Journal of Sports Sciences

ISSN: (Print) (Online) Journal homepage: www.tandfonline.com/journals/rjsp20

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To cite this article: Rasoul Dokht Abdiyan, Anita Sadeghpour, Azin Alizadehasl, Azizeh Ahmadi, Daryoush Saed, Ali Asghar Ravasi, Ali Akbarnejad, Majid Maleki, Akram Shariati, Atabak Shahed & Mahdiye Aziminia (2023) Effect of resistance exercise on cardiac perturbations and systolic performance: A cross-over randomized trial comparing volumes and techniques, Journal of Sports Sciences, 41:12, 1196-1206, DOI: 10.1080/02640414.2023.2260636

To link to this article: https://doi.org/10.1080/02640414.2023.2260636

JOURNAL OF SPORTS SCIENCES
2023, VOL. 41, NO. 12, 1196–1206
https://doi.org/10.1080/02640414.2023.2260636

Routledge
Taylor & Francis Group

PHYSIOLOGY AND NUTRITION

Effect of resistance exercise on cardiac perturbations and systolic performance: A cross-over randomized trial comparing volumes and techniques

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ABSTRACT

This study investigated the magnitude and time-course of resistance exercise (RE) technique induced transient cardiac perturbations. Twenty-four participants were assigned to one of four arms: sets to failure or non-failure with 8–10 repetition maximum (RM), and sets to failure or non-failure with 15RM. Echocardiographic and blood pressure (BP) data were recorded at baseline and 30 min, 6 h and 24 h post-exercise. In all groups end-systolic circumferential wall stress (cESS), and ratio of transmitral inflow velocities (E/A) were significantly decreased while posterior wall thickness (PWT), global circumferential strain (GCS), GCS strain rate (GCSR), global longitudinal strain rate (GLSR), and stroke volume (SV) were significantly increased for up to 6 h of follow-up. In the 15RM groups, left ventricular (LV) mass and interventricular septal thickness (IVST) were significantly increased, and left atrial (LA) area was significantly decreased ($p < 0.05$) compared to the 8–10 RM groups. In the 15RM groups, RE decreased global longitudinal strain (GLS) and increased ejection fraction (EF) ($p<0.01$). After RE, transient cardiac perturbations, the reduction in LA compliance, and the improvement in LV myofibril geometry were volume dependent and influenced more by sets to failure technique. RE increased GCS and reduced the afterload, thus helping to preserve SV and EF.

ARTICLE HISTORY
Received 7 November 2022
Accepted 12 September 2023

KEYWORDS
Resistance exercises; afterload; strain; athlete; heart; left ventricular hypertrophy

Introduction

Regular resistance exercise (RE) through modify cardiac structure and function has a wide range of cardiovascular health benefits (Bird et al., 2005; Fleck & Kraemer, 2014). However, it was previously reported that RE, regardless of intensity, reduced left ventricular (LV) systolic function and concomitantly reduced afterload (Stöhr et al., 2017). In addition, previous research has established that exercise volume is the most influential variable in health outcomes, and recent work by Freitas et al. showed that cardiac haemodynamic responses are RE volume-dependent (de Freitas Brito et al., 2019). The relationships among these factors have become important in light of recent evidence suggesting that high-volume endurance exercise induces significant cardiac dysfunction (Kim & Park, 2020; La Gerche et al., 2012; Stewart et al., 2016). However, work by Stewart et al. showed that endurance exercise intensity had an important effect on ventricular dysfunction (Stewart et al., 2017).

Much uncertainty exists regarding the effects of the magnitude of RE volume and intensity. Moreover, debates continue about the best RE techniques to achieve health benefits (Bompa & Buzzichelli, 2019). Sets designed to result in failure are a type of RE done to induce momentary voluntary fatigue, and are among the techniques favoured to enhance muscle strength and hypertrophy. Higher relative force production during sets to failure increases intramuscular and intervascular pressure (Campos et al., 2002; Kraemer et al., 2002). However, non-failure sets at the same intensity and volume are associated with higher-quality performance, lower stress, and shorter recovery times (Haff et al., 2008; Iglesias-Soler et al., 2015). To date the role of RE techniques (sets to failure vs, non-failure sets) in LV mechanical abnormalities and vascular responses has received little attention.

Systemic LV parameters play a critical role in the maintenance of cardiac performance (D'Ascenzi et al., 2014; Negishi et al., 2017). Global longitudinal strain (GLS) and global circumferential strain (GCS) are the parameters most sensitive to early myocardial changes, and were found to be superior predictors of postoperative LV function (Kouzu et al., 2011). The coupling of these two factors enables LV competence to preserve stroke volume (SV) and ejection fraction (EF) from significant fluctuation (Ito et al., 2020). Research with mathematical geometry models suggested that GCS makes a relatively greater contribution to SV and EF (Stokke et al., 2017). Earlier work by Stöhr et al. (Stöhr et al., 2017) showed that RE, regardless of intensity, reduced LV wall stress and vascular resistance while improving cardiac output (Q) and GCS strain rate.

Recent evidence suggests that high-intensity endurance exercising induces reductions in GLS, GCS and SV (Stewart

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Supplemental data for this article can be accessed online https://doi.org/10.1080/02640414.2023.2260636

et al., 2016). In addition, post-ultramarathon running LV mass and interventricular septal thickness (IVST) showed substantial augmentations related to posterior wall thickness (PWT), which occurred alongside decreased SV and EF (Kim & Park, 2020). The nature of acute cardiac perturbations following RE remains unclear and has not been well studied in humans who undertake strenuous RE. Currently, there are no data on the assessment of magnitude, technique, and time-course of RE in inducing cardiac dysfunctions.

The present study was designed to assess the effect of the time-course of the appearance of cardiac perturbations and protective compensatory effects by comparing different RE volumes and techniques, with the aim of facilitating the interpretation of day-to-day chronic adaptations to strength training. The specific hypotheses of this study were that 1) acute high-volume sets to failure would induce perturbations in cardiac function and increase wall thickness for up to 24 h, and 2) RE would increase circumferential shorting and decrease the afterload, as relatively significant contributions to post-exercise cardiac performance.

Materials and methods

Subjects

Twenty-eight young men, age 20 to 25 y were selected by simple random sampling with SPSS software, and each was assigned a numeric code. In order to qualify for inclusion, criteria were included body fat under 15%, BMI under 30 (kg. m^{-2}). Eligibility was verified with a questionnaire to obtain information on their exercise experience and medical history, and to rule out any clinical signs related to cardiovascular disease, medical advice not to perform RE, alcohol use, smoking, and use of ergogenic supplements during the 2 weeks before initial testing. Four individuals were excluded from the study based on their questionnaire responses. The participants included in the study were healthy weight-trained men who had performed regular RE training during the previous 18 months. This study was approved by the Cardiovascular Medical and Research Center, and ethical issues involving human participants were overseen by the Research Ethics Committee in compliance with the 1975 Declaration of Helsinki. The participants signed an Informed Consent Form to become eligible to enter the study.

