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ORIGINAL ARTICLE

Evidence of increased electro-mechanical delay in the left and right ventricle after prolonged exercise

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Abstract We assessed the time delay from the onset of QRS (Q) to peak systolic (S′) and diastolic (E′) tissue velocities in the left (LV) and right ventricle (RV) before and after prolonged exercise. Nineteen well-trained runners (mean ± SD age, 41 ± 9 years) had tissue-Doppler echocardiography performed before and after an 89 km ultra-marathon race. Longitudinal tissue motion was analysed in LV basal and mid-wall segments and RV free wall. Electro-mechanical coupling was assessed by the delay between Q and S′ as well as E′ tissue velocities. Average data for all segments were adjusted for the R–R interval. Comparisons were made by paired *t*-tests. An increase in electro-mechanical delay (EMD) was reported post-exercise in systole (Q–S′ LV: 131 ± 20 vs. 175 ± 27 ms; RV: 171 ± 34 vs. 258 ± 35 ms; $P < 0.05$) and diastole (Q–E′ LV: 486 ± 51 vs. 647 ± 44 ms; RV: 500 ± 80 vs. 690 ± 75 ms; $P < 0.05$). Further, post-race peak tissue velocities in basal LV and RV wall segments were reduced ($P < 0.05$). Recovery from prolonged running was associated with an increased "EMD", and reduced peak tissue velocities, in both ventricles.

Keywords Echocardiography · Systole · Diastole · Cardiac fatigue

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Introduction

Acute strenuous exercise can result in skeletal muscle fatigue that has been associated with an increase in the electro-mechanical delay (EMD; Horita and Ishiko 1987; Zhou 1996). The possibility that acute exercise could result in myocardial muscle fatigue was not contemplated until recently (George et al. 2008). Despite a growing evidence base that LV and RV function may be altered during the early recovery period from prolonged exercise (Shave et al. 2008), the concept of "cardiac fatigue" remains controversial (Shave et al. 2008) and putative mechanisms are not understood (Scott and Warburton 2008).

Tissue-Doppler imaging (TDI) provides the capability to assess both; (1) global and segmental myocardial tissue velocities during systole and diastole, and (2) the time delay or EMD from electrical signal (onset of the QRS complex of the ECG) to peak of systole (S′) or early diastole (E′) wall motion. Reduced peak tissue velocities after prolonged exercise have been reported (e.g. George et al. 2005; Neilan et al. 2006a, b; La Gerche et al. 2008).

The measurement of cardiac EMD in athletes who complete prolonged exercise has not been performed, although Sahlen et al. (2009) have suggested ECG time-intervals may be increased after prolonged exercise. Cardiac EMD data could provide important mechanistic insights with respect to "exercise-induced cardiac fatigue."

Cardiac timing data (onset QRS to peak S′) using TDI (Yu et al. 2003) has been used to assess both intra-ventricular (LV segment to LV segment) and inter-ventricular (LV to RV segments) synchrony in healthy subjects (Ng et al. 2008) and patients with heart failure (Yu et al. 2003). Despite evidence that the impact of prolonged exercise on ventricular function may induce segmental specific changes in wall function (Douglas et al. 1990b; George et al. 2009), there has been no attempt to quantify movement (dys)synchrony within or between ventricles as a consequence of acute endurance exercise. The primary aim of this study was to assess EMD in the LV and RV, alongside measures of S′ and E′, before and after prolonged exercise. A secondary aim was to determine the degree of intra (LV) and inter (LV–RV) ventricular wall motion synchrony after prolonged exercise.

Methods

As described in a previous study (George et al. 2009) we recruited, by advertisement at pre-race registration, 19 highly-trained ultra-marathon runners (16 males; mean ± SD age, 41 ± 9 years; body mass 71.9 ± 9.3 kg; height 1.72 ± 0.09 m; training 12.2 ± 6.3 years and 88 ± 50 km week^{-1}). The Comrades Marathon (89 km) is run between Pietermaritzburg and Durban in South Africa. Race pace was self selected and reflected in a broad range of finishing times (404–757 min). Environmental conditions were still, no precipitation and the temperature was 20°C at midday. Ethics approval was obtained locally and written informed consent was provided by all participants prior to testing. Exclusion criteria included any personal and/or early family history of cardiopulmonary disease, including diagnosis and treatment for hypertension, angina, myocardial infarction, and peripheral vascular disease.

