lead ECG (Kossmann et al., 1967) giving leads V1R-V6R. Each of the 6 precordial leads V1R-V6R was analysed for J point elevation (>1mm), ST segment elevation (>1mm), T wave amplitude, R wave amplitude, R' wave amplitude and S wave amplitude. The ST segment was calculated using the J point and J point plus 80ms and integrating

the difference between the two points and termed ST segment integral. Data analysis was performed using computerised analysis software (CardeaScreen, Stanford University, CA).

Statistics

All variables were analysed for normality of distribution using a Shapiro-Wilk test. Pre to post-race peak values for each variable were compared using Student's Paired T-tests using commercially available software (IBM SPSS v21) and statistical significance was set as $P < 0.05$.

Results

Demographics

There were 376 participants in the 2014 Western States Endurance Run, 296 of these completed the race. Overall race finish times ranged from 14:53 to 29:57 hours. For the 30 athletes included in the analysis for this study, race finish times ranged from 18:19 to 29:52 hours. There was a significant increase in heart rate (58 ± 7 to 78 ± 10 bpm) and reduction in systolic (128 ± 11 to 120 ± 14 mmHg) and diastolic (84 ± 8 to 78 ± 10 mmHg) BP pre to post-race ($P = 0.004$). There was no change in body mass pre to post-race ($P = 0.08$). The two case study athlete demographics are as follows: Case study 1, female, 30 years old, 13 years of training, 15 hours per week, 6 days per week, 60 miles per week, 33 previous ultra-marathons, 12 ultra-marathons in the last 2 years, 14th place in last ultra marathon. Case study 2, male, 40 years old, 12 years of training, 15 hours per week, 6 days per week, 80 miles per week, 20 previous ultra marathons, 4 in the last 2 years, 12th place in last ultra marathon.

Standard and posterior 12-lead ECGs

Pre to post-race there was a 29% increase in P wave amplitude ($P = 0.001$), 4% increase in corrected QT interval ($P < 0.001$), 38% increase in J point amplitude ($P = 0.03$) and 22% increase in ST segment integral amplitude ($P = 0.049$) and a 10% decrease in RV5SV1 (LVH) ($P = 0.005$). There was no change in T wave amplitude, R wave amplitude in V1, S wave amplitude in V5, QRS axis or RV1SV5 (RVH) from pre to post race. Based on interpretation derived from standard ECG criteria, there was no evidence of RA or LA enlargement, isolated voltage criteria for LVH and RVH or right axis deviation pre or post-race in any athlete. One athlete presented with borderline left axis deviation (QRS axis – 31 degrees) which was maintained from pre to post-race. One athlete had a QTc interval of 449 ms meeting the criteria for long QT syndrome at baseline, post-race this number increased to 11 athletes. No abnormal T wave inversions were evident pre-race. There were 11 cases of T wave inversion ≥ 1 mm in lead V1, 8 of these were maintained post-race whereas 6 new cases were also evident post-race. There was one case of T wave inversion ≥ 1 mm lead aVF pre-race which was not present post-race and one new case of T wave inversion in lead V2 post-race that was not present at baseline. No evidence of T wave inversion was evident in leads I, II or V3-V6 pre or post-race. There was no evidence of pre to post-race myocardial ischemia as documented on posterior ECGs.

Right-sided 12-lead ECG

There was a 12% ($P = 0.04$) reduction in S wave amplitude in lead V1R from pre to post-race (see Table 2). There was a 23% ($P = 0.01$) and 38% ($P = 0.03$) increase in J point amplitude in leads V1R and V2R respectively from pre to post-race (see Table 2). A converse response was evident in lead V6R with an 80% ($P = 0.03$) depression in J point amplitude from pre to post-race (see Table 2). There was a 22% ($P = 0.05$) increase in ST segment integral in V2R and V3R (see Table 2 and Figure 1) consistent with ST elevation and a 27% ($P = 0.03$) decrease in ST segment integral in lead V6R from pre to post-race (see Figure 1). No significant ST segment elevation was evident in leads V4R-V6R. All ST segment changes were below criteria for clinical diagnosis of myocardial ischemia/infarction. T wave inversion was evident in leads V2R-V6R in 50, 57, 73, 90 and 83% of athletes respectively.

Despite there being significant cohort changes in right-sided ECG parameters, close inspection of individual ECG traces pre and post-exercise demonstrated heterogeneous changes in specific individuals. This is notable in two case studies where the degree of response is marked (see Figures 2 and 3). These two case reports demonstrate P pulmonale, ST segment elevation in the right-sided precordial leads V1R-V6R with marked T wave changes alongside the development of R' waves in lead V1R and V2R.

