The Impact of Distinct Exercise Training Modalities on Echocardiographic Measurements in
Patients with Heart Failure and Reduced Ejection Fraction

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Abstract

Introduction

Exercise training is an important component of multidisciplinary heart failure management. However, the effects of aerobic training (AT) versus resistance training (RT) on cardiac function in heart failure with reduced ejection fraction (HFrEF) are not well defined. We aimed to evaluate the impact of these exercise modalities on echocardiographic parameters.

Methods

Participants with stable HFrEF (ejection fraction <50%) were randomised to 12 weeks of AT, RT, or untrained control. Exercise was performed at matched relative intensities of each training modality (50-70% of maximum). Echocardiography and cardiopulmonary exercise testing were performed at baseline and after 12 weeks of training.

Results

Thirty-eight participants were randomised and 12 in each group completed the intervention (mean age 61.5 ± 1.7 years, 89% males). Peak oxygen consumption (VO_2) increased from 14.5 ± 1.3 to $17.2 \pm 1.6 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ after AT and 13.7 ± 1.2 to $16.4 \pm 1.1 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ after RT (both $p < 0.001$). In the AT group, there was a decrease in septal e' (0.052 ± 0.004 to $0.041 \pm 0.004 \text{ m/s}$), an increase in E/e' (18.2 ± 3.1 to 23.8 ± 3.5), left atrial volume (86 ± 9 to $99 \pm 10 \text{ ml}$) and right ventricular end-diastolic area (18 ± 1 to $20 \pm 1 \text{ cm}^2$) ($p < 0.05$ for all), but these were unchanged in the control and RT groups. There were no significant changes in left ventricular diameters or volumes, or right ventricular fractional area change after exercise.

Conclusions

There is a differential effect of AT versus RT on some echocardiographic parameters in HFrEF. AT was associated with evidence of **worsening** myocardial diastolic function, whilst this was not apparent after RT. Further studies are indicated to investigate the **long-term** clinical **significance** of these adaptations.

Key words: Cardiac failure, echocardiography, resistance exercise, aerobic exercise, exercise training, diastolic function

Abbreviations:

HFrEF – heart failure with reduced ejection fraction

AT – anaerobic training

RT – resistance training

VO₂ – oxygen consumption

Introduction

Exercise intolerance is a cardinal feature of heart failure with reduced ejection fraction (HFrEF). Accordingly, structured exercise training is an important adjunct to medical therapy in contemporary heart failure management.¹⁻³ Historically, exercise interventions have focused on aerobic training (AT) (walking, cycling), which have been shown to increase peak oxygen consumption (VO_2 peak), improve New York Heart Association (NYHA) functional class and quality of life,⁴ and reduce hospitalisation⁴ and mortality.^{5, 6} Resistance training (RT), which involves lifting weights, has become more widely prescribed in patients with HFrEF in recent years, to address clinically important peripheral sequelae such as reduced skeletal muscle mass and strength.^{7, 8}

The beneficial effects of AT appear to be predominantly **due** to peripheral adaptations, with only modest improvements in global left ventricular function reported in a meta-analysis of 812 patients.⁹ Even when RT and AT are combined, no improvement in left ventricular ejection fraction was observed.⁹

Exercise training results in an array of cardiovascular, respiratory, neurohormonal and skeletal muscle adaptations.¹⁰⁻¹³ During aerobic exercise, VO_2 , cardiac output and heart rate increases whilst peripheral vascular resistance decreases leading to a volume load on the myocardium.¹⁴ However, during resistance exercise, increases in VO_2 and cardiac output are less pronounced, due to increased peripheral resistance.¹⁴ The effect is a significant pressure load on the myocardium.¹⁴ Chronic exposure to the characteristic haemodynamic responses to AT versus RT is likely to result in different adaptations to the cardiovascular system.¹⁵

We have previously reported a differential effect of AT versus RT over 12 weeks on conduit vessel structure; RT increased artery diameter, decreased wall thickness, and decreased the wall:lumen ratio of the brachial artery, whereas AT increased artery diameter without modifying wall thickness or wall:lumen ratio.¹² However, very little data currently exists on the effects of AT alone versus RT alone on cardiac function in patients with HFrEF. In the current manuscript we present the impact of these different exercise modalities, performed at similar relative intensities and training volumes, on echocardiographic parameters of cardiac function in patients with stable HFrEF.