Design

In a repeated measures design, all subjects underwent echocardiographic scans up to 24 h prior to the race and within 60 min (34 ± 10 min) of race completion (in the medical tent). With subjects supine resting brachial artery blood pressure was assessed twice via standard auscultation. Body mass was assessed using portable scales (Model A3JJT1K, Hansen, UK) with participants in running shorts and vest after being towel dried.

Data collection

A single experienced sonographer was used for all image acquisitions. Ventricular function measurements were made in the left lateral decubitus position. The full scan protocol has been reported previously (George et al. 2009) and included the biplane assessment of ejection fraction (EF) and the Doppler flow assessment of the ratio of peak early to peak atrial trans-mitral filling velocities (E/A). Scans were performed with a commercially available ultrasound system (Vivid 7, GE Medical, Horton, Norway). A single lead ECG was monitored throughout the scan to assess HR and determine the onset of Q-wave as well as to assess QRS duration. Longitudinal myocardial velocities were assessed using colour TDI mode. Adjustment of the colour gain and PRF ensured optimal signal to noise ratio without aliasing, whilst the sector width was reduced to optimize frame rates at greater than 150 FPS. In each view, careful adjustment ensured parallel alignment (<25°) of longitudinal movement with the ultrasound beam. Apical 4-chamber, 2-chamber and long-axis views were utilized to allow image acquisitions of six LV wall segments (septum, lateral, inferior, anterior, posterior and anteroseptal) at both the basal and mid-wall levels. The right ventricular free wall was recorded from an apical 4-chamber orientation at the basal and mid-wall level. All acquisitions were stored over three cardiac cycles, to DVD, for offline analysis.

Data analysis

Offline analysis was performed by a single trained sonographer who was blinded to participant identity. Quantitative analysis (Q-analysis) was utilized to assess longitudinal tissue motion in the basal and mid LV and RV wall segments using EchoPac (GE Medical). A 6 mm × 6 mm sample volume was placed in the middle of the segment of interest. The time to peak S′ and E′ were measured from the onset of the Q-wave from the ECG in all 12 LV and 2 RV wall segments. Global timing data were obtained by averaging all sites in the LV and RV. All timing data were adjusted to the concomitant R-R interval as there was a difference in pre- and post-race heart rate (60 ± 8 to 79 ± 9 beats min^{-1}). Peak tissue velocities were measured during systole (S′), early diastole (E′) and atrial systole (A′) from six basal LV and one RV wall segment. Global peak tissue velocities for the LV were obtained by averaging the six basal-wall segments. Intra-observer reliability for TDI assessment of peak wall velocities as well as timing events were calculated via intra-class correlation (range between 0.693 and 0.993; all $P < 0.05$).

Intra-ventricular synchrony was assessed using the standard deviation of time to peak S′ (T_s) and of time to peak E′ (T_d) from the 12 myocardial segments (Yu et al. 2003), as well as the maximum dispersion between any two of the 12 segments (Bordachar et al. 2004). Inter-ventricular synchrony was assessed by comparing the difference in T_s of the basal free wall RV and basal septal LV segments from pre- to post-exercise.

Statistical analysis

Comparisons between pre- and post-exercise data were made by paired *T*-tests. Delta (post-pre) scores for Q to peak S′/E′ time intervals and peak S′/E′ velocities were correlated via Pearson's product moment analysis. Significance was set at $P < 0.05$ and analysis was performed using software (SPSS v15.0, SPSS Inc, Chicago, IL, USA) with data presented as mean ± SD.