Study design

The study was designed as a cross-over randomized controlled trial with 24 participants distributed, with limited randomized block allocation, in 4 intervention blocks according to sets to failure and repetition maximum (RM) zones: 1) sets to failure 10RM, 2) non-failure 10RM, 3) sets to failure 15RM, and 4) non-failure 15RM. In each exercise zone, randomization was 1:1. Allocation to sets to failure or non-failure and to different sequences was counterbalanced between the cross-over intervention blocks. This had the advantage of providing a total number of 12 observations in each intervention block. Allocation was concealed by keeping the identification code numbers in a sealed envelope. Neither the participants nor the observer knew which intervention block or sequence generation each participant was allocated to. All participants were instructed to come to the laboratory for 3 separate sessions with a recovery period of at least 72 h between sessions to complete the experimental protocol.

RE test

For 48 h before the start of the study, participants refrained from any form of exercise, and used the first session to become acquainted with the tests. The 10RM and 15RM loads were accurately determined for all RE protocols. Exercise sessions took place between 4:00 p.m. and 6:00 p.m. To increase the reliability of measures, test and retest data were obtained in three or four separate sessions with at least 72 h break between them. The order of RE tasks for the upper and lower body was balanced, and the order across test and retest sessions was balanced (Marupudi et al., 2023; Tai et al., 2023). During test – retest sessions two spotters helped and motivated the participants with verbal encouragement. Test sessions began with a warm-up of 5 min running at 50% to 70% maximum heart rate (HR) followed by two sets of 10 repetitions at 50% estimated 10RM and 15RM load. After a 2-min rest, according to the participant's previous rating of perceived exertion (RPE) and estimated number of repetitions-to-failure, the 10RM or 15RM load was performed (Hackett et al., 2012). Tests were stopped when the participant was unable to reach voluntary sets to failure in the course of repetitions. Three attempts with a 3–5 min rest interval were allowed for all RE tests (Pescatello et al., 2014). Intraclass correlation coefficients (ICC) for test – retest values ranged from 0.92 to 0.98.

Subject characteristics measure

Height and weight data were collected between 7:00 a.m. and 8:00 a.m. with a SEKA 700 column scale and height rod (Hamburg, Germany). To estimate body fat percentage, a Harpenden mechanical skinfold caliper (Baty International RH15 9LB, London, England) and the Jackson – Pollack 4-point skinfold thickness method were used (Durnin & Womersley, 1974).

Blood pressure was measured according to European Society of Hypertension guidelines (Parati et al., 2008).

Beat-to-beat blood pressure measurements

A Finometer Pro photoplethysmograph (Finapress Medical Systems BV, Amsterdam, the Netherlands) was used to obtain beat-to-beat blood pressure (BP) measurements on the left hand. The device was calibrated according to the manufacturer's instructions. The participants were seated with their hand stabilized at heart level, and the finger cuff wrapped around their middle finger and connected to the apparatus; BP was recorded for 7 min. The first 2 min of each recording was used to calibrate the upper-arm pressure adjustment to finger pressure. In the last, 1 min of the 5-min continuous recording were noted for systolic blood pressure (SBP) and diastolic blood pressure (DBP) and mean arterial pressure (MAP) (Schutte et al., 2004).

Echocardiography

Comprehensive transthoracic echocardiography was performed by a level III echocardiologist with an EPIQ 7 ultrasound system (Philips Medical Systems, Andover, MA, USA) scanner. All measurements and quantifications were based on the latest ASE/EACVI guidelines (Nagueh et al., 2016). The device was calibrated and signal-to-noise ratios were adjusted to ensure accuracy. Resting comprehensive two-dimensional (2D) and three-dimensional (3D) images were acquired for a minimum of 10 min from the standard apical and parasternal windows. According to multiple Bland – Altman plots, there was complete agreement between the remeasured data and the initially determined values. The left atrial (LA) area was measured at end-ventricular systole when the LA chamber was at its maximum dimension. Area of the LA with the biplane method of disks (modified Simpson rule) was calculated in long-axis (anterior – posterior diameter) and 4-chamber views (longitudinal and transverse diameters). The LV end-diastolic dimension (EDD) and end-systolic dimension (ESD) were measured through the short axis parasternal view at the mid-papillary muscle level (Baggish et al., 2020). End-diastolic PWT and IVST were measured in the parasternal LV long-axis view (Stokke et al., 2017). End-diastolic relative wall thickness (RWT) was calculated with the formula: $RWT = (IVST + PWT)/EDD$ (Dzudie et al., 2007). LV mass was calculated with the Devereux formula and body surface area (BSA) (Devereux et al., 1986). Philips Advanced Cardiac 3D Quantification software (Philips Medical Systems, Andover, MA, USA) was used to measure LV ejection fractions (EF) (Stokke et al., 2017). Early filling (E-wave) and peak late diastolic (A-wave) velocities were determined in the apical 4-chamber view at the level of the mitral valve leaflets, and the E/A ratios were calculated. Mitral annulus early diastolic velocity (e') was measured at the septal and lateral mitral annulus, and the E/e' ratios were computed (Mizuguchi et al., 2008). Simpson's biplane method was used to measure 2D end-diastolic volume (EDV) and end-systolic volume (ESV). SV was derived from Doppler waveforms acquired at the LV outflow tract. SV was calculated from the cross-sectional area (CSA) of the left ventricular outflow tract (LVOT), and the velocity time integral (VTI) in the LVOT with the formula: $SV = LVOTCSA \times VTI$. Q was calculated as $Q = SV \times HR$. Systemic vascular resistance (SVR) was calculated with the formula: $SVR = (MAP - CVP)/Q$, where CVP is central venous pressure (Kadappu & Thomas, 2015; Mizuguchi et al., 2008; Nagueh et al., 2016). LV meridional wall stress (MWS), end-systolic circumferential wall stress (cESS), LV endocardial fractional shortening (eFS), midwall fractional shortening (mFS), LV contractility, vascular – ventricular coupling (VVC), arterial elastance index (Eal), ventricular end-systolic elastance index (ELVI), stroke volume index (SVI), end-systolic volume index (ESVI), and total arterial compliance index (TACI) were calculated with the relevant formulas summarized in Supplemental-1 (Currie et al., 2019; De Simone et al., 1994; Ito et al., 2020; Mizuguchi et al., 2008; Stokke et al., 2017). LV strain was analysed with a semi-automated speckle-tracking method. GLS and global longitudinal strain rate (GLSR) were determined by averaging longitudinal strain and strain rate from the apical 4-, 3-, and 2-chamber views. GCS and global circumferential strain rate (GCSR) were determined by averaging strain in the parasternal short-axis view (Mizuguchi et al., 2008; Stewart et al., 2017).

