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The impact of 24 weeks of supervised endurance versus resistance exercise training on left ventricular mechanics in healthy untrained humans

David L. Oxborough PhD[1], Angela Spence PhD[2,3] Keith P. George PhD[1], Frederieke Van Oorschot BSc[1,4], Dick H. T. Thijssen PhD[1,4] and Daniel J. Green PhD[2]

[1] Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Tom Reilly Building, Byrom Street, Liverpool, UK
[2] School of Human Sciences (Exercise and Sports Science), The University of Western Australia, Nedlands, Australia
[3] School of Physiotherapy and Exercise Science, Curtin University, Bentley, Australia.
[4] Radboud Institute for Health Sciences, Radboud University Nijmegen Medical Center, The Netherlands

Address for Correspondence:

Dr David Oxborough,
Reader in Cardiovascular Physiology
Research Institute for Sport and Exercise Sciences
Tom Reilly Building
Liverpool John Moores University
Liverpool
L3 3AF
Email: d.l.oxborough@ljmu.ac.uk
Tel: 0151 904 6231

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ABSTRACT

Background: In addition to the well-known cardiac structural adaptation to exercise training, little work examined changes in LV mechanics. With new regional and global indices available we sought to determine the effect of 24 weeks endurance *versus* resistance training on LV mechanics.

Methods and Results: 23 male subjects were randomly allocated to either a 24-week endurance- or resistance-training program. Pre- and post-training 2D echocardiographic images were acquired. Global LV mechanics (strain [ε]) were recorded in longitudinal, circumferential and radial planes. Rotation was assessed at apical and basal levels. In addition, longitudinal ε-volume loops, across the cardiac cycle, were constructed from simultaneous LV ε (longitudinal and transverse strain) and volume measurements across the cardiac cycle as a novel measure of LV mechanics. Marginal differences in ε and rotation data were found between groups. Post-training, we found no change in global peak ε data. Peak basal rotation significantly increased after training with changes in the endurance group (-2.2±1.9^o to -4.5±3.3^o) and the resistance group (-2.9±3.0^o to -3.4±2.9^o). LV ε-volume loops revealed a modest rightward shift in both groups.

Conclusions: Whilst most global and regional indices of LV mechanics were not significantly altered, 24 weeks of intense supervised exercise training increased basal rotation. Further studies that assess LV mechanics in larger cohorts of subjects and those with cardiovascular disease and risk factors may reveal important training impacts.

Key words: cardiac, imaging, echocardiography, speckle-tracking,

NEW AND NOTEWORTHY

This study builds on previous work by our group and presents a comprehensive assessment of cardiac mechanics following dichotomous exercise training programmes. We highlight novel findings in addition to the inclusion of strain-volume loops, which shed light on subtle differences in longitudinal and transverse contribution to volume change throughout the cardiac cycle. Our findings suggest that training has an impact on basal rotation and possibly strain-volume loops.

INTRODUCTION

Chronic exercise results in structural remodelling of the left ventricle (LV) of the heart, which is mediated by many facets of the exercise stimulus as well as individual characteristics [3]. Historically, a differential pattern of LV structural remodelling has been linked to endurance versus resistance training [11]. An eccentric LV hypertrophy (increased cavity size and concomitant increase in wall thicknesses) has been reported in athletes that undergo endurance training, with a concentric hypertrophy (increased wall thickness with no change in cavity size) observed in resistance trained athletes [8,11]. This dichotomous phenotype based on training exposure has been challenged in a meta-analysis [21] and empirical research [5,18,20], with a greater degree of structural remodelling occurring with endurance training [18]. Whether different exercise training paradigms have differential effects on LV function has received less attention, with most information focused on standard, global parameters such as stroke volume and ejection fraction [11].

Data from meta-analyses [21,15] provide compelling evidence that resting global LV systolic and diastolic function are not different between athletes and healthy controls. Despite this, some individual studies have highlighted lower ejection fraction [10] and/or supernormal diastolic functional indices [7] in various athletic groups. This lack of consistency is likely driven by the inherent limitations of conventional global indices of function and the heterogeneity of athlete demographics using a cross-sectional approach [3].