Results

Completion of the race (586 ± 80 min) resulted in a small decrease in body mass (post-race: 71.0 ± 11.3 kg), but a drop in systolic blood pressure (117 ± 11 to 105 ± 6 mmHg; $P < 0.05$). Post-race data for EF (71 ± 5 to 64 ± 6%, $P < 0.05$) and E/A (1.47 ± 0.35 to 1.25 ± 0.30, $P < 0.05$) were reduced. QRS duration did not change from pre- (105 ± 21 ms) to post-exercise (101 ± 25 ms). R–R adjusted timing data for the LV and RV are presented in Table 1. In the LV the time from onset of QRS to peak of S′ and E′ was delayed in virtually all wall segments as well as in global LV scores ($P < 0.05$). Data for the RV followed the same pattern.

Table 1 R–R adjusted timing data (mean ± SD, ms) for segmental and global wall function in the LV and free wall segments in the RV

	Q-peak S′		Q-peak E′	
	Pre-race	Post-race	Pre-race	Post-race
LV				
Septum-basal	134 ± 30	179 ± 39*	476 ± 51	633 ± 43*
Septum-mid	152 ± 45	204 ± 50*	497 ± 58	668 ± 51*
Lateral-basal	122 ± 35	168 ± 34*	468 ± 55	626 ± 51*
Lateral-mid	113 ± 28	156 ± 26*	480 ± 63	658 ± 59*
Inferior-basal	131 ± 24	183 ± 39*	457 ± 68	623 ± 42*
Inferior-mid	153 ± 34	197 ± 47*	491 ± 56	657 ± 48*
Anterior-basal	111 ± 19	156 ± 26*	488 ± 59	643 ± 46*
Anterior-mid	111 ± 19	156 ± 25*	493 ± 59	664 ± 48*
Posterior-basal	136 ± 45	166 ± 37	477 ± 50	622 ± 55*
Posterior-mid	124 ± 33	166 ± 35*	494 ± 55	642 ± 58*
Anteroseptal-basal	141 ± 37	180 ± 47*	501 ± 50	659 ± 57*
Anteroseptal-mid	142 ± 37	191 ± 45*	508 ± 48	668 ± 49*
Mean	131 ± 20	175 ± 27*	486 ± 51	647 ± 44*
RV				
Free wall-basal	167 ± 32	259 ± 40*	496 ± 76	679 ± 76*
Free wall-mid	175 ± 39	257 ± 31*	517 ± 82	700 ± 75*

Q-peak S′ refers to Q wave to peak systolic tissue velocity, Q-peak E′ refers to Q wave to peak diastolic tissue velocity

LV left ventricle, *RV* right ventricle

* Pre- to post-race difference $P < 0.05$

Segmental peak tissue velocities for the LV and RV are presented in Table 2. In the LV, due to a velocity reduction in a number of basal segments; S′ (3/6), E′ (5/6) and A′ (2/6), mean basal peak S′, E′ and A′ were reduced post-race. In the basal RV free-wall a reduction in S′, E′ and A′ was observed post-race ($P < 0.05$). The data for mean basal peak velocity and time to peak S′ and E′ are combined in Fig. 1. This provides a clear representation that reduced peak S′ and E′ is associated with delay in time to peak S′ and E′ in both the LV and RV. Delta values for time from Q to peak S′ and peak S′ velocity were correlated ($r = -0.787$, $P < 0.005$) in the LV (Fig. 2) but not in the RV ($r = -0.293$). Delta values for time from Q to peak E′ and peak E′ velocity were not correlated ($r = -0.198$) in the LV (Fig. 3) or RV ($r = -0.244$).

Within the LV the SD of individual variability in Ts (29 ± 12 vs. 27 ± 17 ms; $P = 0.70$) and T_d (25 ± 13 vs. 30 ± 8 ms; $P = 0.10$) did not change pre- to post-race. Whilst both minimum and maximum T_s were greater after prolonged exercise, the R–R adjusted individual dispersion (maximum T_s to minimum T_s) across the 12 LV wall segments was not changed (86 ± 35 to 83 ± 46 ms). Data for T_s in both RV basal free wall (167 ± 32 vs. 259 ± 40 ms; $P < 0.005$) and LV basal septum (123 ± 25 vs. 170 ± 26 ms; $P < 0.005$) increased pre- vs. post-exercise. The change in T_s was greater in the RV (82 ± 45 ms) than the LV (36 ± 26 ms; $P < 0.005$) suggesting a greater absolute and relative delay in RV activation post-exercise.

