REGIONAL AND GLOBAL LEFT VENTRICULAR FUNCTION FOLLOWING A SIMULATED 5 KM RACE IN SPORTS-TRAINED ADOLESCENTS

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Abstract
The effects of a short, high-intensity bout of exercise, on cardiac systolic and diastolic function are not well understood in adolescent athletes. Consequently, the aims of the study were to evaluate global left ventricular (LV) systolic and diastolic function, as well as segmental wall motion responses (cardiac strain), prior to as well as 45 and 225 min following a simulated 5 km cross-country race. Twenty trained, adolescent males (age:15.2±0.7 years) volunteered for exercise testing. LV fractional shortening and the ratio of early (E) and late (A) peak flow velocities, reflected global systolic and diastolic function, respectively. Peak longitudinal mitral annular septal tissue velocities were also determined during systole and diastole. Longitudinal strain (ε) and strain rates were determined across the LV. LV fractional shortening was significantly ($P<0.05$) higher 225 min post-race (37.6 ± 5.8%) compared to pre-race (34.5 ± 4.7%) and 45 min post-race (34.9 ± 5.4%). This difference was abolished after adjusting for post-race heart rates. There was a significant ($P<0.05$) decrease in the E:A ratio at both 45 min (2.04 ± 0.57) and 225 min post-race (2.20 ± 0.66) compared to the pre-race value (2.80 ± 0.68). When these data were adjusted for post-race heart rates, these pre-post-race differences in E:A ratio were abolished. There were no significant alterations in either tissue Doppler velocities or longitudinal ε. The evidence suggests that a 5 km race does not lead to any significant post-exercise attenuation in global or regional LV systolic and diastolic function in trained adolescents.

KEY WORDS: Cardiac Function, Adolescent, Athletes, Exercise

INTRODUCTION:

The effects of an acute bout of exercise, on left ventricular (LV) systolic and diastolic function are not well understood in adolescent athletes [1, 2]. Nie et al.,[1] used conventional M-mode and Doppler echocardiography to demonstrate reductions in LV ejection fraction (EF) and trans-mitral flow velocities (E:A) following two, 45-min runs at ventilatory threshold in a group of adolescent athletes (14.5 years). Hauser et al., [2] are the only group to evaluate both global and regional left ventricular function following endurance exercise in children and adolescents. In this study, 27 children (mean age: 12. 6 years) had cardiac assessments pre and post an age-adapted triathlon circuit. Whilst M-mode echocardiography demonstrated no global change in left ventricular end-systolic dimension and EF following the race, regional, 2-D speckle tracking analysis demonstrated a reduction in longitudinal and circumferential strain post-race. These measurements were captured at a single time-point following the triathlon. One of the unique aspects of the present study, was to provide an early and late recovery assessment of these young athletes and, therefore, provide an evaluation of the longer-term changes that accompany an acute exercise bout.

In adults, endurance and ultra-distance events have been associated with transient impairments in LV function. These have taken the form of blunted systolic and/or diastolic LV function [3]. In the main, global measures of LV function have been used to investigate the post-exercise decrements in systolic and diastolic function. For example, the use of pulsed-wave Doppler technology has demonstrated reductions in E:A as an indicator of compromised diastolic function [4] and a reduced EF has been used as a marker of systolic dysfunction following exercise [5]. More recently, indices of regional myocardial function, such as LV strain and LV strain rate have been used to provide insight into intrinsic contractility and relaxation [6,7]. Evidence of impaired longitudinal [8] and circumferential strain [9] post-exercise has been noted using these regional markers. Furthermore, inherent

variability in the global and regional indices of post-exercise LV function exist and, consequently, George et al. [7] mapped the individual pre- to post-exercise changes in these indices, to highlight the inter-individual segmental differences to prolonged exercise. A unique aspect of the present study was an interrogation of the individual profiles of the adolescent athletes, pre-to-post exercise, to explore any inter-individual variability in regional LV function.