Material and Methods

Subjects

Eligible participants with HFrEF (ejection fraction <50%) were screened via detailed medical history, physical examination and plasma biochemical/haematological profile. **Participants were recruited from a specialist heart failure service and were receiving stable medical therapy for at least 3 months, including blood pressure and lipid lowering therapy. Participants were excluded if they were current smokers, had renal impairment (creatinine >250umol/L or estimated glomerular filtration rate <30ml/min/1.73m²), hepatic impairment (bilirubin >50mmol/L), hyperuricemia, exercise-induced ischaemia, severe aortic stenosis, hypertension despite treatment (systolic blood pressure >160mmHg or diastolic blood pressure >90mmHg at rest) or prior heart transplant.** Participants were randomised to 12 weeks of AT, RT, or an untrained control group who were asked to maintain their usual activity for 12 weeks.

Experimental measures

Cardiopulmonary fitness and muscular strength were assessed at baseline, six and 12 weeks of follow-up in all groups, while echocardiography was conducted at baseline and 12 weeks follow-up.

Cardiopulmonary fitness

Cardiopulmonary exercise testing was undertaken on a cycle ergometer (Corival, Lode, Groningen, The Netherlands) by an experienced clinical exercise physiologist who has conducted over 500 cardiopulmonary exercise tests previously in clinical cohorts (AM), supported by a cardiologist or cardiology registrar. Initial workload was set at 25 W with 25 W increments every 3 minutes until volitional exhaustion. Heart rate and rhythm were measured continuously with a 12 lead electrocardiogram (Cardiosoft, GE Marquette, UK). Blood pressure was measured manually with a mercury sphygmomanometer and rating of perceived exertion was assessed using the Borg Category Scale in the final 30 seconds of each stage and at peak exercise.¹⁶

Oxygen consumption (VO_2) during exercise was calculated from minute ventilation (V_E), measured using mass flow ventilometry, and simultaneous mixing chamber analysis of expired gas fractions (Vmax , Sensormedics, Yorba Linda, USA). Gas analysers and flow probes were calibrated before each test according to standard operating procedures. VO_2 and VCO_2 peak were averaged over 40 seconds during the final minute of exercise prior to volitional exhaustion and expressed relative to body weight ($\text{ml.kg}^{-1}.\text{min}^{-1}$).

One-repetition maximum strength assessment

Following a general warm up and stretching, participants were given a demonstration on each piece of equipment and instructed in correcting lifting and breathing technique, including to avoid excessive hand-gripping and Valsalva Manoeuvre. Participants were then asked to lift a low intensity weight, estimated to be less than 50% of the 1 repetitions maximum (RM) (based on the participant's gender and body mass) six times, to warm up the specific muscle groups involved in the exercise. After 2 minutes recovery, the weight was increased and the participant was required to lift the weight once only. If the weight was lifted successfully, it was increased again, and after a further 2 minutes recovery the new weight was attempted. This process continued until the maximum weight, measured to the nearest 2.5kg, was found that could be lifted once with good technique and through a full range of motion. All machine settings were documented so that these could be reproduced at follow up assessments.

Echocardiographic assessment

A certified sonographer performed complete transthoracic echocardiography on each participant at baseline and after 12 weeks using standard imaging planes as per the American Society of Echocardiography and European Association of Cardiovascular Imaging.^{17, 18} All studies were analysed offline by a cardiologist blinded to the allocation of exercise.