Discussion

As with other studies of prolonged exercise performance this study reported a decline in global measures of systolic (EF) and diastolic (E/A) function of the LV during post-race recovery. In addition, and the unique finding from the current study, the time from onset of QRS to peak S′ and E′ tissue velocities were lengthened after a bout of prolonged strenuous exercise, consistent with an increased cardiac EMD. The delay was concomitant with a decline in basal wall segment peak S′ and E′ tissue velocities in both LV and RV.

Timing

We report a lengthening of the R-R adjusted time from onset of the electrical signal (Q-wave of the ECG) to the onset or peak velocity of wall segment motion during systole and early diastole, which is independent of QRS duration. The time from onset of Q-wave to peak S′ was lengthened by c. 44 ms or 34% and was consistent across all wall segments at both basal and mid-wall levels suggesting a global change in time intervals. As the cardiac time

Table 2 Basal segmental peak tissue velocity data (mean ± SD, cm s^{-1}) for the LV and basal free wall data for the RV

	Peak S′		Peak E′		Peak A′	
	Pre-race	Post-race	Pre-race	Post-race	Pre-race	Post-race
LV						
Septum	7.50 ± 0.98	6.65 ± 1.12*	9.75 ± 1.63	7.85 ± 1.62*	7.45 ± 1.91	6.26 ± 2.19*
Lateral	8.52 ± 1.71	8.47 ± 1.90	12.18 ± 2.49	10.30 ± 2.50*	5.85 ± 2.36	5.49 ± 3.06
Inferior	7.87 ± 1.17	6.79 ± 1.82*	10.57 ± 2.78	9.13 ± 2.39*	7.27 ± 2.23	6.81 ± 3.34
Anterior	8.52 ± 2.74	7.87 ± 2.13	10.95 ± 2.82	10.19 ± 1.97	6.85 ± 1.70	6.14 ± 2.47
Posterior	7.66 ± 2.14	6.45 ± 2.64*	11.17 ± 3.03	8.15 ± 3.31*	5.31 ± 2.68	4.99 ± 4.02
Anteroseptal	7.68 ± 1.46	7.22 ± 1.26	9.34 ± 1.86	7.44 ± 1.55*	7.23 ± 1.95	5.55 ± 1.85*
Mean	7.96 ± 1.20	7.23 ± 1.45*	10.66 ± 1.99	8.84 ± 1.75*	6.66 ± 1.81	5.88 ± 2.43*
RV						
Free Wall	11.55 ± 2.53	9.52 ± 2.11*	11.60 ± 3.67	7.55 ± 2.98*	10.61 ± 4.17	6.66 ± 3.55*

Peak S′ referes to systolic peak tissue velocity, peak E′ referes to early diastolic peak tissue velocity, peak A′ refers to atrial diastolic peak tissue velocity

LV left ventricle, *RV* right ventricle

* Pre- to post-race difference $P < 0.05$

interval to peak S′ has lengthened it comes as no surprise that the subsequent time interval to peak E′ was also increased and consistent in all segments. The current data provide additional support to the observation of Sahlen et al. (2009) that prolonged exercise resulted in an increase in QT(c) and the time interval from peak-end of the T-wave, which is associated with ventricular repolarization. The same pattern of timing changes also occurred in the two RV wall segments post-exercise. The lengthening of these periods suggests a cardiac EMD that is prolonged after ultra-endurance exercise.