All these previously cited studies, characterize responses in highly-trained adolescent or adult runners. Within the paediatric population, highly-trained endurance athletes represent a very small percentage of the active adolescent population. The background of the athletes selected in the present study (team sport players) and the distance selected for the race (5 km) is much more reflective of the scenario that exists in most school physical education classes and school sports events. Consequently, quantifying the global and regional changes in left ventricular function in this population has greater relevance for the active adolescent population. It was hypothesised that there would be some transient, reversible changes in global and regional systolic and diastolic left ventricular function following the simulated 5 km cross-country race.

The specific aims of the study were to: 1) evaluate global and regional LV systolic function prior to and following a 5 km cross-country race, 2) evaluate global and segmental LV diastolic function prior to and following a 5 km cross-country race and 3) describe the individual trajectories of the global and regional markers of myocardial function, prior to and following a 5 km cross-country race.

METHODS

Subjects:

Twenty, sports-trained, adolescent males (mean ± SD, age: 15.2±0.7 years) from the same school volunteered for exercise testing (Table 1). The subjects were all involved in systematic training in team sports, track and field or a combination of both (mean: 7 years of training, 11 months per year, 4 times per week, 5.2 hours per week and been competing in races or team sports for 5.3 years). All subjects underwent a physical examination and completed a medical history questionnaire. None of the subjects were taking any medications that would influence cardiovascular function. There was no early family history of cardiovascular disease. Maturity status was determined using the Tanner self-assessment procedure [10]. Informed written parental permission and subjects' written assent were obtained prior to participation. The study was approved by an institutional research ethics committee. Physical activity, training and pre-test questionnaires were completed prior to the start of the study.

Study Design:

At the pre-race evaluation, after determination of mass and stature using standard techniques, body composition was assessed by the sum of 4 skinfold sites (biceps, triceps, sub-scapular and supra-iliac). Resting blood pressure, echocardiographic scans and a 12-lead ECG were also conducted.

The boys were then placed into 4 race groups (5 individuals in each group) by the Head of Sport at the school, based upon athletic potential and predicted finishing times. On two subsequent days, 4 x 5 km Cross-Country (2 on each day) races took place around the perimeter of the school. To control for any possible circadian variation on running performance, all races took place in the morning, within 1.5 hours of each other. The

environmental conditions on the 2 days ranged from 11-13°C and relative humidity was between 88-94%. Weather conditions were bright and the condition of the cross-country course was fine on both race-days.

Prior to the start of each race, all of the boys were fitted with Polar Heart Rate (Kempele, Finland) monitors to quantify the internal physiological load during the run and pre-race questionnaires were completed. Following this, all athletes underwent a thorough warm-up, which included, jogging, stretching and a series of acceleration runs. Two research assistants used hand-timing to determine the finish times and all runners were given the exact time (45 min post-race and 225 min post-race) that they needed to return to the research team for the post-race measurements. Following the race, all athletes completed a standardised cool-down and all heart rate monitors were removed for data down-loading. All athletes were given a 500 mL sports drink and a carbohydrate energy bar to aid in the post-race recovery. All participants were then told to rest passively, and not engage in any physical activity following the end of the race and the completion of all the testing, four hours later.

Forty five and 225 minutes after the end of the cross-country race, blood pressure and echocardiographic data were obtained from all participants. This process was completed within 20 minutes for each athlete. Body mass (Tanita, Arlington Heights, Ill, USA) was also measured 45 min post-race. The order of post-race testing was linked to the finishing position in the race (i.e. the boy who finished first, underwent post-race testing first).