Left ventricular size was quantified using linear and volumetric measurements in the parasternal long-axis, apical four-chamber and apical two-chamber views. These measurements include end-diastolic and end-systolic volume and diameter. Left ventricular ejection fraction was based on the modified Simpson's biplane method of discs. Left atrial volume and right atrial area were measured in the apical four-chamber view. Left ventricular diastolic parameters were assessed by the mitral valve E-wave velocity, mitral valve A-wave

velocity, mitral E-wave deceleration time and mitral annular early diastolic (e') velocity.¹⁹

The mitral valve E-wave velocity and the mitral valve A-wave velocity were used to obtain the E/A ratio. The mitral valve E-wave velocity and the septal e' velocity were used to obtain the E/e' ratio.

Right ventricular size was quantified using linear measures of diameter at the base and measures of area in diastole and systole in the apical four-chamber view. Right ventricular fractional area change was then calculated.^{17, 18} Doppler measures of right ventricular haemodynamics included maximum tricuspid regurgitant velocity and velocity time integral through the right ventricular outflow tract.²⁰ Pulmonary vascular resistance and pulmonary artery systolic pressure were derived from these parameters²⁰

Exercise training intervention

Resistance training was undertaken in a circuit-training format using machine stack weights (Pulsestar, UK) for the following exercises: leg press, leg extension, hamstring curl, hip flexion (L and R), dual biceps curl, pectoral deck, lat. pull down and incline press. For the first six weeks of training, weights were maintained at 50% to 60% 1RM, and a 2:1 (60:30 seconds) work:rest ratio was applied. For the second 6 weeks, weights were increased to 60% to 70% 1RM with a 1:1 (45:45 seconds) work:rest ratio. Work:rest ratios were guided by an external auditory cue. Participants commenced with one set of the nine exercises, and increased to two, then three sets as tolerated over the first fortnight of training. A three-minute rest period occurred between each set, for a total of 46.5 minutes of exercise per session.

Aerobic training commenced at 50% to 60% of baseline VO_2 peak and progressed to 60% to 70% of the VO_2 peak after 6 weeks, so that the relative intensity was consistent with the RT group. Exercise duration was gradually increased as tolerated over the first fortnight of training, so that each session involved 20 minutes of cycling and 20 minutes of treadmill walking. These were separated by a 6.5-minute rest period to match the total duration of the RT group exercise intervention.

Control group participants were asked to maintain their usual activities for the 12-week period, but no formal exercise instruction was provided.

Statistical analyses

Statistical analyses were performed using SPSS version 23.0. All descriptive data are reported as mean $\pm$ **standard error of the mean (SEM)**. Paired t-test was used for comparison of means. A two-way ANOVA with post-hoc comparison tests was conducted to determine the difference between echocardiographic parameters before and after exercise for each group. The assumption of homogeneity was assessed by Levene's test of homogeneity of variances. When the assumption was satisfied, a standard two-way ANOVA with Tukey post-hoc comparison tests was conducted. However, when the assumption was violated, a two-way Welch ANOVA with Games-Howell post-hoc comparison tests was implemented instead. Statistically significant changes between baseline and post-exercise parameters are assumed at $p < 0.05$.

The Royal Perth Hospital Ethics Committee approved the study and all participants provided written informed consent.

Results

Participant Characteristics

Thirty-eight untrained HFrEF patients were randomised. Ischaemic heart disease was the predominant cause of heart failure (64%), followed by idiopathic cardiomyopathy (31%). Two subjects from the RT group withdrew from the study due to work commitments in one and non-cardiac illness in the other. Baseline characteristics for the participants completing the study are presented in Table 1.

Left ventricular ejection fraction was significantly lower at baseline in the RT compared with the control group ($p < 0.05$). There were no other significant differences between groups at baseline.