RE protocol

The experimental design is shown in Figure 1. We used multi-joint free weights RE respectively include: front squat, bench press, deadlift, push press, forward lunge, weighted pull up, Romanian lift, weighted parallel bar dips, and bent-over barbell row. Warm-up consisted of 8 min running at 50% to 70% maximum HR and two sets of 10–15 repetitions at 50% 10RM.

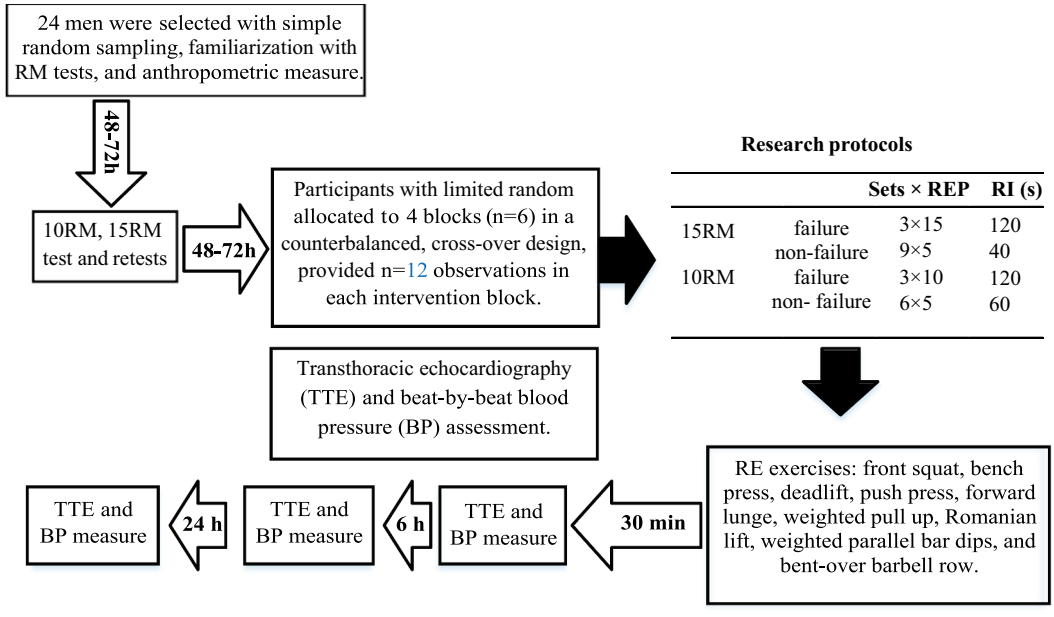

Figure 1. Experimental design of the study.RE: resistance exercise, REP: repetition, RI: rest intervals, RM: repetition maximum.

During the RE protocol participants were reminded not to perform the Valsalva manoeuvre, and were allowed to drink water ad libitum. Sets to failure were performed in three circuits with a 5 min rest between them, adjusted to the rest interval for each RE protocol. Non-failure 15RM exercises were done in 9 circuits with a 5 min rest interval between circuits. Non-failure 10RM exercises were done in 6 circuits with a 5 min rest every two circuits. Five men chosen at random took part in the RE protocol pilot testing. Exercise volume, intensity, and rest interval were balanced between the two techniques in each RM zone. Volume index and intensity index equations were used to calculate total workloads (Supplemental-1) (Haff, 2010).

Statistical analyses

The data were normalized with the Shapiro – Wilk test. Test – retest reliability was calculated as the intraclass correlation coefficients (r). The significance levels for the effect of RE was set at $\alpha<0.05$ according to two-way mixed ANOVA (group × time) for repeated measures. Changes after follow-up periods (pre-test, 30 min post-test, 6 h post-test, and 24 h post-test) and interactions between groups were verified with the Bonferroni post hoc test. Mauchly's test for significance (sphericity) was done with lower-bound (epsilon statistic) correction. Levene's test was used to verify equality of the error variances between groups. All data are reported as the mean ± standard deviation (SD), mean difference (MD), 95% confidence interval for the difference (CI), and effect size (ES). To report ES we used the scale proposed by Rhea (2004): <0.2 trivial, 0.2 to 0.5 small, 0.5 to 0.8 moderate, and ≥0.8 large effect (Rhea, 2004). Cohen's d for within- and between-participant variables was calculated to determine ES (Supplemental-1). Pearson's r correlation coefficient was calculated to identify significant correlations between LV parameters. All data analyses were done with SPSS (IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY), and all graphs were created with GraphPad Prism 8.

Results

RE volume

Table 1 summarizes some of the main characteristics in each study group.

Volume index ($kg/kg^{0.67}$) in the sets to failure 15RM group (1538.31 ± 172.87) was significantly greater (MD = 374.34, CI = 197.98–550.71, d = 2.39, $p = 0.001$) than in the 10RM group (1163.96 ± 138.24).

Volume index in the non-failure 15RM group (1542.66 ± 180.12) was significantly greater (MD = 376.22, CI = 198.29–551.03, d = 2.32, $p = 0.001$) than in the 10RM group (1166.44 ± 141.39).

Acute RE and blood pressure

SBP, DBP, MAP, and SVR measures and comparisons are presented in Figure 2. A comparison of the results shows the greatest decrease (MD = 8.02, CI = 3.99–12.01, d = 1.03, $p = 0.001$) in SBP (119.25 ± 9.94) at 30 min and the greatest decrease (MD = 11.58, CI = 4.26–18.90, d = 1.47, $p = 0.001$) in DBP (61.50 ± 7.74) at 6 h in the sets to failure 10RM group. There was a significant decrease in MAP and SVR after the acute RE, and these decreases were maintained for up to 6 h and 24 h respectively ($p<0.01$).

LV mass and regional wall thickness

Figure 3 provides an overview of the changes in cardiac wall thickness. Significant increases in PWT were seen after the acute RE ($p<0.05$). In the sets to failure 15RM group, PWT (10.90 ± 1.52) showed a marked increase (MD = 1.55, CI = 0.33–2.76, d = 1.38, $p = 0.006$) at 30 min. There was a significant interaction effect in IVST between the two sets to failure zones ($p = 0.025$, d = 0.78). On average, IVST in the sets to failure 15RM group (11.86 ± 1.31) increased significantly (MD = 1.49, CI = 0.20–2.78, d = 1.06, $p = 0.015$) compared to the 10RM group (10.37 ± 1.55) at 30 min. The difference in LV mass between the sets to failure 15RM group (277.33 ± 39.34) and 10RM groups (201.42 ± 37.18) was significant (MD = 51.93, CI = 0.63–103.28, d = 1.03, $p = 0.046$) at 30 min.