Any adaptation of LV structure and geometry will change myocardial fibre alignment that could then influence LV mechanics [1]. LV mechanics reflect the "real" 3D activation and movement of the myocardium and recent advances in echocardiographic techniques such as LV strain (ε) imaging facilitates the assessment of global and regional LV mechanics from longitudinal, circumferential and radial planes. This approach also determines the nature and magnitude of LV rotation and overall twist. Some "athlete-control" cross-sectional studies have employed these techniques [4] to determine potential training-related changes in LV mechanics. However, the design limitations and self-selection bias associated with cross-sectional studies cannot attribute causality to training status and differences in LV mechanics. To determine cause-and-effect, longitudinal intervention studies are required but this evidence base is currently limited in terms of the impacts on mechanics.

We have previously described, in this cohort, no change in global longitudinal ε after 24 weeks of endurance or resistance training [18]. This provided a very limited insight into training related changes in LV mechanics. The impact of different training interventions upon a more comprehensive assessment of LV mechanics is currently lacking. It is possible that the addition of transverse Ɛ assessment in the longitudinal plane as well as the evaluation of Ɛ across the cardiac cycle (temporal assessment rather just peak data) may reveal changes that were not apparent from assessment of global indices. We have recently developed a novel method of simultaneously assessing longitudinal ε, transverse ε and volume (ε-volume loops) across the cardiac cycle [14]. It is possible, using this approach, to independently assess the ε-volume relationships in systole and diastole and hence subjectively evaluate the relative contribution of systolic and diastolic ε to volume change. The utility and insight provided by this technique has already been demonstrated in acute exercise [9] and clinical [6] settings, where it is apparent that ε-volume loops are sensitive to changes in acute and/or chronic loading on the LV. With likely training-related changes in LV loading, ε-volume loops could provide novel insights into the adaptability of LV mechanics to physiological adaptation of the LV. In view of this, we sought to comprehensively assess the impact of 24 weeks of closely supervised and centre-based endurance and resistance training on LV mechanics in healthy untrained male participants. Specifically, we introduce the novel assessment method of ε-volume loops to evaluate the

impact of exercise training on the link between LV structure and function in a longitudinal exercise training setting. Our null hypothesis was that endurance and resistance exercise would induce similar effects on LV mechanics.

METHODS

Twenty-three young healthy male subjects (mean ± SD age: 27.4 ± 5.5 years) volunteered to take part in the study. Prior to recruitment, participants underwent pre-participation screening which involved a detailed medical history, physical examination, standard blood panels and a physical activity questionnaire. All participants were free of known cardiovascular, liver, renal, respiratory and metabolic disease, were not taking any prescribed medication, were non-smokers and were considered untrained (defined as undertaking no structured exercise). The study was approved by the Human Research and Ethics committee of the University of Western Australia and conformed to the Declaration of Helsinki. Data related to cardiac structural remodelling in this study have been published previously [17,18].

Study design

Subjects were randomly allocated to either the resistance-training group (RES, n=13) or the endurance-training group (END, n=10). Participants completed a 24-week RES or END training programme. During this training period, subjects attended three 1-hour exercise training sessions per week. To improve compliance with the programme, an experienced exercise physiologist supervised all exercise sessions. To minimise the risk of injury, regular stretching and core strengthening were included in the training. Participants were assessed for anthropometrics, aerobic fitness and strength conditioning at baseline and post 24-week training. In addition, a detailed echocardiogram was undertaken at both data collection points.

Exercise training interventions

Endurance training

The endurance training programme consisted of three training-phases and was divided into eight 3-week mesocycles. In mesocycle 1-4 each hard-load week was followed by an easy week and in the remaining four mesocycles, two weeks were hard loaded, with the third week acting as a recovery week. To ensure participants training at correct intensity, VO_2peak values were determined upon which the training paces were based. Heart rate monitors (Polar F1, Finland) were worn to monitor intensity. Specific training details are recorded in Spence et al. (2011) [18].

Resistance training

The resistance training programme focused on Olympic-style weightlifting and associated exercises. The programme consisted of three training-phases divided into six 4-week mesocycles. Each mesocycle commenced with three weeks progressive loading, peaking in the third week, followed by one week of recovery. Specific training details are recorded in Spence et al. (2011) [18].

Basic measurements

All participants underwent whole body DXA assessment (Lunar Prodigy, GE Medical Systems, Madison, WI, USA) before and after training to determine total fat mass, total lean mass and body fat percentage. Body mass and stature were assessed via standard anthropometric techniques. Aerobic fitness was assessed by a treadmill based graded exercise test [18] and muscular strength was assessed by 1RM for bench press and squat exercises [18]. Brachial artery blood pressure was assessed by automated sphygmomanometry (Dinamap, Critikon, USA).

