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Original Scientific Paper

Physical training in patients with chronic heart failure of ischemic origin: effect on exercise capacity and left ventricular remodeling

Artur Klecha[a], Kalina Kawecka-Jaszcz[a], Bogumiła Bacior[a], Aleksandra Kubinyi[a], Mieczysław Pasowicz[b], Piotr Klimeczek[b] and Robert Banyś[b]

[a]I Cardiac Department, Jagiellonian University Medical College, Kraków and [b]Center for Diagnosis and Rehabilitation of Heart and Lung Diseases, the John Paul II Hospital, Kraków, Poland

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Background **Physical training is a well-known complementary treatment for chronic heart failure (CHF); however, many aspects require further studies. One of them is the impact on remodeling of the left ventricle (LV). The purpose of this study was to evaluate the effect of 6 months of training on LV, exercise capacity and safety issues in patients with ischemic CHF.**
Methods **Fifty patients (mean age 60.1 ± 9.2 years) with ischemic CHF, New York Heart Association (NYHA) classification class II and III and left ventricular ejection fraction (LVEF) ≤ 35% were randomized into groups: undergoing 6-month training (25 patients) and not trained (25 patients). In both groups at baseline and at 6 months a cardiopulmonary exercise test and magnetic resonance imaging (MRI) with evaluation of LV were performed. Training was limited by the achievement of 80% of the predicted heart rate at Vo_{2peak} achieved at the baseline cardiopulmonary exercise test.**
Results **All patients completed the 6-month observation. No serious adverse events were found in either group. Exercise capacity improved only in the trained group (Vo_{2peak} increased by 31%). At 6 months in the trained group there was a tendency towards an improvement in some LV parameters: ejection fraction, end-diastolic volume and wall motion score index (WMSI), whereas an opposite trend was seen in the controls ($P<0.05$, $P<0.05$ and $P<0.01$ for comparison of LVEFs, end-diastolic volumes and WMSIs, respectively).**
Conclusions **Six-month training in ischemic CHF patients is a safe modality. Training improves exercise capacity. There was no negative impact on LV morphology, and a trend towards improvement of functional parameters on MRI may suggest an anti-remodeling effect of training in patients with ischemic CHF.** ***Eur J Cardiovasc Prev Rehabil*** **14:85–91 © 2007 The European Society of Cardiology**

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Introduction

Chronic heart failure (CHF), a major cause of morbidity and mortality, is a serious public health problem that increases economic burden. Despite remarkable progress in the diagnosis and treatment of cardiovascular diseases, the number of patients afflicted with CHF is rising yearly. The reasons for this situation include aging of the populations in developed countries and a high prevalence of coronary artery disease and arterial hypertension, which are major causes of CHF [1].

The European Society of Cardiology, representing countries with a population of over 900 million, estimates that there are at least 10 million people with known heart failure [2]. Furthermore, the prognosis of heart failure is poor. Five-year survival is 30–40%, and patients with end-stage disease have only a 50% of chance of surviving 1 year [2].

One of the first and main manifestations of CHF is reduced exercise capacity, thus decreasing quality of life.

Correspondence to Artur Klecha MD, PhD, I Cardiac Department, Jagiellonian University Medical College, Kopernika 17 Street, 31-501 Kraków, Poland
Tel: +48 12 424 73 00; fax: +48 12 424 73 20;
e-mail: arturklecha@poczta.onet.pl

The etiology of heart failure is multifactorial. Impairment of central mechanisms (reduced cardiac output), changes in peripheral organs (skeletal muscles, respiratory system) and compensatory neurohormonal changes determine the severity of symptoms, and affect the course and long-term prognosis of heart failure [3,4].

Myocardial injury leading to mechanical impairment is the most common cause of CHF. Altered hemodynamic conditions and neurohormonal alterations promote further structural changes in the heart, referred to as remodeling. The remodeling progresses and involves the primarily unchanged myocardium, although the triggering factor is usually resolved [5]. This leads to alterations in left ventricular geometry, volume and function, and secondarily also in the other cardiac chambers and valvular apparatus, as well as to progressive hemodynamic impairment and changes in the conduction system.