Participants in all groups were taking typical heart failure medication, which are described in detail in our previous publication.¹² To reiterate briefly, there was a high usage of neurohormonal blockade therapy consistent with guideline recommendations. In the control and AT groups, all participants were receiving an angiotensin converting enzyme inhibitor or angiotensin II inhibitor compared with 75% in the RT group. At least 80% of participants in each group were receiving beta-blocker therapy and over half of the cohort was on an aldosterone antagonist.

Cardiorespiratory fitness

The effects of exercise training on cardiorespiratory fitness have been reported previously and are summarised as follows; VO_2 peak increased from 14.5 ± 1.3 to $17.2 \pm 1.6 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ in the AT group and 13.7 ± 1.2 to $16.4 \pm 1.1 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ in the RT group (both $p < 0.001$), but decreased from 15.1 ± 1.3 to $14.1 \pm 1.1 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ in the control group ($p < 0.01$).¹² Exercise

time increased from 589 ± 47 to 716 ± 64 seconds in the AT group ($p < 0.001$) and 587 ± 67 to 680 ± 82 seconds in the RT group ($p < 0.05$), but did not change significantly in the control group (570 ± 62 to 543 ± 60 seconds).¹² Maximal heart rate, did not change significantly in the AT group (130 ± 6 to 128 ± 8 bpm), the RT group (120 ± 8 bpm to 117 ± 6 bpm) or the control group (117 ± 7 to 111 ± 8 bpm).¹²

Muscle strength

Muscle strength before and after exercise training are summarised in Table 2. RT was associated with significantly increased strength for all muscle groups except the bicep muscles after 12 weeks. In the AT group, only a significant increase in left hip flexion and hamstring strength was seen after 12 weeks.

Echocardiographic Outcomes

Echocardiographic measurements pre- and post-intervention are summarised in Table 2.

There was a significant difference in the change in septal e' velocity ($p < 0.01$), E/e' ($p < 0.01$) and right ventricular fractional area change ($p < 0.05$) between groups after exercise.

Left ventricular systolic function

Left ventricular ejection fraction increased in the RT group ($p < 0.05$) but not the AT group.

No significant changes for left ventricular diameter or volume occurred after exercise training for all groups.

Left ventricular diastolic function

E/e' increased significantly in the AT group ($p < 0.05$), but no significant change occurred in the control and RT groups after exercise training. The difference in change of E/e' between

the control and AT groups was significant ($p<0.05$), as well as between AT and RT groups ($p<0.01$). The septal e' velocity was the primary driver in the change in the ratio in the AT group from pre- to post-training, decreasing from 0.052m/s to 0.041m/s ($p<0.05$). The decrease in septal e' velocity in the AT group was significantly different to the increase in both the control ($p<0.05$) and the RT groups ($p<0.05$). In addition, left atrial volume increased significantly in the AT group ($p<0.05$), but not the RT or control groups. No significant changes were observed in mitral valve E-wave velocity, A-wave velocity or E-wave deceleration time in any group following the intervention period.

Right ventricular function

Right ventricular diameter decreased in the RT group ($p<0.05$), but no significant change occurred in the AT group from pre- to post-training. However, the difference in the change in right ventricular diameter between these groups approached significance ($p=0.058$).

Similarly, right ventricular end-diastolic area increased in the AT group ($p<0.05$) after training, with the change in right ventricular area between the AT and control groups approaching significance ($p=0.058$).

Individual E/e' , left atrial volume and right ventricular diameters pre- and post-exercise training for each group are presented in Figure 1.

Discussion

To our knowledge, this is the first randomised controlled study to compare the effects of AT and RT on left-heart and right-heart function in participants with HFrEF. We observed a differential effect of AT and RT on some echocardiographic parameters of cardiac structure

and function, whereby AT was associated with progression in some indicators of diastolic dysfunction, whilst RT was not associated with such change.