Table 1 - Standard 12 lead electrocardiogram variables

Variable	Pre-race	Post-race	P value
P wave amplitude (mV)	1.23 ± 0.51	1.59 ± 0.52*	0.001
T wave amplitude (mV)	-0.05 ± 1.69	0.17 ± 1.96	0.45
J point amplitude (mV)	0.32 ± 0.35	0.44 ± 0.40*	0.039
ST integral	0.49 ± 0.43	0.60 ± 0.52*	0.049
QRS axis (°)	41.91 ± 33.72	44.30 ± 35.86	0.344
RV5SV1 (mV)	27.53 ± 7.80	24.69 ± 7.49*	0.006
RV1SV5 (mV)	5.37 ± 2.70	5.50 ± 2.01	0.242
QTc interval (ms)	421.93 ± 16.91	438.58 ± 19.88*	<0.001

* denotes statistical significance, $P < 0.05$.

Table 2 - Right-sided 12-lead electrocardiogram variables

Variable	Pre	Post	P value
LEAD V1R			
R wave amplitude (mV)	5.91 ± 2.98	5.92 ± 2.78	0.97
R' wave amplitude (mV)	0.36 ± 0.91	0.64 ± 1.74	0.38
S wave amplitude (mV)	-13.82 ± 6.50	-12.13 ± 6.39*	0.044
J point amplitude (mV)	0.92 ± 0.57	1.13 ± 0.62*	0.014
T wave amplitude (mV)	5.12 ± 3.12	4.99 ± 3.27	0.75
LEAD V2R			
R wave amplitude (mV)	2.86 ± 2.08	2.77 ± 1.83	0.422
R' wave amplitude (mV)	0.75 ± 2.23	0.72 ± 1.95	0.755
S wave amplitude (mV)	-9.44 ± 4.62	-9.07 ± 4.46	0.381
J point amplitude (mV)	0.32 ± 0.35	0.44 ± 0.40*	0.034
T wave amplitude (mV)	-0.05 ± 1.69	0.17 ± 1.96	0.25
LEAD V3R			
R wave amplitude (mV)	1.92 ± 1.27	2.10 ± 1.36	0.12
R' wave amplitude (mV)	0.77 ± 1.94	0.71 ± 1.92	0.38
S wave amplitude (mV)	-6.81 ± 3.94	-6.69 ± 3.77	0.77
J point amplitude (mV)	0.15 ± 0.30	0.24 ± 0.30	0.11
T wave amplitude (mV)	-0.61 ± 1.31	-0.19 ± 1.58	0.12
LEAD V4R			
R wave amplitude (mV)	1.44 ± 0.88	1.43 ± 0.94	0.90
R' wave amplitude (mV)	0.69 ± 1.37	0.79 ± 1.92	0.14
S wave amplitude (mV)	-4.59 ± 3.41	-4.72 ± 2.89	0.71
J point amplitude (mV)	0.04 ± 0.24	0.10 ± 0.22	0.18
T wave amplitude (mV)	-0.95 ± 0.93	-0.78 ± 1.09	0.33
LEAD V5R			
R wave amplitude (mV)	0.83 ± 0.60	0.81 ± 0.64	0.78
R' wave amplitude (mV)	0.70 ± 0.92	0.67 ± 0.95	0.65
S wave amplitude (mV)	-2.96 ± 2.19	-2.71 ± 1.78	0.55
J point amplitude (mV)	-0.03 ± 0.15	-0.05 ± 0.15	0.52
T wave amplitude (mV)	-1.30 ± 0.48	-1.22 ± 0.57	0.24
LEAD V6R			
R wave amplitude (mV)	0.88 ± 0.71	0.76 ± 0.68	0.21
R' wave amplitude (mV)	0.41 ± 0.69	0.41 ± 0.73	0.99
S wave amplitude (mV)	-1.90 ± 1.94	-1.62 ± 1.72	0.32
J point amplitude (mV)	-0.05 ± 0.12	-0.09 ± 0.11*	0.029
T wave amplitude (mV)	-1.18 ± 0.46	-1.14 ± 0.53	0.49

* denotes statistical significance, $P < 0.05$.