Skeletal muscle EMD lengthens after acute exercise (Horita and Ishiko 1987; Zhou 1996). Speculative mechanisms for this reduction include reduced skeletal muscle membrane excitability, reduction in cytosolic Ca^{2+} concentration, reduced myofibrillar Ca^{2+} sensitivity and/or metabolic derangement (Zhou 1996). Similar mechanisms could be working in cardiac muscle after prolonged exercise although "membrane excitability" is an unlikely candidate as QRS duration was not altered. "Downstream" effects within the cardiomyocyte are more likely and thus could reflect changes in calcium mobilization, sequestration, down-regulation of energy turnover and myofibrillar ATP-ase activity or a combination of factors. Despite this speculation the current data do point to an intrinsic source for the changes in cardiac function observed during recovery from the completion of a prolonged bout of exercise. Zhou (1996) found a recovery to baseline skeletal muscle EMD within 10 min of completion of an acute bout of exercise. The recovery time of cardiac EMD after prolonged exercise should be studied but is likely to be longer after more prolonged exercise.

The post-exercise increase in cardiac EMD was not correlated with changes in EF and E/A. The lack of a correlation may not be too surprising, given that EF and E/A as global functional indices are influenced by factors other than the degree of EMD (e.g. load, heart rate, compliance).

Peak tissue velocities

The prolonged exercise undertaken in the current study resulted in a decline in segmental and mean basal peak S′, E′ and A′ tissue velocities in the LV. A decline in E′ after a 42-km marathon has been widely reported in a number of basal wall segments (George et al. 2005; Oxborough et al. 2006; Neilan et al. 2006a, b; Hart et al. 2007), although these studies have tended to report limited changes in S′. The drop in basal S′ in the current study could reflect the extra cardiac work associated with a longer running distance of 89 km. Others have speculated that diastolic changes after prolonged exercise may occur earlier than alterations in systolic function (George et al. 2005; Middleton et al. 2007), possibly because of reliance of diastolic function on a smaller (endocardial) proportion of the myocardial mass. The assessment of peak tissue velocities after exercise of greater duration than a 42-km is limited (La Gerche et al. 2008). After an Ironman Triathlon, La Gerche et al. (2008) only reported changes in basal septal E′ in those with noticeable wall motion abnormalities and thus comparison to the current study is difficult.

Whilst the analysis of RV performance after prolonged exercise has received less attention than the LV, a post-race decrease in S′, E and A′ was observed in the basal free wall segment and broadly supports studies that employed 2D