An increase in E/e' was observed following AT. In contrast, there was no significant change in E/e' following RT. In the current study, the change in septal e' velocity differed following the two exercise interventions, while mitral E-wave velocity was unchanged. Left ventricular diastolic function is influenced by left ventricular relaxation, restoring forces (early diastolic suction) and left ventricular chamber stiffness, which determine cardiac filling pressures. We hypothesise that the chronic exposure to the volume load associated with increased venous return that occurs during AT may have resulted in the change in diastolic function. This is supported by the finding of increased E/e' , left atrial volume and right ventricular volume after AT. Although, the change in septal e' velocity observed in the current study may also reflect a reduction in myocardial tissue velocity, this would need to be confirmed in future larger studies.²¹ However, it is important to note that both AT and RT resulted in similar improvements in VO_2 peak, so the changes in diastolic parameters observed following AT did not translate to a deterioration in aerobic capacity, an important prognostic indicator in patients with HFrEF.^{22, 23} Furthermore, although diastolic and systolic function are closely related, there was no significant change in left ventricular ejection fraction following AT, which may reflect our small sample size.

The increase in E/e' observed following AT in the present study is in contrast to several studies which have shown a reduction^{24, 25} or no change²⁶⁻²⁸ in E/e' following various forms of exercise training in patients with HFrEF. Furthermore, a meta-analysis of 266 HFrEF patients found that exercise training may improve the diastolic E/e' index.²⁹ Disparity between results obtained by previous studies and our study may be due to differences in

baseline characteristics of participants (i.e. use of neurohormonal medications, aetiology and severity of heart failure, and age), echocardiographic measurements with inter-observer variability, use of blinding of outcomes, and exercise training protocols (i.e. exercise frequency, intensity and duration).

In our prior analysis, RT led to a greater degree of peripheral arterial remodelling and vasodilation.¹² It is therefore possible that the lack of decline in diastolic function seen in the RT group is a result of decreased filling pressures from peripheral vasodilation.

The tissue Doppler change in left ventricular diastolic function that we observed is supported by changes in several other parameters. Firstly, left atrial size increased following AT but did not change following RT. Left atrial size is known to reflect long-term left ventricular diastolic function^{30, 31, 32} and predicts for risk of atrial fibrillation.³⁰ Thus, the increase in size of the left atrium in the AT group may reflect remodelling due to volume loading and increased filling pressures.¹⁹

Secondly, right ventricular size increased following AT but did not change following RT.

Significant differences were evident in right ventricular diameter and right ventricular area measurements between the two training groups. Right ventricular dysfunction may occur secondary to left ventricular diastolic dysfunction due to ventricular interdependence from the shared septum.³³ In addition, left ventricular diastolic dysfunction may lead to an increase in pulmonary pressures and subsequent increased right ventricular size.^{11, 34}

We found no significant changes in left ventricular dimensions after 12 weeks of exercise, although there was a small increase in ejection fraction for the RT group. Previous studies

have also demonstrated an improvement in left ventricular ejection fraction; yet, left ventricular dimensions have decreased or shown no change with various exercise modalities.^{9, 25, 35-39} More recently, ventricular and arterial elastance measurements have shown promise as a novel method in assessing overall cardiovascular performance.⁴⁰ This largely load-independent measure has been shown to improve after combined AT and RT.⁴⁰

Overall, the literature on the best exercise training modality in HFrEF remains highly inconsistent as different training modalities favour different benefits.⁹ A previous meta-analysis comparing different exercise modalities found that AT reduces left ventricular volumes and increases left ventricular ejection fraction.⁹ Increased aerobic exercise capacity, as measured by VO₂ peak, may be seen with all forms of exercise training, and does not seem to favour any specific modality.⁴¹ However, interval training also leads to a reduction in left ventricular volumes and an increase in left ventricular ejection fraction in **one** meta-analysis.⁴¹ This finding was in contrast to a different meta-analysis that found no difference in left ventricular remodelling with high-intensity interval AT compared to moderate-intensity continuous AT.⁴² It is important to emphasise these previous meta-analysis and review observations are inferential, have not been based on randomised **controlled** trials that directly compared AT and RT, and we believe that our findings are novel in this regard. Current guidelines recognise the benefits of both aerobic and resistance exercise training in patients with heart failure; however, the optimal protocol is yet to be established.⁴³