Echocardiographic measurements

All echocardiographic images were acquired using a 1.5- to 4-MHz phased array transducer on a commercially available ultrasound system (Vivid I, GE Medical, Horton, Norway). A single, highly experienced sonographer collected all images and was blinded to exercise group allocation. Subjects lay in the left lateral decubitus position and images of the LV were obtained from an apical 4-chamber view and parasternal short-axis view. Two-dimensional

(2-D) image optimisation was performed, including gain, dynamic range and depth to ensure optimal endocardial delineation. In order to define end-systole and end-diastole manually, a trans-aortic continuous-wave Doppler signal was collected.

Conventional 2D and Doppler

LV chamber quantification was undertaken using 2D echocardiography. Structure was determined by assessment of LV wall thickness of the septum (IVS) and the posterior wall (PWT) as well as the internal cavity at end diastole (LVIDd) and end systole (LVIDs). Subsequent calculation of LV mass was undertaken using the linear dimensions and derived from the ASE corrected formula. Trans-mitral Doppler was undertaken using a 4mm pulsed wave sample volume positioned at the tips of the mitral valve in diastole which allowed for the measurement of peak early diastolic velocity (E), peak late diastolic velocity (A) and the calculated E/A ratio. LV volumes at end diastole (LVEDV) and end systole (LVESV) were taken from a Simpsons monoplane method whilst deriving the ε-volume loops as described below.

LV mechanics

Cine loops of LV motion were recorded to DVD in a raw DICOM format and a single experienced, blinded observer analysed the data offline. After calculation of aortic valve closure the observer selected heart cycles of the highest quality and calculated ε across the cardiac cycle using speckle tracking software (Echopac, GE Healthcare, Norway). This process determined ε segmentally tracking natural acoustic markers. The myocardium was manually traced and adjusted so that the region of interest incorporating all of the wall thickness, whilst avoiding the pericardium.

For global longitudinal ε an apical 4-chamber view was used to determine peak ε as well as peak strain rates (SR) during systole and early diastole. Parasternal short-axis views were utilised to calculate peak circumferential ε and SRs, peak radial ε and SRs, as well as peak apical and basal rotation. All ε values were also exported to a spreadsheet (Microsoft Excel 2010, USA).

LV ε-volume loops

Transverse and longitudinal strain values obtained by speckle-tracking were exported to a spreadsheet (Microsoft Excel 2010, USA). These raw data underwent cubic spline interpolation to provide 600 data points across the cardiac cycle. The splined data was then divided into 5% increments of the cardiac cycle and the absolute time from aortic valve closure was recorded at each increment. Simpson's methodology was utilised to calculate LV volume belonging to each ε time point across the cardiac cycle. A graph of the relation between LVε (i.e. transverse and longitudinal ε) and LV volume was then made for each subject and polynomial regression was undertaken to determine the relationship between ε and volume such that ε could be interpolated for 10% increments of LVEDV as previously described [14]. In brief, ε was calculated at each % increment of LVEDV to allow direct comparison between conditions and groups. The difference between ε at any given % of EDV in systole and diastole was calculated to provide a 'gradient'. This assessment provides an indication of the systolic-diastolic (un)coupling i.e. the absolute difference in the magnitude of ε for the same volume in systole and diastole. Mean LV ε- volume loops and the derived indices were calculated and presented as an average of all subjects in each group (RES and END) at each time point (baseline and post 24-week training). In this way, the temporal relationship of ε across the cardiac cycle and its association to volume were presented such that the difference of ε at any given volume in systole and diastole could be inspected. Figure 1 provides a diagram of the longitudinal and transverse loops highlighting (un)coupling as defined above. In addition to an exploratory statistical analysis the loops were visually assessed with regards to the slope of the curve in systole and diastole alongside peak ε and end systolic/diastolic volumes.

