The effect of aerobic continuous training and detraining on left ventricular structure and function in male students

Javad Mahdiabadi[1], Mohammadali Mahdiabadi[2], Toba Kazemi[3]

[1]*Belarusian State University of Physical Culture, Minsk, Belarus*
[2]*Birjand University of Medical Sciences, Birjand, Iran*
[3]*Birjand Atherosclerosis and Coronary Artery Research Center, Birjand University of Medical Science, Birjand, Iran*

Abstract

Purpose: Regular exercise training induces cardiac physiological hypertrophy. The aim of this study was to determine the effect of aerobic continuous training and a detraining period on left ventricular structure and function in non-athlete healthy men**.**

Material: Ten untrained healthy male students (aged 18-22 years) were volunteered and participated in countryside continuous jogging programme (3days/week, at 70% of Maximum Heart Rate for 45 min, 8-weeks) and four weeks detraining afterwards. M-mode, 2-dimensional, colour and Doppler transthoracic echocardiography were performed, during resting conditions, before and after the training and after detraining period.

Results: Using t-test, we found significant difference in end systolic diameter and the posterior wall thickness, percentage shortening and ejection fraction after eight weeks training compared to before training. It was found no significant difference in end diastolic diameter, interventricular septum thickness, left atrium diameter, aortic root diameter, heart rate, systolic and diastolic blood pressures. Following four weeks detraining after training, compared with eight weeks of training was a significant difference in end diastolic diameter, percentage shortening and ejection fraction and no significant difference in end systolic diameter, posterior wall thickness, interventricular septum thickness, left atrium diameter, aortic root diameter, heart rate, systolic and diastolic blood pressures.

Conclusions: In general, eight-week aerobic continuous training and a detraining period can affect left ventricular structure and function.

Keywords: **Key words:** aerobic exercise, detraining, left ventricular function, echocardiography, health people.

Introduction

Physical training causes structural and functional changes in the heart, particularly in the left ventricle [14, 22, 25]. These changes constitute the cardiac adaptability phenomenon following the physiological, in contrast with the pathological changes brought about by hypertension and aortic stenosis [26]. In the case of disease, the heart confronts elevated pressures, but physiologically such pressures affect the heart only during physical training. The impact of physical training on cardiac structure and function depends on type, intensity and duration of training, as well as previous physical fitness, genetics and gender [4]. Continuous, long-term physical activities exert an overload on cardiac muscles, resulting in an exogenous hypertrophic pattern with normal ventricular walls and increased ventricular (especially left ventricular) volume [7, 25]. In addition, these individuals have greater diastolic filling volume, left ventricle diameter and mass, ventricular capacity, and stronger myocardial contraction, as explained by the Frank-Starling law [6]. Studies have indicated that all cardiac morphological parameters return to pre-training values after the detraining period [9, 19].

Rodrigues et al. [21] assessed the effects of 6 months of moderate-intensity aerobic training (1 hour/day, 3 times/week) on normal hearts, 23 sedentary men aged 31.1 +/- 3.5 years. After training, there was a significantly decrease in heart rate and significantly increase in Septal and posterior wall thickness. LV diameters and ejection fraction were unchanged. Obert et al. [15] in study on 25 children (11 girls, 14 boys) were enrolled in a 2 month high-intensity aerobic training program and 25 (12 girls and 13 boys) served as controls noted significant increase in left ventricular end-diastolic diameter, whereas left ventricular wall thickness and mass were unchanged. Shortening fraction and regional systolic function were also unchanged. Tjonna et al. [24] found that aerobic interval and continuous training on average three times a week on a treadmill for 16 weeks reduced systolic and diastolic blood pressure in patients with metabolic syndrome. Ciolac et al. [1] investigated the acute effects of continuous and interval aerobic exercise on 24-h ambulatory blood pressure in long-term treated hypertensive patients. They observed a decrease in mean 24-h systolic and diastolic blood pressure. Obert et al. [16] In a study on twenty-nine 10-11 year old boys and girls participated in a 13-week running program (3 x 1 h/week, intensity: > 80% HRmax) as well as after 2 months of detraining showed that LV internal chamber dimension increased (+ 4.6 %, $p < 0.01$) while wall thicknesses concomitantly decreased (-10.7%, $p < 0.05$) after training and all cardiac morphological parameters returned to pre-training values after detraining.