Limitations of our study include the short study period and follow-up, small participant numbers, predominant male cohort, and lack of a combined RT-AT group. **A longer follow-up time may be required to determine whether the observed changes in diastolic function result in left ventricular remodelling. Furthermore,** the limitations inherent to transthoracic

echocardiography should be noted. Volume measurements in all aspects of echocardiography are heavily dependent on geometrical assumptions, adequate image quality, and ultrasound beam orientation.¹⁷ In particular, assessment of right ventricular size and function is notoriously challenging due to the complex geometry of the right ventricle. Additionally, diastolic filling patterns are influenced by a variety of factors, such as loading conditions and ventricular compliance. As echocardiographic measurements are subject to inter-observer variability, all assessments were performed by one certified sonographer and reported by one certified cardiologist. However, intra-observer variability remains a limitation that may potentially account for differences in measurements.

Strengths of our study include a longitudinal study design and randomisation of subjects with the inclusion of a control group. In addition, a cardiologist blinded to the exercise allocation reported the echocardiography images. Exercise training was individually prescribed and closely supervised, which may lead to a higher adherence to active lifestyles.

Conclusions

There is a differential effect of AT versus RT on left ventricular diastolic function parameters in HFrEF. Although AT was associated with worsening diastolic function and adverse left atrial remodelling, VO₂ peak and exercise time improved significantly. Conversely, RT was not associated with adverse changes in cardiac structure and function. Larger trials with longer follow-up are warranted to determine whether the impact of AT on diastolic function has any long-term clinical significance in HFrEF.

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Conflict of Interest

Declaration of interest: none.

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Figure 1 – Change in E/e', left atrial volume (LAV) and right ventricular diameters (RV Diam) pre- and post-exercise training for each group. Significant change from pre- to post-exercise indicated by * ($p < 0.05$).

Table 1 – Subject characteristics at baseline

Characteristic	Control (n=12)	Aerobic Trained (n=12)	Resistance Trained (n=12)
Age (years)	64.4 ± 2.4	61.3 ± 2.8	58.8 ± 3.5
Male gender	11	11	10
Weight (kg)	86.5 ± 4.0	89.2 ± 4.5	81.2 ± 5.1
BMI (kg/m ²)	30.1 ± 1.3	30.4 ± 1.1	28.4 ± 0.8
SBP (mmHg)	123 ± 6	127 ± 4	125 ± 6
DBP (mmHg)	69 ± 3	70 ± 2	71 ± 4
Total cholesterol (mmol/L)	3.29 ± 0.21	3.57 ± 0.35	4.01 ± 0.17
Triglycerides (mmol/L)	1.07 ± 0.12	1.50 ± 0.20	1.72 ± 0.21
HDL-C (mmol/L)	1.16 ± 0.15	0.99 ± 0.06	0.97 ± 0.07
LDL-C (mmol/L)	1.63 ± 0.20	1.96 ± 0.29	2.25 ± 0.17
Diabetes mellitus	6	7	3
Rest HR (bpm)	63 ± 4	65 ± 3	67 ± 4
NYHA Class			
I	4	3	3
II	6	8	6
III	2	1	3
Etiology			
IHD	9	7	7
ICM	3	3	5
NC	0	1	0

BMI - body mass index, SBP - systolic blood pressure, DBP - diastolic blood pressure, **HDL-C – high-density lipoprotein cholesterol**, **LDL-C – low-density lipoprotein cholesterol**, HR - heart rate, NYHA - New York Heart Association, IHD - ischemic heart disease, ICM - idiopathic dilated cardiomyopathy, NC - non-compaction