INSERT FIGURE 1

Statistical analysis

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) (version 23.0, Chicago IL, USA), and the critical α was set at 0.05. All data are presented as mean ± SD. A p-value <0.05 was considered to be statistically significant. In order to compare the effect of 24 weeks of progressive training intervention (endurance vs.

resistance) on LV mechanics, two-way ANOVAs (training intervention by time) were used with pairwise post-hoc Bonferroni tests where significant interaction terms were observed. The exploratory analysis of the ε-volume loops including ε at %EDV and the calculation of (un)coupling were assessed using the same process i.e. two-way ANOVAs (training intervention by time). By way of a check for any small alterations in loading and heart rate that occurred with training, we undertook additional two-way ANCOVA's with delta HR and SBP as separate co-variates. Our group have previously presented good inter and intra-observer variability of the ε-volume loop in healthy trained participants [14].

RESULTS

As previously presented [18] exercise training was effective in modifying various functional, anthropometric and cardiac structural indices. For example, there was a significant main effect for time for systolic blood pressure, mean arterial pressure, resting HR, maximal HR, bench press, squat, total strength, total body fat and total lean mass (Table 1). In all cases, apart from strength data and lean body mass, these data decreased post-training. A significant time by intervention interaction effect was noted for resting heart rate (larger drop in endurance group) as well as bench press and total strength (larger increase in resistance group).

INSERT TABLE 1

The impact of training on LV structure, is presented in Table 2. There was a significant main effect for time with respect to LV mass and PWT with greater values post-training in endurance and resistance groups.

INSERT TABLE 2

Left Ventricular Mechanics

Basal rotation demonstrated a significant main effect of time with the increase in basal rotation after endurance training higher than after resistance training (Table 3). There was a significant main effect of time for the time to peak longitudinal ε with both groups having a longer time to peak longitudinal ε post-training. These significant main effects remained after covariate analysis for small changes in HR and SBP. All other measures of global LV mechanics were not different between groups or across the training intervention.

INSERT TABLE 3

Longitudinal and transverse ε-volume loops are presented in Figure 2 and Table 4 for both training groups pre- and post-intervention. There was a close coupling of longitudinal and transverse systolic and diastolic temporal ε-volume characteristics at baseline. Inspection of Figure 2A-D reveals a modest rightward shift of both transverse and longitudinal ε-volume loops. There was no apparent change in longitudinal ε-volume coupling regardless of intervention type. There was a modest increase in transverse systolic-diastolic uncoupling in both groups, which is more visually apparent as a result of endurance training. This is supported by an exploratory statistical assessment of (un)coupling / gradient values for the main effect of training on transverse ε-volume loop, particularly at 60%EDV (Table 4).

INSERT FIGURE 2 AND TABLE 4

DISCUSSION

This is the first randomized, centre-based and closely supervised exercise training study to assess the impact of different modalities of exercise training on LV mechanics in humans. The key findings were that, 1) exercise training augmented basal rotation, 2) neither form of

290 training had discernible effects on other global measures of peak LV mechanics, and 3)
291 exploratory transverse ε-volume loops were uncoupled as a result of training.

292

293 The impact of training on LV structure has been presented and evaluated previously [18] and
294 generally supported the concept that greater adaptation occurred after the imposition of an
295 endurance training stimulus. This supports recent cross-sectional data [20,21] and suggests
296 that concentric cardiac structural adaptation to the haemodynamic loading associated with
297 exercise occurs substantially less frequently than previously proposed [5]. Whether both
298 training modes, and specifically the endurance training programme which had a
299 measureable effect on LV mass and LVEDV [18], would alter global and regional peak LV
300 mechanics and the novel ε-volume loops has not been reported previously.

301

302 To our knowledge, our work is the first to demonstrate that basal rotation increased with
303 training. Whilst there was a modest difference between modalities, from an absolute
304 perspective, this may have some potential physiological relevance since rotation
305 importantly affects both systolic and diastolic function. Weiner *et al.* reported increased
306 apical rotation and derived LV twist, but preserved basal rotation, in elite rowers who were
307 subsequently tracked over training cycles [22]. Our findings differ from these results, in that
308 basal rather than apical rotation was the parameter altered by training. Our study extends
309 the Weiner findings, in that we randomized previously untrained subjects into dichotomous
310 interventions, whereas the rowing intervention in elite athletes described by Weiner *et al*. is
311 considered a mixed modality training stimulus. The exact mechanism for training-induced
312 changes in basal and/or apical rotation and consequent twist are currently unknown and
313 require further study. Whilst a change in apical rotation is easier to conceptualise as a
314 consequence of training, it is a strength of the present study that relative impacts on both
315 apical and basal mechanics can be derived. A change in basal rotation will impact on the
316 "wringing out" effect of cardiac contraction, even in the absence of obvious apical
317 adaptation. It is possible to speculate that our novel observation relating to basal rotation
318 may impact more, in terms of ventricular interdependence, that a change in apical

behaviour. The impact of training in subjects with *a priori* impairment in cardiac function will provide an interesting comparator in future studies.