Discussion

The key findings from this study are evidence of ST segment elevation in leads V1R and V2R, ST depression in lead V6R and T wave changes in leads V1R-V5R following the race with no concomitant changes in the standard or posterior 12-lead ECGs. Importantly, the data in the current study demonstrated a heterogeneous response with some athletes demonstrating clear ST segment and T wave changes in the right-sided leads and generation of R' waves in the anterior precordial leads as demonstrated in the case reports. This study applied right-sided precordial leads in the assessment of electrical changes following a bout of prolonged endurance exercise. Although a clear response across the whole sample is not evident, some data are indicative of a heterogeneous response to the 100 mile ultra-marathon with some individuals demonstrating a response similar to that seen in the presence of an elevated RV afterload (Akula et al., 2003; Chia et al., 1997). This varied response to an exercise stimulus and the consequent RV afterload has been reported previously using both standard 12-lead ECG (Lord et al., 2014) and echocardiography (George et al., 2009; Oxborough et al., 2011).

Standard 12 lead ECG

In the standard 12-lead ECG, there was post-race evidence of repolarization changes with J point, ST segment and T waves changes following the 100 mile ultra marathon. Kucher and colleagues (2003) presented similar 12-lead ECG findings in patients following pulmonary embolism and suggest that this was consistent with RV strain (Kucher et al. 2003) and these finding are also supported by data from Lord et al. (2014) where similar repolarization changes are evident in the presence of a marked elevation in RV afterload. There was a significant increase in QTc interval, with 11 athletes meeting criteria for long QT syndrome following the race. Repolarisation changes (Sahlen et al. 2009) and increased QTc interval (Stewart et al. 2014; Lord et al. 2014) have been previously reported in response to prolonged strenuous exercise and may be related to increased parasympathetic activity in the recovery stage. Chan-Dewar et al (2010) demonstrated an increased electromechanical delay in both ventricles following prolonged endurance exercise and it is pertinent to consider that electrical conduction abnormalities may be implicated. The current study also revealed a decreased summation of R wave in lead V5 and S wave in lead V1 (criteria for LVH). Whilst speculative, this may be

indicative of a reduced LV filling and therefore indicative of LV volume decrease as opposed to LV mass as seen in studies using echocardiography to quantify LV morphology following prolonged endurance exercise (Oxborough et al. 2010, 2011; La Gerche et al. 2012).

The elevation in J point and ST segment in leads V1R-V3R and converse depression in lead V6R were demonstrated across the cohort following a 100 mile ultra-marathon and indicate repolarisation changes specific to the RV. T wave changes were also evident in numerous athletes in leads V2R-V4R following the ultra-marathon giving further support to a change in repolarisation. These changes likely indicate an exercise-induced right-sided adaptation in electrical conduction and structure following prolonged endurance exercise. The mechanism behind these changes and the clinical implications for the athlete are at present unknown but these post-exercise electrical adaptations are likely to represent physiological modifications in response to the disproportionate workload placed on the RV during exercise (La Gerche et al., 2011) and could be linked to those reported using echocardiographic data following prolonged strenuous endurance exercise.

There was a significant increase in P wave amplitude in lead II from pre to post-race in the current study and although the value does not reach the criteria for P pulmonale (Stein et al., 1975; Stein et al., 1991), this may be indicative of right atrial (RA) enlargement or restrictive filling in the presence of an acute pressure/volume overload on the RA (Stein et al., 1991). Interestingly, P pulmonale has been reported in cases where an acute afterload is placed on the right side of the heart and the increase in P wave amplitude in the current study may therefore indicate a manifestation of the transient elevation in the pulmonary artery pressures previously reported following ultra-endurance exercise (Kovacs et al., 2009; La Gerche et al., 2011). In this case, the increase in P wave amplitude could be indicating both right atrial enlargement and/or a pressure overload on the right atrium.

Right and left atrial enlargement has been reported using transthoracic echocardiography (Oxborough et al., 2010) and studies have also observed RV dilatation (La Gerche et al., 2012; Oxborough et al., 2011) and a reduction in LV end diastolic volume (La Gerche et al., 2012) in recovery from ultra-

endurance exercise. Cardiac function has also been assessed using echocardiography and a global decrease in both systolic and diastolic RV (Neilan et al., 2006; Poh et al., 2008) and LV (Banks et al., 2011; George et al., 2009) function in recovery from prolonged endurance exercise reported in conjunction with the acute structural adaptations. The right-sided ECG changes evident in the current study may represent an electrical adaptation linked to these structural and functional changes and provide further insight into the acute electrical adaptations in response to a bout of ultra-endurance exercise. The mechanistic implications for this data with respect to post-exercise cardiac dysfunction are important to consider and they may provide further support for a disproportionate exercise load on the RV. If exercise induces a pressure and volume overload on the RV, there may be acute structural changes or micro-injury to the myocardium, which may ultimately influence ECG data. Acute ECG changes can also be caused by disturbances to blood electrolytes (Diercks et al., 2004), especially after a prolonged endurance exercise bout (Overgaard et al., 2002; Stewart et al., 2014). Core temperature, autonomic nervous system activity and inflammatory responses may also have a role in acute ECG changes following prolonged endurance exercise. At present, the potential mechanisms responsible for these acute ECG changes are indeterminate and the variables alluded to influence ECG data warrant further investigation to allow firm conclusions to be drawn on post-exercise electrical responses.