As seen in Table 2, RWT increased significantly after the acute RE ($p<0.05$). The increase was greatest (MD = 1.80, CI = 0.54–3.06, d = 1.30, $p = 0.002$) in the sets to failure 15RM group (13.18 ± 1.65) at 30 min. EaI and ELVI decreased significantly after the acute RE, and the decreases were maintained for up to 24 h ($p<0.05$). There was a significant interaction effect in EaI between the sets to failure 15RM and 10RM group ($p = 0.033$, d = 0.75) at 6 h.

Table 1. Baseline demographic characteristics in experimental groups.

Characteristics	10RM group	15RM group	p
Number of athletes	12	12	
Age (years)	23.17 ± 2.48	22.75 ± 2.13	0.66
Height (cm)	177.42 ± 3.70	179.17 ± 4.90	0.27
Weight (kg)	83.53 ± 6.61	85.08 ± 6.37	0.64
Body fat (%)	11.72 ± 1.86	12.86 ± 1.75	0.17
Body mass index ($kg.m^{-2}$)	26.60 ± 1.46	26.47 ± 1.06	0.79
Body surface area (m (Bird et al., 2005)	2.01±.09	2.04±.09	0.48
Training (years)	7.13 ± 1.33	8.01 ± 1.41	0.77
Training (h/week)	14.08 ± 2.77	13.58 ± 2.02	0.62
Cardiac index (L/min/m (Bird et al., 2005)	2.44±.44	2.40±.39	0.81
LV mass index (g/m (Bird et al., 2005)	105.85 ± 19.67	109.24 ± 18.20	0.66

Note: Data are shown as means ± SD.

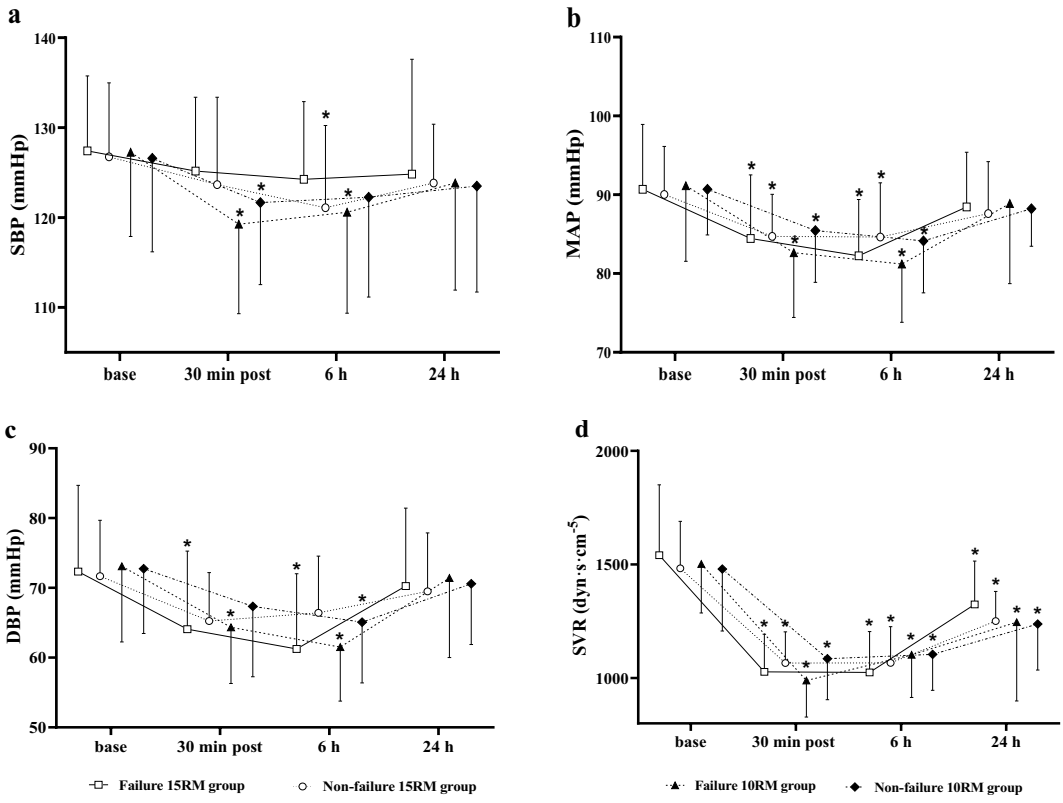

Figure 2. Post-RE changes in blood pressure from baseline at 30 min, 6 hours and 24 hours follow-up. **A**, SBP. **B**, MAP. **C**, DBP. **D**, SVR. Data are presented as means and SD. *denotes significant change compared to baseline (p<0.05).

Hemodynamic responses

There was no evidence that RE had an influence on ESV, ESD, EDD, average e′ or E/e′ ratio (p>0.05) (Supplemental-2). VVC, TACI, mFS, eFS, and contractility measures and their comparisons are presented in Supplemental-2. No significant differences were found during 24 h of follow-up (p>0.05).

The data in Table 3 show that EDV (149.33 ± 19.18) increased in the sets to failure 10RM group (MD = 15.41, CI = 3.84–26.99, d = 0.76, $p = 0.004$) at 30 min, and increased (MD = 16.75, CI = 1.68–31.81, d = 0.82, $p = 0.022$) in the sets to failure 15RM group (150 ± 23.97) for up to 6 h. The difference in LA area between the two RM group was significant for up to 24 h after exercising (p<0.05). There was a significant interaction effect on LA area between the two sets to failure groups ($p = 0.014$, d = 0.83) at 30 min. The LA area in the sets to failure 15RM group (17.66 ± 1.3) was significantly great decrease (MD = 1.75, CI = 0.002–3.51, d = 0.90, $p = 0.037$) compared to the 10RM group (19.42 ± 2.42) at 24 h.

It was clear that RE had a significant effect on HR, SV and Q that lasted up to 6 h (p<0.05). On average, SV showed greater increases (MD = 18.30, CI = 8.36–28.25, d = 2.42, $p = 0.001$) in the sets to failure 15RM (97.90 ± 13.07) and (MD = 13.19, CI = 3.24–23.14, d = 1.81, $p = 0.004$) non-failure groups (93.41 ± 9.96) at 6 h, and these increases remained significant for up to 24 h (p<0.01). EF appeared to be affected and increased (MD = 6.60, CI = 2.50–10.70, d = 0.76, $p = 0.001$) in the sets to failure 15RM (64.52 ± 8.17) and non-failure groups (63.88 ± 5.55) at 6 h (p<0.01). After RE, enhancement of the A wave was noted in 15RM (48.17 ± 9.27) and 10RM (47.25 ± 9.55) groups at 30 min, and a significant decrease in the E wave (81.07 ± 15.1) and E/A ratio (1.87 ± 0.3) was also seen in sets to failure 15RM group, lasting up to 6 h (p<0.05).