This is the first study, to our knowledge, to systematically assess ε-volume loops after a randomised trial of exercise training in humans. We have previously demonstrated changes in ε-volume loops in an acute exercise setting [9] as well as in a long-term clinical scenario [6] both a likely consequence of changes in cardiac work and loading. In the current study, inspection of the ε-volume loops highlighted some potential differences in the impact of training modalities. Specifically, inspection of Figure 2a and 2c is suggestive that endurance training was associated with a somewhat larger change in systolic-diastolic uncoupling in the transverse plane relative the resistance group (Figure 2b and 2d). It is important to emphasise, however, that this finding is largely based on visual inspection of the loops. It is possible that a longer or more intense set of training interventions, or a study of a larger sample size, may in future reveal distinct impact of training modality on LV mechanics.

In general, neither training intervention resulted in systematic changes in global peak indices of LV mechanics. Data for global peak radial and circumferential ε adds to that already reported for peak longitudinal ε [18]. Previous athlete-control group comparisons of peak ε generally support the lack of training related differences [13], suggesting that training has a limited impact on global mechanics. It is interesting to note that when Nottin *et al.* investigated peak ε in different layers of LV tissue there was some evidence that this data was lower in trained cyclist compared to sedentary controls, which the authors suggested supported a greater "exercise-reserve" of ε to underpin higher levels of LV functional performance during exercise. This difference was not noted in the endurance trained group but different level (sub-endocardial vs. sub-epicardial) analysis was not undertaken. A previous training study [2] reported an increase in peak longitudinal ε after 90 days of rowing exercise in a group of experienced athletes. The intensity, duration, volume and mode of exercise would likely be different between experienced rowers and previously sedentary individuals, limiting the relevance of a direct comparison between

studies. More randomized, controlled within-subjects, longitudinal studies are required in different population groups employing a range of exercise stimuli.

We should note some important study-specific limitations. The interpretations of the outcomes of this study are limited to young healthy men undertaking a specific time and mode-limited exercise regimen. Whether the same outcomes would be apparent in different groups, including clinical or rehabilitation populations, requires further study. There is some evidence that training adaptations differ in women, and future studies should address these questions specifically [23]. We reported global peak LV mechanical data for ε only although temporal data are captured in the ε-volume loops. Further studies evaluating peak and temporal ε data in the RV and both atria are required. The impact of training on ε-rate in both systole and diastole might also prove insightful and has received scant attention to date. It is well established that cardiac structural and functional changes occur at different rates and by limiting our outcome measures to 24 weeks it is important to recognise that changes in structure and function may occur with different time courses, as seen in vascular adaptations to training [19]. As stated above, changes in volume may impact fibre alignment and, thus, mechanics. It is also possible that changes in HR or afterload impact on our findings, although training induced changes in these variables were modest in the present study and within a normal physiological range. A further important limitation is related to the variability that can be apparent in measures of echocardiographically-derived LV mechanics in humans (as indicated in Table 4 and Figure 2). Despite a well-controlled, randomized design, powered appropriately to detect changes in cardiac structure with training, we must nevertheless acknowledge that the present study recruited small numbers to both training arms and a larger trial, particularly involving clinical populations in whom changes may be more apparent, should be undertaken in future.

CONCLUSIONS

Although global indices of LV mechanics were not significantly altered, 24 weeks of intense supervised exercise training was related to an increase in basal rotation in the present

study. This was accompanied by a modest rightward shift in LV ε-volume loops and some degree of systolic–diastolic uncoupling in transverse ε-volume loops after training.