Echocardiographic Measurements:

All echocardiographic procedures at pre- and post-race were performed by a pediatric cardiologist (TWR). Measurements of resting LV dimensions and wall thicknesses were

obtained by M-mode echocardiography using two-dimensional guidance (VividQ Ultrasound System, GE Ltd, Horton, Norway), with subjects in the left lateral decubitus position. Images were recorded over 5 s of spontaneous respiration. In the parasternal long axis view, immediately distal to the tips of the mitral valve leaflets, LV end diastolic dimension (LVED) was measured from the posterior edge of the ventricular septum to the endocardial surface of the posterior wall, coincident with the Q wave of a three-lead ECG, inherent to the ultrasound system. LV end systolic dimension (LVES) was measured as the shortest distance between septal and posterior walls during systole. LV Fractional shortening (LVFS%) was calculated as (LVED – LVES)/LVED X 100 [11].

Peak blood flow velocities across the mitral valve during early (E) and late (A) diastole was determined using pulsed-wave Doppler interrogation of transmitral blood flow. The Doppler sample volume was placed at the tips of the open mitral valve leaflets, in an apical 4-chamber view, parallel to flow, guided by colour flow mapping. Pulsed-wave, tissue Doppler imaging was employed to determine peak longitudinal mitral annular septal tissue velocities in systole (S'), early (E') and late (A') diastole. The apical 4-chamber view was used, with care taken to ensure longitudinal septal motion in line with the transducer beam and sample volume. E/E' was calculated as an estimate of LV filling pressure and thus preload.

All images were digitally stored and analysed off-line with software inherent to the ultrasound system. The average of three-five consecutive cardiac cycles was calculated and recorded. Arterial blood pressure was recorded in the left arm by an automated blood pressure cuff (Boso, Bosch, Jungingen, Germany). Heart rate was assessed by 12-lead electrocardiogram (Schiller AT10+, Baar, Switzerland).

Post-hoc analysis of apical 4-chamber clips were completed using dedicated software (Echopac, GE Ltd, Horton, Norway). Longitudinal myocardial strain (ε) and strain rate (SR) of the LV septum was completed using semi-automated speckle tracking software. Peak longitudinal ε as well as peak strain rates in systole (SSR), early (ESR) and late (ASR) diastole were calculated for 3 septal wall segments (base, mid and apex) and then averaged.

Statistical Analyses

Findings were expressed as mean ± standard deviation. Significance of changes in variables over time were examined by a one-way, repeated measures (pre-race day, 45 min post-race and 225 min post-race) ANOVA, with post-hoc comparisons by Bonferroni-corrected t-tests. Heart rate at 45 min and 225 min post-race were used as co-variates when analysing changes in the E:A ratio and LVFS from pre-to post-race. Statistical significance was defined as $P \leq 0.05$. SPSS version 20 (Chicago, IL) was utilised for all statistical analyses.

Results

The mean 5 km race time for the four groups was 22.70 ± 2.68 minutes, with a range of 18 min: 28 s to 27 min: 14 s. The mean heart rate at the end of the race was 200 ± 9 beats·min^{-1}, with a range from 185-214 beats·min^{-1}.

There was a significant ($P<0.05$) increase in body mass, 45 min after exercise (Pre Race: 59.1 ± 8.2 kg vs. 45 min Post-Race: 59.4 ± 8.2 kg). Heart rate was significantly elevated at both 45 min and 225 min post-race compared to pre-race values (Table 2). There was no alteration in LV dimensions across the study period and this was concomitant with no changes in pre-load (E:E') from pre-to post-race. Despite this, there was a significant increase in LVFS at 225 min post-race compared to both pre-race and 45 min post-race measurements. These

differences were abolished, however, when fractional shortening changes over time were statistically adjusted, using post-race heart rate as a co-variate. Peak S' tissue velocities were not altered across the study period (Table 2). Data for peak longitudinal ε, as well as SSR were not significantly different from pre-to-post race (Table 4).

Doppler and tissue Doppler data for LV filling indices are reported in Table 3. There was no change in peak E filling velocities across the study. Peak A-wave filling velocity was significantly higher at both 45min and 225 min post-race compared to the pre-race value. Consequently, there was a significant decrease in the E:A ratio at both 45 min and 225 min post-race compared to the pre-race value. When the E:A ratio was adjusted for post-exercise heart rates at both 45min post-race and 225 min post-race, all pre- to post- race differences in the E:A ratio were abolished. There were no alterations in diastolic tissue-Doppler velocities or diastolic SR (Table 4).