The results of correlation analyses after RE are summarized in Table 4. A positive correlation was found between GCS and EF at all follow-up times ($r = 41$, $p = 0.003$). There was a significant positive correlation between GCS and SV at 6 h ($r = 40$, $p = 0.005$).

LV strain and wall stress responses

As shown in Figure 4, cESS decreased significantly after the sets to failure 15RM (139.24 ± 27.59) and 10RM (132.68 ± 26.35) groups, and the decreases lasted up to 6 h (p<0.01). The data shown in Table 2 indicate that MWS decreased significantly in both sets to failure 15RM (94.21 ± 16.43) and 10RM (3.83 ± 0.66) groups (p<0.05).

There was a significant reduction (MD = 1.14, CI=–0.12–2.29, d = 2.15, $p = 0.001$) in LV-GLS (20.72 ± 1.53) in the sets to failure 15RM and (MD = 1.63, CI = 0.48–2.78, d = 1.01, $p = 0.002$) non-failure group (20.01 ± 1.28) at 30 min. A decrease in GLSR was seen after the acute RE, and in the sets to failure 15RM (–0.86 ± 0.21) and 10RM (–0.8 ± 0.2) groups the decrease lasted up to 6 h (p<0.05).

Significant improvements in LV-GCS (31.06 ± 3.99) and GCSR (–2.67 ± 0.67) were seen after the sets to failure 15RM group, and were maintained for up to 6 h (p<0.01).

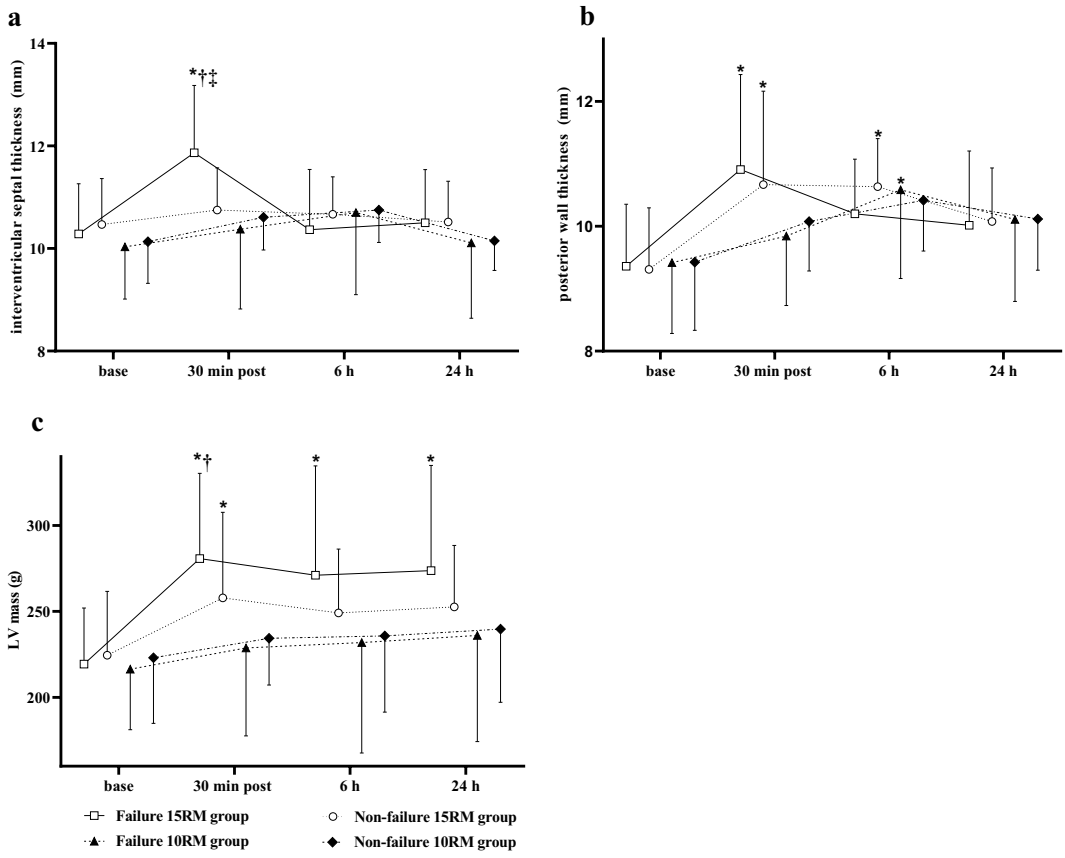

Figure 3. Post-RE changes in cardiac myofibril geometry from baseline at 30 min, 6 hours and 24 hours follow-up. **A**, IVST. **B**, PWT. **C**, LV mass. Data are presented as means and SD. *denotes significant change compared to baseline ($p < 0.05$). † significant compared to CF 8-10RM zone ($p < 0.05$). ‡ significant interaction with CF 8-10RM zone ($p < 0.05$).

Discussion

The main finding of this study is that high-volume RE can transiently perturb LV function and trigger a substantial reduction in LA area compliance lasting for up to 24 h. A further finding of interest is that high-volume 15RM sets to failure and nonfailure induced significant increase in LV myofibril geometry. Another important finding is that RE, regardless of the technique, had successive effects on improvements in GCS, GCSR and afterload. Improvements in GCS seen after RE contributed to preserving SV and EF. Also, the present findings provide a potential explanation for the ventricular and vascular interaction response to post-exercise hypotension (PEH). High-load RE, regardless of differences in volume, intensity or technique, may cause PEH through decreases in cESS and SVR.

Strain entails tissue deformation, and strain rate is the rate of this deformation; both of these effects arise from the interactions between contractile forces and external loading (Murai et al., 2017). The present findings highlight that RE volume is a key factor in devising a daily RE program, given that high-volume RE induced reductions in GLS and LA compliance. In contrast to previous studies, reduced LV function was not accompanied by LA dilation as commonly seen in endurance athletes (D'Ascenzi et al., 2021). This result may be explained by the fact that LV longitudinal dysfunction impaired mitral annular motion towards the apex, and possibly decreased LA cavity volume compliance (Negishi et al., 2017). The linear correlation between LA area and GLS seen immediately after RE is evidence in support of this mechanism. However, it is unknown whether high-volume RE can be beneficial to athletes with LA enlargement. Further research is required to ascertain the advantages of RE for rehabilitation in athletes with LA dilation.