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DISCLOSURES

None

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Figure and Table Legends

Table 1 - Subject characteristics at baseline and after 24 weeks of training

Table 2 - Echocardiographic conventional measurements at baseline and after 24 weeks Training

Table 3 - Peak global LV ε and rotation data at baseline and after 24 weeks training

Table 4 – LV longitudinal and transverse strain-volume relationship at increments of % EDV

Figure 1 – Diagrammatic representation of the LV ε-volume loop highlighting the concept of Coupling

Figure 2 – Longitudinal and transverse ε-volume loops at baseline and following 24 weeks endurance training *(2A – Endurance Longitudinal Strain, 2B – Resistance Longitudinal Strain, 2C Endurance Transverse Strain, 2D – Resistance Transverse Strain).*

Table 1 - Subject characteristics at baseline and after 24 weeks of training

	Endurance group (n=10)		**Resistance group (n=13)**	
Variable	**Baseline**	**Post training**	**Baseline**	**Post training**
Age	28.4 ± 1.9	-	26.6 ± 1.3	-
Body size and composition				
Height(m)	1.79 ± 0.02	-	1.81 ± 0.02	-
Weight(kg)	78.0 ± 5.4	78.3 ± 5.5	81.7 ± 4.2	83.3 ± 4.4
BSA(m^2)	1.96 ± 0.07	1.97 ± 0.08	2.02 ± 0.06	2.04 ± 0.06
BMI (kg/m^2)	24.2 ± 1.3	24.3 ± 1.4	24.7 ± 1.0	25.2 ± 1.0
Total body fat (%)†	22.7 ± 2.4	21.1 ± 2.5	23.1 ± 2.0	21.4 ±2.1
Total fat mass (kg)	17.7 ± 2.9	16.7 ± 3.1	18.6 ± 2.3	17.8 ± 2.5
Total lean mass (kg)†	56.9 ± 2.9	58.3 ± 3.0*	59.7 ± 2.3	62.0 ± 2.2
Cardiorespiratory measures				
SBP(mmHg)†	122 ± 2	119 ± 2	125 ± 1	119 ± 2
DBP(mmHg)	69 ± 3	68 ± 2	71 ± 2	70 ± 2
MAP(mmHg)†	87 ± 3	85 ± 2	89 ± 1	86 ± 2
Resting HR (bpm)‡	65 ± 3	58 ± 2	66 ± 3	65 ± 2
Maximum HR (bpm)†	197 ± 3	193 ± 2	200 ± 2	196 ± 2
Aerobic Fitness				
VO_{2peak} ($L\ min^{-1}$)	3.5 ± 0.2	3.8 ± 1.8	3.6 ± 0.3	3.6 ± 0.2
VO_{2peak} ($mL\ kg^{-1}\ min^{-1}$)	45.8 ± 1.6	49.3 ± 2.2	44.0 ± 2.5	44.0 ± 2.2
Strength measures				
Bench press (kg)†‡	58 ± 5	61 ± 5	58 ± 5	69 ± 5*
Squat(kg)†	89 ± 7	122 ± 5	97 ± 6	139 ± 4
Total strength (kg)†‡	147 ± 10	183 ± 9*	155 ± 10	208 ± 9*

†P < 0.05 time effect by ANOVA (baseline, post training); ‡P < 0.05 time x intervention (endurance, resistance) interaction effect by ANOVA; ∗significantly different from pre-training at P < 0.05;
BSA, body surface area; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HR, heart rate.

495

496 **Table 2 –** Echocardiographic conventional measurements at baseline and after 24 weeks
497 training

	Endurance Group (n = 10)		**Resistance Group (n = 13)**	
	Baseline	**Post 24 week Training**	**Baseline**	**Post 24 week Training**
LV mass, g †	157.78 ± 46.25	179.57 ± 42.57	178.14 ± 36.02	186.0 ± 33.90
LVEDV, ml	106 ± 25	112 ± 25	114 ± 24	118 ± 22
LVIDd, cm	4.73 ± 0.46	4.65 ± 0.32	4.83 ± 0.31	4.71 ± 0.44
LVIDs, cm	3.25 ± 0.43	3.01 ± 0.22	3.13 ±0.32	2.98 ±0.36
IVS, cm	0.95 ± 0.16	1.09 ± 0.14	1.11 ± 0.12	1.09 ± 0.12
PW, cm †	1.06 ± 0.16	1.18 ± 0.22	1.09 ± 0.20	1.17 ± 0.18
E, $m.s^{-1}$	0.76 ± 0.18	0.76 ± 0.19	0.71 ± 0.18	0.71 ± 0.14
A, $m.s^{-1}$	0.38 ± 0.10	0.35 ± 0.10	0.35 ± 0.05	0.35 ± 0.06
E/A Ratio	2.11 ± 0.70	2.27 ± 0.60	2.09 ± 0.65	2.10 ± 0.74
†P < 0.05 time effect by ANOVA; LV mass, left ventricular mass; LVEDV, left ventricular end diastolic volume; LVIDd, Left ventricular internal diameter in diastole; LVIDs, Left ventricular internal diameter in systole; IVSd, interventricular septal thickness; PWT, posterior wall thickness, E, early diastolic filling velocity; A, late diastolic filling velocity				