Individual changes in LVFS (Fig 1) demonstrated that some individuals deviated slightly from the general cohort response. The first and second fastest runners had an increase in LVFS 45 min after the race. Longitudinal ε (Fig 2) in four athletes was elevated 45 min post-race but with a rebound decline at 225 min post-race but this was not related to their finishing times.

Individual data for E' (Fig 3) demonstrated some variability over quite a small data range. The second fastest runner in the cohort demonstrated a decline in E' from pre to to 45min post race and 45 min to 225 min post-race. Individual data for ESR and ASR demonstrated one unique response (Fig 4 and 5). The fastest runner in the cohort over 5 km (18 min: 28 s)

demonstrated blunted ESR and ASR responses, 45 min post-race. These returned to pre-race levels 225 min post-race.

Discussion

The major findings from this study were: 1) an elevation in LVFS during later recovery post-race, which was abolished after adjusting for post-race HR 2) no significant changes in S', or longitudinal ε during systole pre-to-post race, 3) a decrease in the E:A ratio from pre-to post-race, which was abolished after adjusting for post-race heart rates, with limited changes in diastolic tissue velocities and SR, and 3) evidence of some heterogeneity in individual LV responses post-race that may be mediated by fitness/running speed.

The 5 km race had no apparent effect on pre-load, based upon the lack of change in: body mass, LVED and E:E'. The increase in body mass 45 min post-race, compared to pre-race levels, was a product of the standardized fluid intake that all runners consumed at the cessation of the race. Consequently, it is possible to speculate that plasma volume was maintained and, therefore, reduced venous return (altered pre-load) was not a factor in determining post-race cardiac function.

Overall, the findings with regard to global measures of systolic function in the extant literature are equivocal. In the present study, global systolic function, as reflected by FS, was transiently elevated following the race, in the absence of any change in LV structure or volume. After adjusting for post-race HR, however, these differences were abolished. Rowland et al., [12] demonstrated no significant change in LV FS following a 4 km road race. Global systolic function in the form of EF has also been demonstrated to be elevated post-race, in adult marathon runners [13]. These authors speculated that this was a

compensatory mechanism for the reduced LV dimensions noted after the race. Whereas, Nie et al., [1] demonstrated a reduction in EF in adolescent athletes, following two 45 min runs, with no change in LVED. Across all the studies cited here, the changes in systolic function were deemed clinically insignificant by the various research groups and may well be related to elevated HR at post-exercise assessments. Post-exercise tachycardia was confirmed as the basis for the elevated FS response in the present study. The present study demonstrated that there was no change in TDI derived systolic measures or longitudinal strain (peak) post-race, therefore intrinsic contractility was not altered by the 5 km race. These findings are in direct contrast to the pattern of systolic change noted in events of greater volume and duration in child and adult populations [9, 14]. Using tissue-Doppler imaging, reductions in septal and lateral wall peak deformation (strain) were noted in adult marathon runners following the completion of the Boston Marathon [14]. Altered (blunted) regional LV systolic function, were also noted in young, child tri-athletes [9] after the completion of age-group events (pre-race: -18.9% vs post-race: -16.9%). Consequently, it is possible to speculate that transient changes in peak strain may be a product of temporary myofibril dysfunction during more prolonged endurance events for adult and adolescent athletes. The implication of this lack of regional systolic functional change in the present study suggests that these athletes can cope with the stress of a 5 km race relatively easily and recover to baseline quickly.