Inhibition or regression of this process is one of priorities in CHF treatment. Results of many drug trials show that angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers, beta-adrenolytics and aldosterone antagonists have anti-remodeling properties [6,7]. However, the final result is still unsatisfactory. For this reason, the search for newer pharmacological approaches and non-pharmacological interventions in heart failure patients continues.

Physical training is now considered an adjunctive therapeutic tool in the management of chronic heart failure. The beneficial effects of training have been confirmed with respect to exercise tolerance, quality of life, autonomic function, endothelial function, and biochemical and ultrastructural parameters of skeletal muscles [8–10]. Belardinelli *et al.* [11] were the first to demonstrate, in a randomized study, that survival was improved in CHF patients undergoing physical training. This positive effect was confirmed by Piepoli [12] in the ExTraMATCH meta-analysis (nine randomized trials with 801 patients).

Opinion is divided regarding the effect of physical training on the failing left ventricle. There are data showing negative [13,14], positive [15,16] or neutral [17–19] effects of training on the remodeling process.

The purpose of the present study was to evaluate the effects of 6-month outpatient physical training on the left ventricle, exercise capacity and safety issues in patients with ischemic CHF [New York Heart Association (NYHA) class II and III].

Material and methods

The study population consisted of 50 patients (38 men, 12 women), ranging in age from 38 to 74 years (mean age 60.1 ± 9.2 years), with ischemic heart failure in NYHA class II and III and with left ventricular ejection fraction (LVEF) below 35%. All subjects gave their informed consent for the study, which was approved by the University Bioethics Committee (approval no. KBET/218/L/2002).

Patients with ischemic heart failure in NYHA class II or III, lasting over 6 months, clinically stable for at least 6 weeks on unchanged treatment and with LVEF $\leq 35\%$ were enrolled.

Exclusion criteria included uncontrolled arterial hypertension, history of major ventricular arrhythmias, acute coronary syndrome, percutaneous coronary intervention (PCI) or brain event 3 months prior to the study, atrial flutter/fibrillation or other arrhythmia making it impossible to perform magnetic resonance imaging (MRI), previous coronary artery bypass graft (CABG) surgery, implanted cardioverter-defibrillator and/or pacemaker or the presence of metal parts in the body, signs of osteoarticular dysfunction excluding participation in physical training, diabetes mellitus, chronic lung disease and major anemia.

According to the protocol, all enrolled patients received an ACE inhibitor (lisinopril) and a beta-blocker (carvedilol) in maximal tolerated doses. Other drugs were used optionally if indicated. Patients were randomized either to a physical training group (25 patients, group A) or a control group (25 patients, group B). At baseline and at 6 months all subjects underwent clinical examination, cardiopulmonary exercise test and MRI.

Clinical examination was performed to analyze age, sex, body mass index (BMI), duration, etiology and severity of CHF (NYHA class) and the presence of arterial hypertension, prior myocardial infarction(s), accompanying diseases and medication.

The cardiopulmonary exercise test was performed on a treadmill (Marquette Electronics Case15, Milwaukee, Wisconsin, USA), according to the modified Bruce protocol. Metabolic parameters were measured using a Sensor Medics Vmax 29 C-2130 Spirometer (Sensor Medics Corp., Yorba Linda, California, USA). An electrocardiographic exercise test was performed following the guidelines of the European Society of Cardiology [20]. Medical treatment was not discontinued before the test. The test was terminated at the point of physical exhaustion and/or dyspnea. Peak oxygen consumption (Vo_{2peak}) was determined as the mean of measurements taken in the last 30 s of the exercise. The anaerobic threshold (AT) was determined non-invasively via the V-slope method during a progressive exercise test [21].

Magnetic resonance imaging was performed using a 1.5 Tesla system (Magnetom Sonata Maestro Class; Siemens, Erlangen, Germany). A circularly polarized 400 mm body array was used for both radio frequency transmission and the reception of the nuclear magnetic resonance (NMR) signal. The system was equipped with actively shielded gradient coils, giving gradient strengths of up to 40 mTm_1 with rise times of 1 ms. All images were obtained in breathhold.