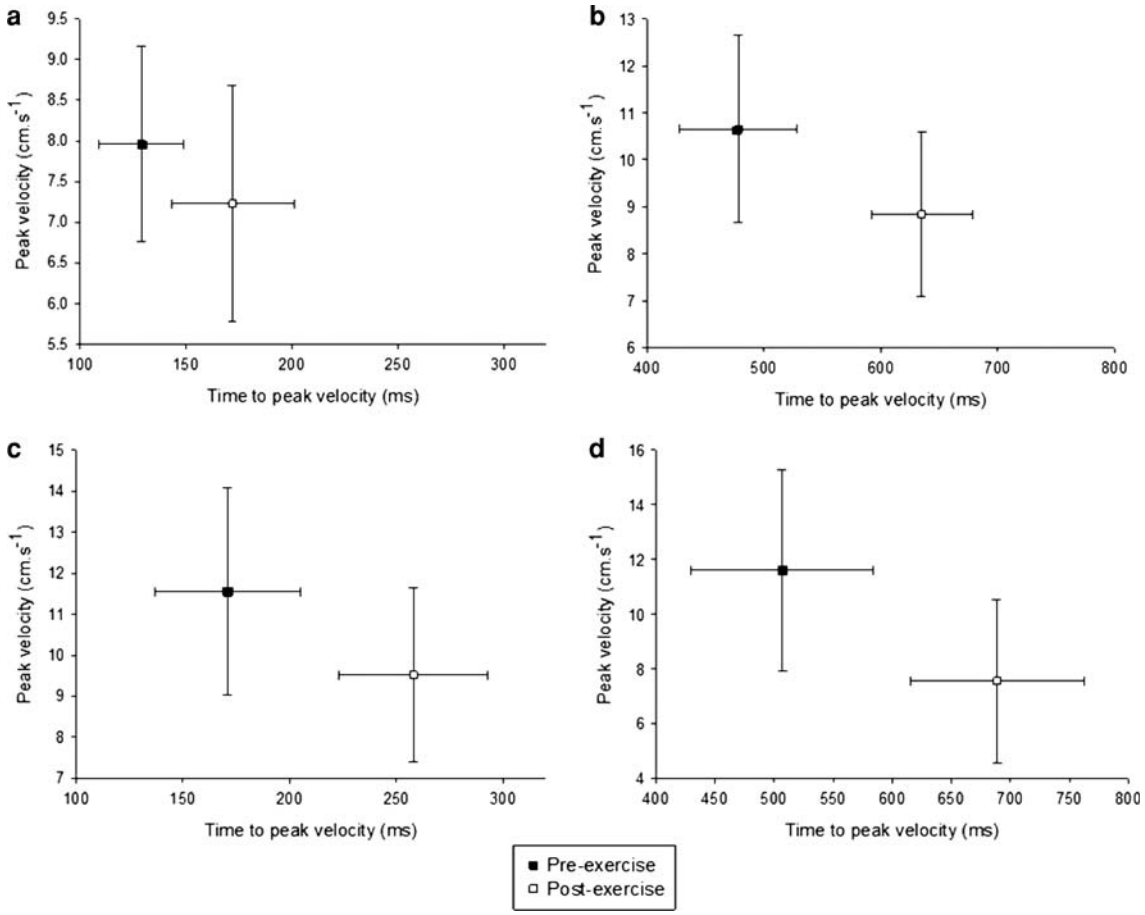

Fig. 1 Composite plot of the impact of prolonged exercise upon **a** basal peak S′ tissue velocity and time from QRS onset to peak S′ tissue velocity in the LV (significant increase in time to peak S′), **b** basal peak E′ tissue velocity and time from QRS onset to peak E′ tissue velocity in the LV (significant decrease in peak E′ and increase in time to peak E′), **c** peak basal free wall S′ tissue velocity and time from QRS onset to peak S′ tissue velocity in the RV (significant decrease in peak S′ and increase in time to peak S′) and **d** basal free wall peak E′ tissue velocity and time from QRS onset to peak E′ tissue velocity in the RV (significant decrease in peak E′ and increase in time to peak E′)

and Doppler techniques (Douglas et al. 1990b; Davila-Roman et al. 1997). To date to report RV TDI data after prolonged exercise, Oxborough et al. (2006) reported no changes in S′ and a similar reduction in E' in both the RV and LV after a 42-km marathon, whereas La Gerche et al. (2008) reported a drop in RV basal free wall S′ (below 11.5 cm s^{-1}) in more athletes after an Ironman triathlon (7/26) than before (1/26). A relatively larger increase in RV work during exercise, compared to the LV, might underpin differential ventricular responses after prolonged exercise (Douglas et al. 1990a). Despite this, evidence for a differential effect of prolonged exercise on the RV, compared to the LV function, is not broadly supported by the current study.

The mechanisms responsible for the decline in LV and RV peak tissue velocities after exercise may relate to alterations in haemodynamic loading or heart rate that occur at the post-race assessment. As observed in an initial report (George et al. 2009), indices of LV preload (end-diastolic dimension, area and volume) were not altered post-race and estimates of afterload (blood pressure, wall stress) were reduced. The relationship between heart rate and tissue velocities, within the current range of data, is such that an elevated heart rate post-exercise should result in an increase in S′, E′ and A′ (Giannaki et al. 2008). It seems unlikely, therefore, that a reduction in preload or an increase in heart rate could account for the reductions in S′, E′ and A′ observed post-race. Whilst there have been some data linking changes in ß-adrenergic receptor sensitivity to post-exercise changes in systolic function (Hart et al. 2006; Scott et al. 2007), these parameters were not assessed in the current study and have not previously explained changes in diastolic function after prolonged exercise (Hart et al. 2007). A recent review (Scott and Warburton 2008) speculated about other potential mechanisms; altered calcium handling and other intracellular metabolic changes, but limited data exist for an intrinsic change in human cardiomyocyte function after prolonged exercise. The link to an increased EMD investigated in the current study provides another potential explanation. The change in both the timing and velocity data, combined with the correlation in the