Many studies have been performed on competitive athlete's heart and most studies have investigated the effect of intense continuous training. One of the most important effects of regular physical training is the adaptation of the cardiovascular system. The basic importance of an "athlete's heart" is manifested in two

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fields: public health and competitive sport. Low intensity training is the most appropriate and the least dangerous type of physical load to maintain an optimal state of health of an individual. Considering the limitations in studying continuous training with low intensity and detraining, the present study was designed to assess its effects on the left ventricular structure and function in untrained healthy males.

Material and methods

Participants. The study was performed on ten non-athletic male students of the Azad Islamic University of Birjand (Iran) aged from 18 to 22 years (20.5 ± 1.58 years, 72.65 ± 9.19 kg, 174.2 ± 6.40 cm), took part in the study. The study protocol was approved by the research ethics committee of Payame Noor University of Tehran and was carried out in accordance with the Declaration of Helsinki. Each participant gave informed consent before enrolment. The students were not professional athletes and did not have any sports category. The criterion for cardiovascular health was the data obtained from the questionnaire devised by the researcher. Subsequently, the subjects' ECGs were studied to confirm their cardiac health. Before the initiation to participate in the study, the subjects were informed of the process and filled out the medical sport questionnaire and the consent form.

Training programme. Training programme was designed including a 45-minutes countryside continuous jogging with 70% of the maximum heart rate (MHR), three times a week for eight weeks. The subjects warmed up for 10 min before starting the main programme, and cooled down for 10 min after the main programme. All the training sessions were supervised by the researcher. After eight weeks of training, four weeks detraining was considered.

Echocardiography. The subjects had echocardiographic examinations before and after eight weeks training and after four weeks detraining. Before echocardiography, the students' anthropometric data were taken into consideration (height, cm; weight, kg). Ultrasonic examination was carried out before and after continuous training and detraining at rest. In the examination the wall thickness of the left ventricle and its cavity diameters were measured: left ventricular end diastolic dimension (LVEDD, mm); left ventricular end systolic dimension (LVESD, mm); percentage ejection fraction (%); percentage shortening fraction (%); interventricular septum thickness (IVST, mm); left ventricular posterior wall thickness (LVPWT, mm) left atrium diameter (LA, mm) and aortic root diameter (AO, mm). The thickness of walls was measured in the diastolic phase of the cardiac cycle.

These variables were measured at the echocardiography ward in the Birjand Vali-asr Hospital by a cardiologist. Echocardiographic examination was carried out on the unit Esaote Biomedica (Italy) with application of M-modal, two-dimensional color and Doppler transthoracic [5, 10].

The heart beat while resting was measured by 60-s count, Systolic blood pressure (SBP, mm Hg) and diastolic blood pressure (DBP, mm Hg) were measured with mercury blood pressure devices of Richter model no more than 2 times with 2–3-minute intervals on the right hand with the subject in the sitting position.

Maximum heart rate was determined by the formula:

$$HRmax = 220 \text{ beats/min} - \text{age}.$$

Statistical Analysis. Descriptive statistics, a t-test for paired data was used to assess differences between pre- and post-tests as well as detraining, and P equal to or less than 0.05 was considered as the significance level. Data normality was checked with Kolmogorov – Smirnov test.