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501 **Table 3 -** Peak global LV ε and rotation data at baseline and after 24 weeks training

PARAMETER	ENDURANCE BASELINE (Mean ± SD)	ENDURANCE TRAINED (Mean ± SD)	RESISTANCE BASELINE (Mean ± SD)	RESISTANCE TRAINED (Mean ± SD)
Longitudinal ε (%)	-18 ± 2	-18 ± 2	-16 ± 2	-17 ± 2
TTP longitudinal ε (ms)†	347 ± 17	380 ± 54	366 ± 34	378 ± 34
Circumferential ε (%)	-20 ± 4	-19 ± 3	-21 ± 4	-19 ± 5
TTP circumferential ε (ms)	364 ± 31	382 ± 57	390 ± 33	402 ± 46
Radial ε (%)	48 ± 17	47 ± 17	53 ± 19	41 ± 18
TTP radial ε (ms)†	365 ± 40	423 ± 89	436 ± 28	443 ± 82
Apical Rot (°)	10.4 ± 4.6	9.4 ± 5.2	9.0 ± 4.1	10.4 ± 3.5
Basal Rot (°) ‡	-2.2 ± 1.9	-4.5 ± 3.3	-2.6 ± 2.9	-3.7 ± 2.8
Twist (°)	12.0 ± 5.1	13.4 ± 8.3	10.7 ± 5.4	13.4 ±3.6
†P < 0.05 time effect by ANOVA; ‡P = 0.05 time effect by ANOVA; TTP, time to peak. Rot, rotation.				

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505 **Table 4 –** LV longitudinal and transverse strain-volume relationship at increments of % EDV

Parameter	**END(n=10)** **Baseline** **Mean ± SD**	**END(n=10)** **Post** **Mean ± SD**	**P** **T-test**	**RES(n=13)** **Baseline** **Mean ± SD**	**RES(n=13)** **Post** **Mean ± SD**	**P** **T-test**	**P-values ANOVA**		
LONGITUDINAL Systolic strain							**T**	**I**	**T*I**
90% EDV(%)	-2.4±1.09	-2.5±0.3	0.975	-3.0±0.2	-3.0±0.3	0.978	0.993	0.134	0.965
80% EDV(%)	-5.7±0.5	-5.5±0.5	0.882	-6.1±0.5	-6.1±0.5	0.938	0.939	0.315	0.860
70% EDV(%)	-8.9±0.7	-8.6±0.7	0.839	-9.2±0.6	-9.3±0.6	0.897	0.925	0.440	0.798
60% EDV(%)	-12.1±0.7	-11.8±0.7	0.804	-12.2±0.6	-12.4±0.6	0.844	0.927	0.557	0.739
50% EDV(%)	-15.3±0.7	-15.0±0.7	0.764	-15.3±0.6	-15.5±0.7	0.782	0.945	0.716	0.673
40% EDV(%)	-18.6±0.7	-18.2±0.8	0.719	-18.3±0.6	-18.6±0.7	0.743	0.987	0.943	0.619
LONGITUDINAL Diastolic strain									
90% EDV(%)	-1.5±0.4	-1.9±0.4	0.428	-2.2±0.4	-2.6±0.3	0.348	0.214	0.104	0.883
80% EDV(%)	-3.6±0.6	-4.0±0.6	0.611	-4.7±0.5	-5.2±0.6	0.348	0.318	0.097	0.926
70% EDV(%)	-6.3±0.7	-6.6±0.8	0.758	-7.6±0.6	-8.1±0.7	0.336	0.432	0.121	0.860
60% EDV(%)	-9.8±0.7	-9.9±0.8	0.947	-10.9±0.6	-11.3±0.8	0.371	0.605	0.180	0.726
50% EDV(%)	-14.0±0.7	-13.7±0.9	0.813	-14.5±0.6	-14.9±0.8	0.533	0.873	0.349	0.587
40% EDV(%)	-18.9±0.7	-18.1±1.0	0.608	-18.4±0.7	-18.8±0.9	0.722	0.866	0.882	0.514
LONGITUDINAL SYS-DIA Gradient									