Of note is that after RE, the improvements observed in GCS and GCSR lasted up to 6 h. These results further support the idea that an acute bout of high-volume RE does not reduce systolic LV function. This finding is consistent with results published by Stöhr et al. (2017), who reported that GCS and GCSR were significantly enhanced compared to their baseline values after RE at 60% 1RM (Stöhr et al., 2017). Another notable finding is that RE acutely increased EDV, and an inverse relationship was found between LA area and circumferential strain at 30 min. The mechanisms for this response require further examination; however, the data reported here suggest that RE, through an increase in EDV, led the LV to become more spherical. So, any contribution from circumferential fibres will increase further in relation to longitudinal fibres, and as a result of augmented GCS and GCSR, the myocardium by buffering response compensates any longitudinal dysfunction. Moreover, improvements in GCS and GCSR along with decreased GLS and GLSR may act as a systemic permissive mechanism by limiting LA compliance and thus benefiting rehabilitation in athletes with LA enlargement symptoms.

Table 2. Measures of cardiovascular functional performance during baseline and 24 h of follow-up.

		Baseline	30 min post	6 h post	24 h post
Eal (mmHg ml^{-1} m^{-2})					
15RM group	failure	2.97 ± 0.51	2.62 ± 0.21*	2.33 ± 0.25*‡	2.6 ± 0.4*
	non-failure	2.9 ± 0.43	2.63 ± 0.42	2.37 ± 0.29*	2.52 ± 0.36*
10RM group	failure	3.01 ± 0.63	2.45 ± 0.32*	2.64 ± 0.42*	2.57 ± 0.61*
	non-failure	2.94 ± 0.47	2.54 ± 0.42*	2.62 ± 0.5	2.58 ± 0.45*
ELVI (mmHg ml^{-1} m^{-2})					
15RM group	failure	4.62 ± 0.72	4.31 ± 0.63	4.07 ± 0.66*	4.05 ± 0.51*
	non-failure	4.53 ± 0.9	3.96 ± 0.53*	3.69 ± 0.43*	4.11 ± 0.54
10RM group	failure	4.68 ± 0.75	3.83 ± 0.66*	4.22 ± 1.05	4.16 ± 1.17*
	non-failure	4.44 ± 0.96	3.75 ± 0.57	3.69 ± 0.57*	4.14 ± 0.8*
MWS (%)					
15RM group	failure	111.67 ± 23.08	94.21 ± 16.43*	106.47 ± 14.83	115.11 ± 22.04
	non-failure	110.86 ± 31.51	99.70 ± 22.57	98.23 ± 21.04	105.59 ± 19.96
10RM group	failure	113.84 ± 24.31	101.37 ± 21.52	94.58 ± 21.00*	103.68 ± 24.39
	non-failure	111.86 ± 22.57	101.88 ± 24.82	96.98 ± 20.71	104.04 ± 25.67
RWT (mm)					
15RM group	failure	11.37 ± 0.92	13.18 ± 1.65*	12.17 ± 0.99	11.9 ± 1.2
	non-failure	11.35 ± 1.09	12.74 ± 1.73*	12.66 ± 0.91*	12.06 ± 1.13
10RM group	failure	11.38 ± 1.12	11.87 ± 1.24	12.67 ± 1.66*	12.07 ± 1.39
	non-failure	11.39 ± 1.13	12.16 ± 0.98	12.5 ± 0.91*	12.06 ± 0.86
GLSR (s^{-1})					
15RM group	failure	−0.96 ± 0.14	−0.79 ± 0.15*	−0.86 ± 0.21*	−0.9 ± 0.23
	non-failure	−0.98 ± 0.11	−0.84 ± 0.13*	−0.92 ± 0.14	−0.97 ± 0.19
10RM group	failure	−0.96 ± 0.13	−0.85 ± 0.2*	−0.8 ± 0.2*	−0.92 ± 0.21
	non-failure	−0.97 ± 0.13	−0.82 ± 0.2*	−0.91 ± 0.19	−0.92 ± 0.23
GCSR (s^{-1})					
15RM group	failure	−2.06 ± 0.48	−2.45 ± 0.44*	−2.67 ± 0.67*	−2.31 ± 0.38
	non-failure	−2.04 ± 0.64	−2.49 ± 0.6*	−2.7 ± 0.6*	−2.25 ± 0.53
10RM group	failure	−2.05 ± 0.66	−2.52 ± 0.69*	−2.53 ± 0.45*	−2.28 ± 0.58
	non-failure	−2.03 ± 0.7	−2.48 ± 0.72*	−2.62 ± 0.59*	−2.25 ± 0.49

Data are shown as means ±SD.*Significant change compared to baseline, p<0.05. † Significant compared to failure 10RM group, p<0.05. ‡ Significant interaction with failure 10RM group, p<0.05. Eal = arterial elastance index; ELVI = ventricular end-systolic elastance index; GCSR = global circumferential strain; GLSR = global longitudinal strain rate; MWS= meridional wall stress; RM = repetition maximum; RWT = end-diastolic relative wall thickness.

Our data show that the increase in GCS after RE contributes substantially to SV. These results are in agreement with those obtained with mathematical geometry models, which showed that GCS makes a relatively larger contribution to SV and EF than GLS (MacIver, 2012; Stokke et al., 2017). In this context, the present experimental data suggest that after RE, circumferential shorting may have an important effect on transmural LV systolic function.

Midwall fractional shortening adjusted to wall stress has been suggested as a reliable method to measure LV contractility (Ito et al., 2020). We found that after RE, midwall and endocardial fractional shortening remained constant, and longitudinal dysfunction was not seen to have any physiological effect on LV contractility.

One interesting finding is that RE improved preload through increased PWT and EDV. This suggests that RE may alter the balance between PWT and EDV as one possible mechanism underlying improved preload. Another important finding was that significant changes in LV myofibril geometry were volume dependent: high-volume sets to failure greatly increased LV mass and IVST. This suggests that RE techniques alter the influence of overload pressure between PWT and IVST in the LV towards a more dominant contribution of the interventricular septum and it thus seems possible that acute perturbation of longitudinal strain occurs most frequently at this site. The present results support previous observation by Kim and Park, who established that high-volume workouts significantly increased LV mass, IVST and PWT (Kim & Park, 2020). Although the mechanisms underlying the acute increase in cardiac wall thickness after RE remain unknown at present, this effect may be due to an acute increase in myocardial interstitial fluid content. It has been reported that acute RE disproportionately increases sarcoplasm volume relative to myofibril protein accretion (Roberts et al., 2020). However, Damas et al. found that the acute increase in muscle CSA after RE more likely stems from oedema-induced muscle swelling (Damas et al., 2016).