A lack of change in E, which is indicative that early relaxation patterns are normal post-race, was accompanied by an increase in A post-race. This led to an overall decrease in the E:A ratio. On first evaluation, this could suggest that there was an alteration in the diastolic filling characteristics of the LV following the race [15]. Confounding variables such as altered pre-load (reduced venous return) and elevated post-exercise heart rate will influence the E:A ratio [19]. After adjusting, for post-exercise heart rates, the decrease in the E:A ratio post-race was abolished and this suggests that post-race tachycardia was the primary, explanatory variable,

for the decrement seen in the E:A in the present study [16]. A similar decrement in post-race E:A ratio, as seen in the present study, was noted by Nie at al., [1] in young adolescent runners (14.5 years). These researchers evaluated LV function following 2, 45 min, constant-load runs on a treadmill at a physiological load of between 167-171 beats·min^{-1}. They demonstrated that there was no effect of pre-load or heart rate on the E:A ratio and, therefore, an intrinsic blunting of the relaxation properties of the ventricle could have occurred. It is possible to speculate that transient, reversible, cardiomyocyte damage (stunning) may have occurred and this may play an important role in the alteration in LV function post-race [17]. This pattern of reduced E:A ratio post-race was also noted in younger athletes (mean age 12 years) competing in mini- triathlon events [9]. These authors, however, did not adjust the data for elevated post-race heart rates. Consequently, post-race tachycardia could have been the determining factor for the reduced E:A ratio noted in this study. While, this depression in E:A ratio has been noted in several studies, it is unlikely that this will have significant clinical implications for the young athlete.

This lack of change in LV relaxation patterns (compliance and suction) post-race was also confirmed by the lack of difference in E' pre-to post-race and this absence of change in downstream LV filling factors [18] was also noted following an age-group triathlon in child and adolescent athletes [9]. These results are in marked contrast to the findings in adult athletes following prolonged exercise [19, 20], where significant reductions in early LV diastolic tissue velocity (E') have been noted. The lack of significant changes in ESR and ASR following the race also support the argument that diastolic function was preserved following the 5 km race. These findings are in direct contrast to evidence drawn from the adult literature; where speckle tracking methodologies have demonstrated reduced diastolic strain rates following marathon [6] and ultra-endurance events [5].

One unique finding did emerge from the analyses of the individual responses to the 5km race. The fastest runner in the cohort (18 min 28s for 5 km) demonstrated higher levels of LV compliance pre-race and a significant reduction in myocardial relaxation during early and late diastolic filling, forty five minutes after the end of the race. Therefore, the speed of myocardial relaxation and consequently LV compliance was transiently reduced in this high performance athlete and this does mimic the findings noted following marathons and ultramarathons in adult athletes [3]. Furthermore, this reduction in the rate of myocardial relaxation (increased LV stiffness) was concomittant with an increase in fractional shortening in this athlete. Therefore, this transient reduction in LV compliance was compensated for by an increase in contractility to maintain cardiac output following the race. This reduction, however, led to no adverse clinical outcome in this adolescent runner. This data may also suggest that the intensity of exercise (mean, end-race heart rate for this individual was 200 beats·min^{-1}), is as important as the exercise volume, in triggering transient reductions in cardiac function in young, adolescent runners.

Overall, this study is reflective of both the distance (5 km) and the type of participants that routinely take part in this type of activity at school and the evidence suggests that an acute bout of exercise, at a high exercise intensity, does not lead to any significant, post-exercise attenuation in cardiac systolic and diastolic function in sports-trained adolescent athletes. There were statistically significant changes, but the magnitude of these changes were very small and not biologically important. The recommendation for physical educators is that for sports-trained, active adolescents, taking part in high-intensity activities, which form part of the normal physical education curriculum, these activities should not be contra-indicated.

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Figure Captions

Fig 1 Individual changes in Shortening Fraction: Pre-and post-race

Fig 2 Individual changes in Peak Longitudinal Strain: Pre-and post-race

Fig 3 Individual changes in Septal E': Pre- and post-race

Fig 4 Individual changes in Early Diastolic Strain Rate: Pre-and post-race

Fig 5 Individual changes in Late Diastolic Strain Rate: Pre-and post-race