90% EDV(%)	-1.2±0.4	-0.9±0.4	0.501	-0.8±0.3	-0.4±0.4	0.243	0.185	0.298	0.806
80% EDV(%)	-2.4±0.5	-1.9±0.6	0.519	-1.4±0.4	-0.9±0.6	0.318	0.269	0.138	0.915
70% EDV(%)	-2.9±0.6	-2.4±0.7	0.552	-1.6±0.5	-1.2±0.7	0.415	0.344	0.101	0.847
60% EDV(%)	-2.8±0.5	-2.3±0.7	0.604	-1.4±0.5	-1.1±0.6	0.488	0.413	0.078	0.865
50% EDV(%)	-1.9±0.4	-1.7±0.5	0.764	-0.8±0.4	-0.6±0.5	0.558	0.560	0.062	0.987
40% EDV(%)	-0.4±0.3	-0.6±0.4	0.654	0.1±0.3	0.2±0.4	0.781	0.797	0.156	0.571
TRANSVERSE Systolic strain							**T**	**I**	**T*I**
90% EDV(%)	3.6±0.7	4.8±1.5	0.412	2.0±0.6	4.5±1.3	0.112	0.072	0.382	0.509
80% EDV(%)	7.4±1.1	10.3±2.4	0.226	6.2±1.0	9.3±2.2	0.252	0.097	0.570	0.956
70% EDV(%)	11.8±1.6	16.0±3.1	0.190	11.2±1.4	14.0±2.8	0.448	0.161	0.590	0.760
60% EDV(%)	16.8±1.9	21.8±3.4	0.186	17.1±1.8	18.4±3.1	0.749	0.298	0.529	0.533
50% EDV(%)	22.5±2.4	27.8±3.5	0.204	23.6±2.1	22.7±3.2	0.853	0.573	0.425	0.351
40% EDV(%)	28.9±2.9	33.9±3.7	0.251	30.9±2.7	26.7±3.4	0.477	0.987	0.346	0.223
TRANSVERSE Diastolic strain									
90% EDV(%)	2.7±0.6	2.1±0.7	0.660	1.8±0.6	3.0±0.6	0.233	0.647	0.984	0.275
80% EDV(%)	6.0±1.0	5.2±1.2	0.719	5.5±0.9	6.4±1.0	0.547	0.912	0.667	0.506
70% EDV(%)	10.3±1.3	10.0±1.5	0.896	10.3±1.2	10.5±1.4	0.913	0.993	0.797	0.863
60% EDV(%)	15.5±1.7	16.3±2.0	0.759	16.1±1.5	15.3±1.8	0.794	0.963	0.918	0.696
50% EDV(%)	21.7±2.3	24.2±3.0	0.433	23.1±2.0	20.9±2.7	0.617	0.971	0.665	0.402
40% EDV(%)	28.8±3.1	33.8±4.5	0.329	31.3±2.8	27.1±4.0	0.520	0.993	0.515	0.277
TRANSVERSE SYS-DIA Gradient									
90% EDV(%)	0.8±0.8	3.3±1.6	0.213	0.1±0.7	1.5±1.4	0.223	0.080	0.362	0.568
80% EDV(%)	1.5±1.3	5.6±2.3	0.172	0.7±1.2	2.9±2.1	0.194	0.057	0.415	0.531
70% EDV(%)	1.9±1.6	6.5±2.6	0.164	1.0±1.4	3.5±2.3	0.189	0.053	0.410	0.529

60% EDV(%)	1.9±1.5	5.9±2.2	0.153	0.9±1.3	3.1±2.0	0.184	0.048	0.360	0.532
50% EDV(%)	1.7±1.2	3.9±1.3	0.177	0.5±1.1	1.8±1.2	0.210	0.061	0.263	0.604
40% EDV(%)	1.1±1.2	0.5±1.4	0.772	-0.3±1.1	-0.4±1.2	0.913	0.755	0.423	0.814

Data are presented as mean±SD. END, Endurance trained; RES, Resistance trained; T, Time (baseline, post training); I, Intervention (Resistance or Endurance); EDV, end diastolic volume.

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