Table 2 – Muscle strength before and after exercise

Muscle Group	Control (n=12)			Aerobic Trained (n=12)			Resistance Trained (n=12)		
	Pre	6 week	12 week	Pre	6 week	12 week	Pre	6 week	12 week
Leg Press	80.6 ± 9.6	86.6 ± 11.2	84.4 ± 10.3	98.8 ± 7.6	103.8 ± 6.9	100.4 ± 7.6	86.4 ± 8.1	97.7 ± 7.7†	96.1 ± 8.3*
Incline Press	31.9 ± 4.8	34.1 ± 4.6	32.2 ± 4.4	41.6 ± 5.8	40.4 ± 6.1	39.6 ± 5.9	31.1 ± 4.8	35.5 ± 5.2†	37.0 ± 5.3†
(L) Hip Flexion	38.8 ± 3.4	43.0 ± 3.7	42.3 ± 2.7	40.4 ± 4.0	45.4 ± 4.1*	46.3 ± 4.1*	40.0 ± 3.9	47.5 ± 4.1†	55.4 ± 4.0†
(R) Hip Flexion	36.0 ± 3.2	41.5 ± 3.7†	41.5 ± 2.9*	41.7 ± 3.9	45.4 ± 4.1	44.2 ± 4.1	39.2 ± 4.2	48.0 ± 4.1†	55.4 ± 4.5†
Lat Pulldown	39.7 ± 4.5	38.4 ± 3.3	38.4 ± 3.0	43.3 ± 5.4	45.4 ± 4.5	45.4 ± 4.6	38.2 ± 4.6	42.3 ± 5.3*	44.1 ± 5.2†
Hamstring Curl	37.3 ± 3.3	37.3 ± 2.7	38.2 ± 2.4	42.1 ± 3.0	44.6 ± 2.5*	45.8 ± 2.8†	42.9 ± 4.4	47.5 ± 4.9†	50.8 ± 5.6†
Leg Extension	62.0 ± 7.3	61.1 ± 8.1	63.0 ± 7.0	56.7 ± 4.0	60.8 ± 4.4	64.6 ± 6.1	59.8 ± 7.1	63.5 ± 7.3	68.3 ± 7.9†
Pec Deck	27.2 ± 3.0	29.7 ± 3.3	28.6 ± 2.8	32.5 ± 4.5	32.9 ± 4.9	35.4 ± 5.6	29.1 ± 5.1	35.2 ± 5.3†	36.6 ± 5.2†
(L) Bicep Curl	10.3 ± 1.0	10.0 ± 1.0	10.6 ± 1.1	10.7 ± 1.7	11.0 ± 1.2	11.8 ± 1.4	10.3 ± 1.4	11.0 ± 1.4	11.2 ± 1.3
(R) Bicep Curl	10.7 ± 0.9	10.7 ± 1.1	11.5 ± 1.0	11.5 ± 1.6	14.4 ± 1.8	12.5 ± 1.3	10.2 ± 1.3	10.5 ± 1.3	11.0 ± 1.3
Sum All Groups	321.3 ± 35.6	335.9 ± 36.6	335.5 ± 31.5	419.2 ± 34.4	444.1 ± 31.1*	446.0 ± 37.3*	371.7 ± 40.0	421.0 ± 42.3†	448.2 ± 42.2†

*Significant difference from pre-training (p<0.05)

†Significant difference from pre-training (p<0.01)