Afterload is expressed as LV wall stress, and the combination of circumferential and meridional wall stress can be considered the afterload (Ito et al., 2020). Our data showed that RE had a greater influence on reduced cESS than on MWS. Some studies have reported a significant inverse relationship between wall stress and LV strain, suggesting that strain depends on afterload (MacIver, 2012; Stöhr et al., 2017; Stokke et al., 2017). The results of the present study demonstrate that after RE, a significant inverse linear correlation was found between cESS and GCS, and between LV mass and SVR. These relationships suggest that afterload changes secondarily to LV systolic function. It can therefore be assumed that after RE, the contribution of circumferential shortening and wall thickness have substantial effects on changes in afterload, and thus offer potential advantages in preserving EF and elevating SV.

The present results are consistent with previous studies of athletes, and confirm that RE with a reduced E/A ratio resulted in transient changes in transmitral filling pattern (Stöhr et al., 2015, 2017). The increased A-wave and decreased E-wave represent a transient shift in the pattern of ventricular filling

Table 3. Left ventricle functional performance during baseline and 2 4 h of follow up.

		Baseline	30 min post	6 h post	24 h post
EDV (mL)					
15RM group	failure	133.25 ± 24.61	138.08 ± 24.74	150 ± 23.97*	140.83 ± 21.69
	non-failure	132.75 ± 23.72	138 ± 23.08	145.50 ± 19.56	142.33 ± 18.21
10RM group	failure	133.91 ± 24.50	149.33 ± 19.18*	141.16 ± 33.18	147.25 ± 33.61
	non-failure	135 ± 18.77	146.83 ± 16.48*	145.10 ± 16.13	144.16 ± 17.17
LA area (mm)					
15RM group	failure	19.02 ± 1.09	17.83 ± 1.44*‡	17.46 ± 1.04*	17.66 ± 1.3*†
	non-failure	19.07 ± 1.05	17.97 ± 1.21*	17.21 ± 1.03*	17.32±.79*$
10RM group	failure	18.9 ± 2.07	19.55 ± 2.52	18.83 ± 2.89	19.42 ± 2.42
	non-failure	19.02 ± 1.43	18.06 ± 1.43	17.31±.95*	18.23 ± 1.23
SV (mL)					
15RM group	failure	79.60 ± 14.24	87.05 ± 1.84	97.90 ± 13.07*	88.42 ± 12.12*
	non-failure	80.22 ± 8.94	86.41 ± 1.11	93.41 ± 9.96*	9.24 ± 1.26*
10RM group	failure	8.10 ± 14.17	91.13 ± 15.87*	86.03 ± 19.29	92.57 ± 21.34*
	non-failure	80.64 ± 11.36	89.90 ± 13.32*	88.04 ± 14.36	89.16 ± 9.15
HR (bpm)					
15RM group	failure	61 ± 6	77 ± 6*	67 ± 5*	62 ± 5
	non-failure	62 ± 5	75 ± 4*	69 ± 7*	63 ± 5
10RM group	failure	62 ± 9	75 ± 6*	71 ± 7*	66 ± 10
	non-failure	63 ± 8	72 ± 9*	71 ± 9*	66 ± 9
Q (L min^{-1})					
15RM group	failure	4.84±.77	6.67±.85*	6.53±.87*	5.41 ± 0.61
	non-failure	4.92 ± 0.53	6.43±.68*	6.42±.55*	5.63±.41
10RM group	failure	4.94±.79	6.87 ± 1.44*	6.1 ± 1.49*	6.07 ± 1.66*
	non-failure	5.04 ± 0.86	6.43±.97*	6.21 ± 1.01*	5.83±.92
EF (%)					
15RM group	failure	57.91 ± 14.81	61.83 ± 1.24*	64.52 ± 8.17*	61.71 ± 10.18
	non-failure	59.80 ± 6.86	62.10 ± 6.37	63.88 ± 5.55*	63.11 ± 5.47
10RM group	failure	59.98 ± 3.81	61.12 ± 5.02	6.89 ± 4.09	62.90 ± 2.46
	non-failure	59.66 ± 5.30	61.23 ± 4.51	6.70 ± 4.34	61.76 ± 4.74
A (cm s^{-1})					
15RM group	failure	42.58 ± 9.4	48.17 ± 9.27*	44.75 ± 1.61	43.83 ± 9.51
	non-failure	42.75 ± 9.32	46.25 ± 9.46*	44.17 ± 1.44	43.17 ± 9.85
10RM group	failure	42.08 ± 9.5	47.25 ± 9.55*	43.08 ± 9.51	43.17 ± 9.82
	non-failure	42.75 ± 9.86	46.17 ± 1.52*	43.67 ± 9.51	42.83 ± 1.59
E (cm s^{-1})					
15RM group	failure	86 ± 15.62	81.07 ± 15.1*	81.53 ± 12.43	89.13 ± 16.28
	non-failure	85.63 ± 10.87	82.17 ± 1.57	79.5 ± 9.39*	86.25 ± 14.54
10RM group	failure	85.58 ± 15.51	79.62 ± 15.91*	81.73 ± 15.16	87.8 ± 17.24
	non-failure	85.3 ± 12.65	82.92 ± 13.7	78.25 ± 10*	85.42 ± 1.6
E/A ratio (cm s^{-1})					
15RM group	failure	2.06±.29	1.69±.2*	1.87±.3*	2.06 ± 0.28
	non-failure	2.05 ± 0.27	1.81±.21*	1.85±.26*	2.04±.27
10RM group	failure	2.16±.73	1.79±.75*	2.01±.69	2.16 ± 0.79
	non-failure	2.11 ± 0.6	1.88±.48*	1.88±.47*	2.12±.61

Data are shown as means ±SD.*Significant change compared to baseline, p<0.05. † Significant compared to failure 10RM group, p<0.05. ‡ Significant interaction with failure 10RM group, p<0.05. $ Significant compared to the non-failure 10RM group, p<0.05. A = peak late diastolic left ventricular filling velocity; E = peak early diastolic left ventricular filling velocity; EDV = end-diastolic volume; EF = ejection fraction; E/A ratio = ratio of peak early filling diastolic velocity to peak late diastolic velocity; HR = heart rate; LA = left atrial; RM = repetition maximum; SV = stroke volume; Q = cardiac output.

Table 4. Linear relationships between left ventricle function parameters before and after resistance exercising.