Single-shot techniques were used to localize and define the axes of the left ventricle (LV) using the following scout views: stack of coronal images; transverse midventricular images; oblique sagittal long-axis view; and double-oblique short-axis view. Next, on the basis of the formerly performed scouts, the two ECG triggered breathhold cine acquisition in long-axis orientation (four-chamber and two-chamber views) was performed (FLASH-2D or True-FISP). By using the end-diastolic cine frame of this long-axis view a series of parallel short-axis image planes was defined, covering the entire LV from the mitral annulus level to the apex. Then, steady-state cine MR images encompassing the entire LV were obtained on 9–12 contiguous short-axis imaging planes from the apex to the base. These short-axis (SA) planes were perpendicular to the long axis of the LV and, at every SA, breathhold-cine aquisitions were performed (slice thickness of 10 mm and slice gap 0 mm). In most patients the 2D True-FISP cine sequence [True-FISP: flip angle, 60°; time of repetition (TR), 30 ms; time to echo (TE), 1.08 ms; voxel diameters $1.1 \times 1.25 \times 10\,mm^3$; base resolution, 256×256; phase resolution, 75%] was used. In case of artifacts in the True-FISP sequence it was replaced by cine FLASH-2D (cine gradient echo: flip angle, 30°; TR, 100 ms; TE, 4.8 ms; voxel diameters, $1.1 \times 1.25 \times 10\,mm^3$; base resolution, 256×256; phase resolution, 75%). The aquisition window was adjusted individually to patient average RR interval (TR maximum). LV epicardial and endocardial contours were delineated along the myocardial border in end-diastolic and end-systolic cine frames for every slice by using the Argus package quantitative volumetric analysis [22,23]. The following LV function, thickness and volumetric parameters were calculated: ejection fraction (EF, %), end-diastolic dimension (EDD, mm), end-systolic dimension (mm), posterior wall – diastole (mm), posterior wall – systole (mm), ventricular septum – diastole (mm), ventricular septum – systole (mm), end-diastolic volume (EDV, ml/m^2) and LV mass index (g/m^2).

The intraobserver reproducibilities assessed formally and calculated using the Bland–Altman formula for volumetric parameters in our lab were respectively: EDV, 0%; EF, 4.0%; LV mass index, 4.0%; EDD, 4.0%; ventricular septum, 4%; posterior wall, 5%.

A visual analysis of segmental wall motion and thickening was based on a 17-segment model recommended by the American Heart Association [24]. The following scale of wall motion disorders was used: 1, normokinesis; 2, hypokinesis; 3, akinesis; 4. dyskinesis. The wall motion score index (WMSI) was derived from the summation of individual segment scores divided by the number of interpreted segments.

One investigator who was blinded to the treatment group to which the patient had been assigned, analyzed all MRI examinations.

Physical training model

Patients trained three times a week for 6 months in the outpatient Department of Rehabilitation at I Cardiac Department, Jagiellonian University Medical College. Each patient completed about 72 training cycles. The maximum allowable training load was adapted for each patient individually in a cardiopulmonary exercise test limited by the achievement of 80% of the predicted heart rate at Vo_{2peak} achieved at baseline. Sixty-minute sessions were divided into three parts: 20 min warming up, 25 min training on a cycloergometer and 15 min relaxation exercises. Groups consisted of six subjects [4,25].

Warm-up exercises involved all muscles. Both dynamic and static exercises were performed. The intensity and number of exercises were increased with time. The exercise program comprised bicycling on a Monark cycloergometer (Varberg, Sweden). Pulse rate and ECG were monitored constantly and blood pressure was measured intermittently. The exercise period was preceded by a 2-min test with zero load followed by four 3-min intervals with gradually increasing loads and three 3-min intervals with no load. If at any point in a given stage the predicted heart rate was exceeded (80% of the maximal heart rate at baseline Vo_{2peak}), the workload was decreased.

Warming down was used for psychophysical relaxation to relapse into a state resembling autohypnosis in which the patient is relaxed and conscious of external stimuli.