(L) - left, (R) - right

Table 3 – Echocardiographic parameters before and after exercise

Echo Parameter	Control (n=12)		Aerobic Trained (n=12)		Resistance Trained (n=12)	
	Pre	12 Week	Pre	12 Week	Pre	12 Week
LVEF (%)	38 ± 3	39 ± 3	29 ± 3	32 ± 4	26 ± 3	29 ± 3†
4C LVEDV (mL)	170 ± 20	175 ± 17	185 ± 15	196 ± 18	172 ± 16	178 ± 14
4C LVESV (mL)	108 ± 16	104 ± 18	128 ± 15	133 ± 18	132 ± 15	132 ± 13
LVEDD (mm)	60 ± 2	59 ± 2	63 ± 3	63 ± 3	62 ± 2	63 ± 2
LVESD (mm)	50 ± 2	49 ± 2	54 ± 3	53 ± 3	55 ± 3	54 ± 3
LAV (mL)	88 ± 8	84 ± 6	86 ± 9	99 ± 10†	101 ± 14	100 ± 13
LAV MOD BMI (mL/kg/m ²)	2.90 ± 0.22	2.74 ± 0.17	2.83 ± 0.24	3.25 ± 0.25†	3.75 ± 0.57	3.62 ± 0.47
RA Area (cm ²)	22 ± 2	20 ± 2	21 ± 2	21 ± 2	22 ± 3	22 ± 3
RV Diam (mm)	41 ± 3	40 ± 2	34 ± 2	36 ± 1	42 ± 3	39 ± 3†
RVEDA (cm ²)	20 ± 2	19 ± 2	18 ± 1	20 ± 1†	21 ± 2	19 ± 2
RVESA (cm ²)	12 ± 2	9 ± 2	11 ± 1	12 ± 1	11 ± 2	11 ± 2
RV FAC (%)*	43 ± 3	56 ± 6†	41 ± 6	38 ± 5	48 ± 5	46 ± 4
TR V (m/s)	3.11 ± 0.34	2.87 ± 0.24	2.60 ± 0.25	2.91 ± 0.19	2.66 ± 0.15	2.71 ± 0.15
PV VTI (cm)	19.5 ± 1.1	19.0 ± 1.1	17.4 ± 1.3	19.6 ± 1.9	19.2 ± 1.4	18.9 ± 1.1
PVR (WU)	1.35 ± 0.39	1.01 ± 0.26	0.97 ± 0.30	1.22 ± 0.39	1.26 ± 0.27	1.49 ± 0.19
MV E (m/s)	0.81 ± 0.08	0.77 ± 0.07	0.88 ± 0.08	0.87 ± 0.06	0.70 ± 0.07	0.67 ± 0.08
MV A (m/s)	0.73 ± 0.07	0.62 ± 0.09	0.64 ± 0.07	0.62 ± 0.06	0.66 ± 0.09	0.65 ± 0.09
EDT (msec)	184 ± 9	191 ± 10	181 ± 14	179 ± 10	193 ± 16	200 ± 18
Septal e' (m/s)*	0.042 ± 0.004	0.049 ± 0.004	0.052 ± 0.004	0.041 ± 0.004†	0.043 ± 0.003	0.050 ± 0.003
Septal a' (m/s)	0.068 ± 0.009	0.062 ± 0.007	0.057 ± 0.007	0.065 ± 0.007	0.066 ± 0.009	0.058 ± 0.006
E/A	1.29 ± 0.32	2.68 ± 1.34	1.54 ± 0.29	1.54 ± 0.20	1.32 ± 0.32	1.41 ± 0.42
E/e'*	19.0 ± 2.1	16.8 ± 1.8	18.2 ± 3.1	23.8 ± 3.5†	17.7 ± 3.3	13.8 ± 1.6

*Significant difference detected between groups after exercise (p<0.05)

†Significant difference from pre-training (p<0.05)

LVEF - left ventricular ejection fraction, 4C LVEDV - four-chamber left ventricular end-diastolic volume, 4C LVESV - four-chamber left ventricular end-systolic volume, LVEDD - left ventricular end-diastolic diameter, LVESD - left ventricular end-systolic diameter, LAV - left atrial volume, LAV MOD BMI - left atrial volume modified to body mass index, RA Area - right atrial area, RV Diam - right ventricular diameter, RVEDA - right ventricular end-diastolic area, RVESA - right ventricular end-systolic area, RV FAC - right ventricular fractional area change, TR V - tricuspid valve regurgitation velocity, PVR - pulmonary vascular resistance, MV E - mitral valve E velocity, MV A - mitral valve A velocity, EDT - E-wave deceleration time.