In Case Study 1 (Figure 2), there is new T wave inversion evident in leads V2R-V6R post-race compared to pre-race. Case Study 2 (Figure 3) demonstrates an increase in P wave amplitude pre to post-race in lead II, normalisation of the ST segment and T wave inversion in leads V3R-V5R and development of R' waves in V1R and V2R. Two previous studies analysing right-sided ECG in the presence of an elevated afterload have reported T wave inversion in leads V4R-V6R (Chia et al., 1997) and conversely reported ST segment elevation with flattened T waves in leads V3R-V6R (Akula et al., 2003; Chia et al., 1997). The two cases in the current study demonstrate similar changes to those presented in conditions of RV afterload but to a lesser degree. These two case studies indicate

a varied response to a similar stimulus and further support the notion of heterogeneity in response to an elevated afterload. This is likely dependent on the heterogeneous demographic of the athlete, although given their specific demographics, the two athletes included in these case studies do not seem to be outliers in the data set.

Studying acute ECG changes may help to understand the mechanisms involved in both acute and chronic electrical remodelling seen in endurance athletes and also inform echocardiographic assessments following ultra-endurance exercise. Chronic exercise training in the athlete results in structural and functional cardiac adaptation as determined by diagnostic imaging such as transthoracic echocardiography (Oxborough et al., 2012; Utomi et al., 2014) and the standard 12-lead electrocardiogram (ECG) (Corrado et al., 2009). These changes are well documented and inform the criteria for interpretation of the 12-lead ECG in athletes (Corrado et al., 2010; Drezner et al., 2013) but the adaptation process and mechanisms are not fully understood. The acute changes in the current study of ST segment elevation and the development of R' waves could be linked to chronic adaptation in athletes such as early repolarisation and incomplete right bundle branch block. A longitudinal investigation over a period of training may expose some mechanistic links between acute exercise responses and chronic electrical remodelling.

Limitations

This exploratory study has provided the first data for right-sided precordial leads in the assessment of athletes following prolonged endurance exercise, there are however some limitations. There is a lack of normative data for the right-sided ECG in endurance athletes and therefore the electrical adaptation to endurance training is not known for the right-sided precordial leads. That said, our pre-race data can be considered as a small sample of the typical right-sided ECG in endurance athletes. Data were only acquired at two time points, before the race and immediately following the race and therefore the transience of the observed changes is not known and would be a useful addition in future studies to determine the recovery period. Echocardiographic data at the same time point as the right-sided ECG would provide a full structural and functional assessment of the right heart allowing

structural changes to be correlated with electrical changes to determine any relationship between these variables. Pulmonary/respiratory data may also be a useful addition in future studies to further investigate the relationship between the pulmonary circuit and pressures with the RV in the post-exercise setting. This was not feasible in the current study due to post-race logistics. Changes in body temperature, blood electrolyte balance, skin moisture and autonomic function could all impact on the ECG data following a prolonged endurance exercise bout. Whilst these data were not collected in the current study, all athletes were towed dry in the medical tent at end of race prior to ambulatory measurements. Participants were unlikely to be hyperthermic or have any adverse/severe electrolyte disturbance given that none required any medical attention. Post-race athlete's body mass was not significantly different suggesting hydration had been adequately maintained.

Conclusion

Completion of a 100 mile endurance run results in electrical adaptation in the standard and right-sided precordial ECG leads affecting QTc duration, P wave, ST segment and T wave amplitude post-race. Furthermore, in a number of athletes there were pronounced changes indicative of acute exercise induced afterload and right heart electrical and/or structural adaptation.

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Table and Figure Legends

Table 1 - Right-sided 12-lead electrocardiogram variables, * indicates significant difference pre to post-race

Figure 1 – ST integral pre-race and post-race for leads V1R-V6R, * indicates significant difference pre to post-race, data presented are mean \pm SD

Figure 2 – Case Study 1 demonstrating T wave changes from pre-race to post-race in leads V1R-V6R indicated using red circles. Note precordial leads V1-V6 on the ECG represent right-sided precordial leads V1R-V6R.

Figure 3 – Case Study 2 demonstrating post-race P pulmonale in lead II, development of R' waves in the anterior leads V1R and V2R and ST segment changes in leads V1R-V5R indicated by red circles. Note precordial leads V1-V6 on the ECG represent right-sided precordial leads V1R-V6R.