Results

Absolute values of cardiac structural features of the subjects are summarized in Table 1. The end systolic diameter decreased and the posterior wall thickness, percentage shortening and ejection fraction increased significantly after training ($P \leq 0.05$). The end diastolic diameter, percentage shortening and ejection fraction decreased significantly after detraining ($P \leq 0.05$). The end diastolic diameter after training and the end systolic diameter after detraining did not change significantly

Table 1. Absolute values of left ventricle structural and functional features of the subjects (Means ± SDs)

Variables	Before training	After 8-weeks training	After 4-weeks detraining
LVEDD (mm)	48.8 ± 4.7	49.4 ± 3.9$^{\Psi}$	46.7 ± 3.9
LVESD (mm)	32.3 ± 2.8	29.2 ± 4.5**	31.3 ± 2.1
FS (%)	33.5 ± 5.1	40.2 ± 5.7***, $^{\Psi\Psi}$	32.2 ± 3.8
EF (%)	64.6 ± 6.7	70.1 ± 7.0***, $^{\Psi\Psi}$	60.3 ± 5.4
IVST (mm)	9.8 ± 1.8	10.12 ± 1.8	9.5 ± 1.9
PWT (mm)	7.1 ± 1.1	8.0 ± 1.4*	7.4 ± 1.0
LA (mm)	27.1 ± 3.6	27.8 ± 4.0	27.4 ± 4.9
AO (mm)	25.5 ± 2.5	24.9 ± 2.1	26.1 ± 3.6
HR (bit/min)	73.4 ± 9.0	71.2 ± 6.1	77 ± 9.0
SBP (mmHg)	120 ± 11.7	116 ±11.7	115.5 ± 13.1
DBP (mmHg)	74 ± 9.6	71.5 ± 9.4	70.5 ± 11.6

Notes: ***, **, *: significantly different from pre-training values at p<0.001; p<0.01; p<0.05; $^{\Psi\Psi}$, $^{\Psi}$: significantly different from detraining values at p<0.01; p<0.05.

(P>0.05). No significant difference was observed in the interventricular septum thickness, left atrium diameter, aortic root diameter, heart rate, systolic and diastolic blood pressures after eight week training and also after four week detraining (P>0.05).

Discussion

In the present study, following eight weeks continuous training the left ventricular end diastolic diameter increased no significantly and the end systolic diameter decreased significantly. Rodrigues et al. [21], Park et al. [18] reported no significantly change in the end diastolic diameter and end systolic diameter after aerobic exercise. Conraads et al. [2] showed a decrease in left ventricular end-systolic diameter in the trained heart failure group. Aerobic activities bring about a volume overload which increases the initial diastolic filling at rest and exercise. Decrease of left ventricular systolic diameter after aerobic training may indicate a decrease in residual blood volume in the left ventricle. This implies a better emptying of the ventricle with each systole and as a result of exercise with volume overload on left ventricle [17]. After four weeks of detraining the left ventricular end diastolic diameter decreased significantly and the end systolic diameter had no significant increase. Changes in left ventricular dimensions after detraining shows that, the effects of training on left ventricle returned to that of before training.

Percentage shortening fraction (FS %) and percentage ejection fraction (EF %) increased significantly after training. D'Andrea et al. [3] observed a significant increase in percentage shortening fraction. Haykowsky et al. [8] observed a significant increase in percentage ejection fraction. On the other hand, Park et al. [18] did not observe significant differences in percentage shortening fraction and percentage ejection fraction after aerobic exercise. The significant increase in myocardial contractility could be due to the decreased left ventricular end systolic diameter after training and as a response to the increase in the stroke volume. Thus, increase in the percentage the shortening fraction of the left ventricular muscle fibres showed indicated an increase in the volume of blood pumped by the left ventricle at each of the left ventricular contraction. These results show increased percentage ejection fraction after eight weeks of training and indicates the superiority of left ventricular function after exercise. There was a significant decrease after four weeks detraining in FS% and EF%.