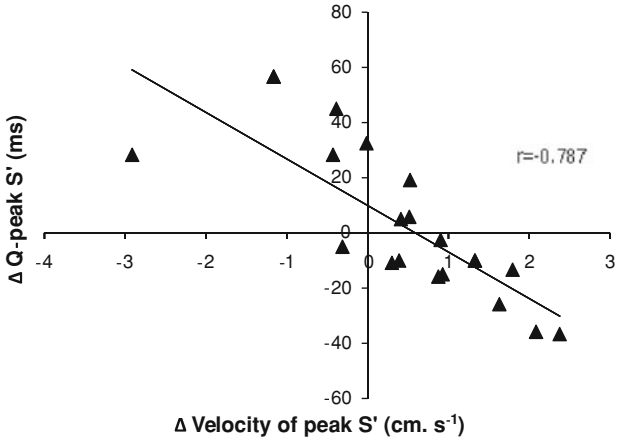

Fig. 2 Scatter plot of delta scores for peak S′ tissue velocity and time from QRS to peak S′ in the LV

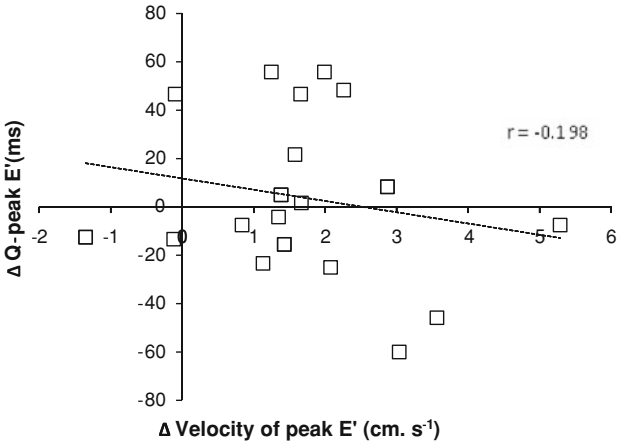

Fig. 3 Scatter plot of delta scores for peak E′ tissue velocity and time from QRS to peak E′ in the LV

LV of delta scores for these variables suggest some association that requires follow-up study.

Correlation of the delta values of the time interval for Q to peak E′ and peak E′ tissue velocity were not significant. This suggests that the lengthening of the time interval to peak E′ was not responsible for the decline in peak E′ tissue velocity post-race. Because peak E′ is largely dependent on the pressure gradient between the left atria and LV rather than any electrical stimulated event, this outcome was expected. The decline in peak E′ post-race may be related to other intrinsic relaxation properties and this requires of further study. The fact that it is concomitant with an increase in Q to peak E′ could be due to the earlier influence on time to S′ which of course precedes early diastole.

Synchrony

Although time intervals were lengthened post-exercise in the LV, the relative dispersion of those time intervals did not change pre-post exercise. Both the SD of T_s and the maximal T_s dispersion across 12 LV wall segments did not change post-race. These data suggest, contrary to some previous evidence (Douglas et al. 1990b; George et al. 2009), that prolonged exercise does not result in significant intraventricular wall segment activation dys-synchrony. The greater absolute and relative increase in RV Ts after the race, compared to the LV, suggests some degree of dyssynchrony between the ventricles.

Limitations

Several possible limitations might apply to the present study. The sample size is small and relatively homogeneous for fitness and encompasses only three female athletes. Further, due to some logistical constraints we could only assess runners immediately post-race and thus studying the time-course of change and recovery post-exercise was not possible. In future investigations, a greater number of time-points post-exercise will inform current debate over the persistence of these changes (Neilan et al. 2006b).

Conclusions

The current study, assessed LV and RV timing delay from electrical to mechanical activity in both systole and diastole as well as peak tissue velocities in multiple wall segments. After performing an 89-km road race, runners demonstrated a lengthened interval from onset Q to peak S′ and peak E′ that was associated with reduced peak velocities in both ventricles. Concomitant with no change in QRS, this suggests a mechanism intrinsic to the myocyte that may be responsible for “exercise-induced cardiac fatigue” specifically during systole.

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