Statistics

Statistical analysis was carried out using the STATISTICA 6.1 PL package (StatSoft Inc., Tulsa, Oklahoma, USA). Unpaired Student's *t*-test was used for baseline characteristics of the two groups for continuous variables, and qualitative variables were tested with chi-squared test (χ^2). All data were analyzed comparing the initial (baseline) with the final study (after 6 months). Multivariate repeated-measures analysis of variance (ANOVA) was used for assessment of differences between the two groups and changes over time within each group, as well as any interaction (different trends over time between the groups). A P value < 0.05 was considered significant. Results are expressed as mean ± SD.

Results

Clinical examination

At baseline the groups did not differ significantly in clinical characteristics (Table 1). The only exception was smoking; the trained group consisted of significantly more ex-smokers.

There were no significant differences in the basic pharmacological treatment. Among drugs used optionally, only nitrates were given significantly more frequently in the control group (Table 2). At baseline in both groups the doses of ACE inhibitor (lisinopril) and beta-blocker (carvedilol) were comparable: in group A the average dose of ACE inhibitor was 11.6 mg daily (range 2.5–20 mg) and for carvedilol it was 25 mg daily (6.25–50 mg). In group B the doses were 12.6 mg daily (2.5–20 mg) for lisinopril and 22 mg (6.25–50 mg) for carvedilol.

All patients in both groups completed the 6-month observation. There were no serious adverse events. Ten patients in group A and 13 patients in group B (40 versus 52%; NS) required modification of pharmacotherapy due to intermittent worsening of CHF. In most cases the dosage of orally given furosemide was increased temporarily (seven patients in group A and eight in the control group). In two patients in group A and two patients in group B it was necessary to reduce the dosage of the ACE inhibitor due to hypotension. In one patient in the trained group and two in controls the dosage of beta-blocker was reduced due to bradycardia. In one patient in group B the dosage of aldosterone antagonist was reduced due to hyperkalemia.

Cardiopulmonary exercise test

At baseline the groups did not differ in exercise capacity. At 6 months exercise tolerance was significantly improved only in the trained group. In the controls cardiopulmonary parameters did not change significantly (Table 3).

Left ventricle

Baseline LV parameters were similar in the two groups. At 6 months there were no significant changes in the trained group, but there was a tendency towards an improvement in LVEF, end-diastolic volume and WMSI, whereas the opposite trend was observed in the controls ($P < 0.05$; $P < 0.05$ and $P < 0.01$ interaction for LVEF, end-diastolic volume and WMSI, respectively) (Table 4).

Table 1 Demographic and clinical data in group A (trained) and group B (control)

	Group A ($n=25$)	P	Group B ($n=25$)
Age (years)	59.6 ± 10.2	NS	61.2 ± 9.5
BMI (kg/m^2)	26.6 ± .9	NS	27.3 ± 3.2
Male gender	20 (80%)	NS	18 (72%)
Arterial hypertension	18 (72%)	NS	19 (76%)
Hyperlipidemia	21 (84%)	NS	19 (76%)
Myocardial infarction in the past	24 (96%)	NS	23 (92%)
Ex-smokers	18 (72%)	$P<0.05$	12 (48%)
Duration of CHF (months)	30.4 ± 11.2	NS	24.5 ± 10.2
NYHA class III	11 (44%)	NS	10 (40%)

BMI, body mass index; CHF, chronic heart failure; NYHA, New York Heart Association.

Table 2 Medication in groups A and B

	Group A ($n=25$)	P	Group B ($n=25$)
ACE inhibitor (lisinopril)	25 (100%)	NS	25 (100%)
Beta-blocker (carvedilol)	25 (100%)	NS	25 (100%)
Loop diuretic (furosemide)	15 (60%)	NS	17 (68%)
Other diuretic	1 (4%)	NS	0 (0%)
Aldosterone antagonist	10 (40%)	NS	8 (32%)
Nitrate	9 (36%)	$P<0.01$	17 (68%)
Acetylsalicylic acid	25 (100%)	NS	25 (100%)
Oral anticoagulant	2 (8%)	NS	2 (8%)
Digitalis	9 (36%)	NS	8 (32%)
Antiarrhythmic (amiodarone)	4 (16%)	NS	5 (20%)
Statin	24 (96%)	NS	24 (96%)
Other hypolipemic agent	1 (4%)	NS	1 (4%)

ACE, angiotensin-converting enzyme.