	Baseline		30 min post		6 h post		24 h post	
	r	p	r	p	r	p	r	p
GCS-EF	0.31	0.03*	0.35	0.01*	0.41	0.003**	0.30	0.03*
GCS-SV	0.20	0.17	0.21	0.16	0.40	0.005**	0.23	0.11
GCS-cESS	−0.20	0.15	−0.32	0.02*	−0.05	0.74	−0.10	0.50
LA area-GLS	0.12	0.40	0.35	0.01*	0.02	0.90	0.13	0.37
LA area-GCS	−0.19	0.18	−0.29	0.04*	−0.04	0.79	−0.10	0.49
LV mass-SVR	0.05	0.76	0.04	0.81	−0.29	0.04*	−0.29	0.4*

*Significance level p<0.05. **p<0.01. cESS = end-systolic circumferential wall stress; EF = ejection fraction; GCS = global circumferential strain; GLS = global longitudinal strain; LA = left atrial; LV mass = left ventricular mass; SV = stroke volume; SVR = systemic vascular resistance.

period towards late diastole, and result in a proportional decrease in early diastolic relaxation in the myocardium.

Hypotension is associated with a decrease in both resting Eal and ELVI, and hence a decrease in their ratio. Eal reflects the arterial load on the ELVI, and is determined by both resistive (i.e., SVR) and pulsatile (i.e., TACI) components (Currie et al., 2019). We found that RE diminished the resistive component, while the pulsatile contribution was unaffected. This suggests

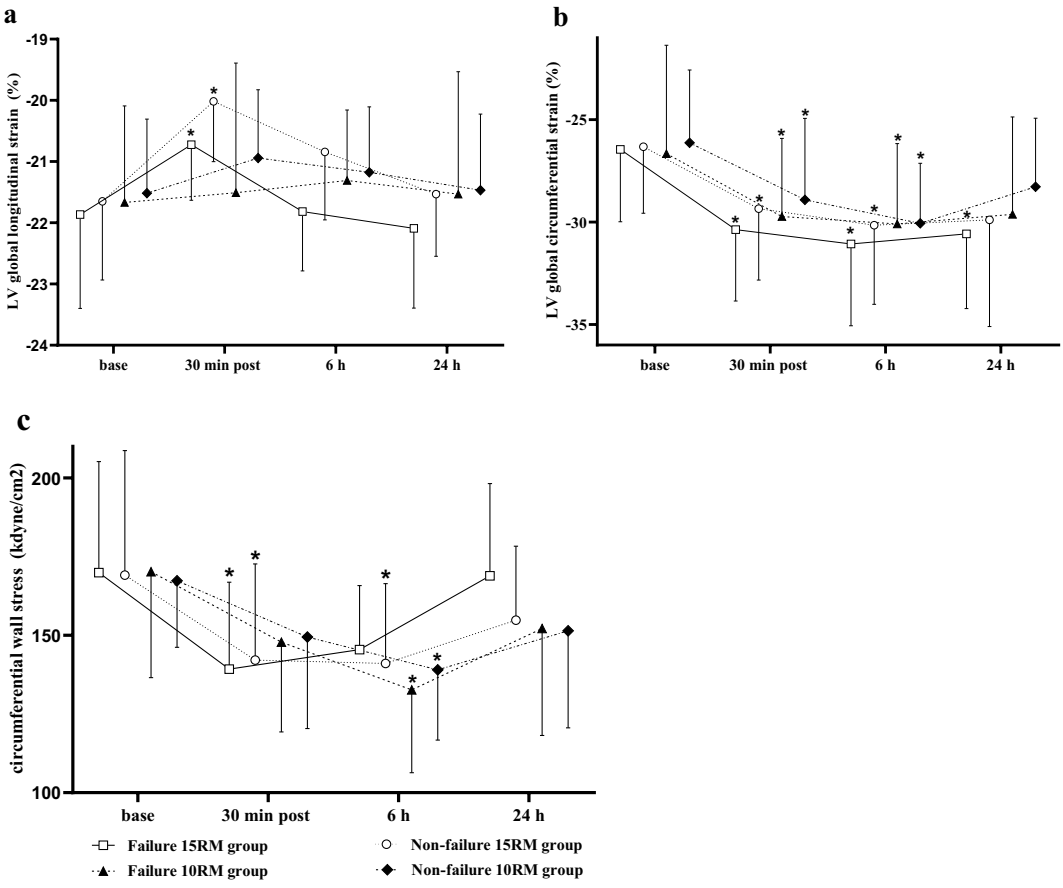

Figure 4. Post-RE changes in LV strain and wall stress from baseline at 30 mint, 6 hours and 24 hours follow-up. **A**, GLS. **B**, GCS. **C**, cESS, data are presented as means and SD. *denotes significant change compared to baseline ($p < 0.05$).

that after RE, there is a potential enhancement of peripheral vasodilatory function, and withdrawing sympathetic nervous system activity allows the LV to maintain stroke efficiency and cardiac reserve.

RE favours the return of BP to pre-exercise values or even lower, a phenomenon called PEH (de Freitas Brito et al., 2019). The present findings confirm that RE with different intensities and techniques triggers the same PEH response as reported in previous studies (Simão et al., 2005; Stöhr et al., 2017). It should be noted, however, that some studies found PEH to be volume dependent (de Freitas Brito et al., 2019; Rezk et al., 2006). We used multi-joint free weights in the RM, which can exert high loads during exercise. Ultimately, our results suggest that high-load RE can attenuate the effects of intensity and volume discrepancy on PEH.

One inherent limitation of the present study is the small sample size. Nevertheless, the cross-over, counterbalanced approach to group allocation along with the use of a repeated measures design potentially provides adequate statistical power to explore in depth some quantitative outcomes related to RE volume and different RE techniques. In addition, the present study balanced mechanical work and rest intervals across both RE techniques. Consequently, our findings provide insights that can help to clarify the origin of transient cardiac perturbations in response to different types of RE in healthy, fit participants and athletes. In this connection we note that participation in this study was limited to athletes undergoing weight training, because the RE protocols required familiarity with free weight exercising. The use of free weights emulated real-world movements and ensured appropriate loads within the RM zone. All athletes enrolled in the study had appropriate echocardiographic windows, although possible changes in these windows might have affected the reported finding and were a factor that was not controlled for.

Conclusion

The present study found that the reduction in LA compliance and transient cardiac perturbations were RE volume dependent. Decreases in LA compliance did not return to normal baseline levels during 24 h of follow-up. The post-RE changes in LV myofibril geometry are volume dependent, and are more strongly influenced by sets to failure technique. RE contributed to increases in GCS and decreases in afterload, while SV and EF were preserved. High-load RE, regardless of differences in volume, intensity, or technique, decreased the afterload and thus led to PEH, and this effect was influenced most by reduced circumferential wall stress and SVR.

Acknowledgments

We appreciate the time and effort contributed by all volunteer participants, and thank K. Shashok (AuthorAID in the Eastern Mediterranean) for improving the use of English in the manuscript.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

The author(s) reported there is no funding associated with the work featured in this article.

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