During endurance training, heart needs to adapt to both volume and pressure load. The heart responds by increasing left ventricular internal diameter and left ventricular wall thickness. Hence the heart responds predominantly with eccentric hypertrophy [11, 13]. After 8 weeks of training, there was no significant increase in interventricular septum thickness and significant increase in posterior wall thickness. Rodrigues et al. [21] reported a significant increase in Septal and posterior wall thickness after aerobic training. Obert et al. [15] noted that the left ventricular wall thickness and mass were unchanged after aerobic training. Increase in diameter of posterior wall and interventricular septum thickness is probably due to an increase in the left ventricular mass index, which can be regarded as an adverse precursor of left ventricular hypertrophy. After detraining interventricular septum thickness and posterior wall thickness decreased no significantly.

After eight weeks training the left atrium diameter (LA) showed no significant change. Also after detraining not observed significant change in LA. The aortic root diameter (AO), after eight weeks training decreased no significantly and after detraining increased no significantly. Several studies have shown that structural and functional changes in the left ventricle during exercise are higher greater than in other parts of the heart [14, 20, 26]. Therefore, the training performed in this study was not effect on LA and AO.

After eight weeks training the resting heart rate (HR) decreased and after four weeks detraining increased no significantly. Meyer et al. [12] observed increase in the heart rate after training. On the other hand, Rodrigues et al. [21] showed decrease in heart rate after aerobic training. Systolic and diastolic blood pressure after training and detraining decreased no significantly. Tjonna et al. [24] Ciolac et al. [1] noted a decrease in systolic and diastolic blood pressure after training, Park et al. [18] founed significantly decrease in systolic blood pressure after aerobic exercise and Shiotani et al. [23] noted a significant decrease in heart rate and no significant changes in blood pressure. In training and competition, endurance-trained athletes sustain long intervals with high cardiac output, high heart rate, high stroke volume and a moderate increase in mean arterial blood pressure. Dynamic exercise imposes a volume load on the left ventricle. The cardiac output of trained endurance athletes may increase from 5 to 6 l/min at rest to up to 40 l/min during maximal exercise. Besides an increase in cardiac output, the blood pressure increases as well, although not to the same extent as during strength training [13]. Lack of change in heart rate, systolic and diastolic blood pressure variables in this study may be due to insufficiently long training and non-athletic subjects. Overall, these contradictory results are probably due to differences in training duration, subjects, experience, ethnicity, and gender. To obtain greater effectiveness, it may be recommended that researchers choose subjects with more available time (full time subjects) and conduct the study on a longer period of time.

Conclusions

In conclusion, increase in diastolic diameter and decrease in systolic diameter shows a volume overload on left ventricle and an enlargement of LV internal chamber dimension and an increase in wall thickness. Thus changes in the thickness of the septum and posterior wall represent the heart adaptations to an excessive stress caused by training program application and increase in left ventricle mass. Increase in the parameters of the cardiac contractile function suggests that the continuous jogging program favors the cardiac muscle strengthening. Eight weeks

aerobic continuous jogging can cause physiological hypertrophy of left ventricle in non-athletes healthy male, systolic function of subjects improves after training and decreases after four weeks detraining. In general, this training program can appropriate and effective training method to improve cardiovascular function, particularly left ventricular function in healthy people.

Conflict of interests

The authors declare that there is no conflict of interests.

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Information about the authors:

Javad Mahdiabadi; http://orcid.org/0000-0002-2442-3206; j_mahdiabadi@yahoo.com; Ph.D student of Exercise Physiology, Belarusian State University of Physical Culture; Pobediteley Ave., 105, Minsk, 220020, Republic of Belarus.

Mohammadali Mahdiabadi; http://orcid.org/0000-0002-9231-9718; mahdiabadi_ma@yahoo.com; MD, Assistant Professor of Internal Medicine, Birjand University of Medical Sciences; Ayatollah ghafari street, Birjand, Iran.

Toba Kazemi; http://orcid.org/0000-0002-6204-4514; drtooba.kazemi@gmail.com; MD, professor of cardiology, Birjand Atherosclerosis and Coronary Artery Research Center, Birjand University of Medical Science; Ayatollah ghafari street, Birjand, Iran.

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