Table 3 Cardiopulmonary exercise data in group A (trained) and B (control) at baseline and at 6 months

	Group A (25 patients)		Group B (25 patients)	
	Baseline	At 6 months	Baseline	At 6 months
Duration (s)	378 ± 44	752 ± 85**	385 ± 49	361 ± 42‡
Workload (watts)	105.6 ± 23.4	143.8 ± 31.7**	110.1 ± 25.1	101.8 ± 21.9‡
Rest HR (bpm)	77 ± 21	70 ± 18*	78 ± 18	76 ± 20†
Peak HR (bpm)	121 ± 30	109 ± 27*	125 ± 29	128 ± 33†
Resting systolic BP (mmHg)	114 ± 18	112 ± 15	116 ± 21	112 ± 17
Resting diastolic BP (mmHg)	77 ± 9	76 ± 10	78 ± 10	75 ± 11
Peak systolic BP (mmHg)	153 ± 23	144 ± 21*	149 ± 21	145 ± 20
Peak diastolic BP (mmHg)	87 ± 8	86 ± 9	86 ± 9	84 ± 8
Vo_{2peak} (ml/kg per min)	14.6 ± 2.9	19.2 ± 3.8**	14.8 ± 3.2	14.1 ± 2.5‡
Vo_2AT (ml/kg per min)	10.4 ± 2.5	12.9 ± 3.2*	10.6 ± 2.8	9.9 ± 2.4‡
VE/Vco_2 slope	38.4 ± 4.1	34.3 ± 3.9*	37.4 ± 4.3	38.8 ± 4.8‡
VE/Vo_2	32.2 ± 5.2	25.5 ± 4.6**	30.6 ± 4.7	31.8 ± 6.1‡

BP, blood pressure; HR, heart rate; VE/Vco_2, ventilatory equivalent for carbon dioxide; VE/Vo_2, ventilatory equivalent for oxygen; Vo_2AT, Vo_2 at anaerobic threshold; Vo_{2peak}, peak oxygen consumption. *$P<0.05$, time effect within group; **$P<0.01$, time effect within group; †$P<0.01$, interaction; ‡$P<0.001$, interaction.

Discussion

Physical training seems to be underestimated and underused in the treatment of CHF, mainly because of poor knowledge about this modality and controversies about its effect on LV remodeling.

Exercise capacity

The present study was designed to analyze the effect of physical training on exercise tolerance in CHF patients. In the group trained for 6 months there was a significant improvement of the most important parameters of

Table 4 MRI assessment of the left ventricle in group A (trained) and B (control) at baseline and at 6 months

	Group A (25 patients)		Group B (25 patients)	
	Baseline	At 6 months	Baseline	At 6 months
Ejection fraction (%)	27.4 ± 5.7	30.2 ± 7.8	28.6 ± 5.2	27.9 ± 5.8†
End-diastolic dimension (mm)	65.6 ± 7.1	63.9 ± 8.3	64.9 ± 8.3	66.4 ± 7.0†
End-systolic dimension (mm)	53.2 ± 6.2	52.6 ± 6.8	52.8 ± 7.8	54.5 ± 7.4
Posterior wall – diastole (mm)	9.1 ± 0.7	9.5 ± 0.8	9.3 ± 0.9	9.3 ± 1.0
Posterior wall – systole (mm)	12.2 ± 1.1	12.1 ± 1.0	12.8 ± 1.3	12.5 ± 1.1
Ventricular septum – diastole (mm)	9.8 ± 0.6	10.1 ± 0.9	10.1 ± 1.2	10.4 ± 1.2
Ventricular septum – systole (mm)	12.7 ± 1.2	12.5 ± 1.0	13.0 ± 1.5	12.7 ± 1.3
End-diastolic volume (ml/m^2)	122.6 ± 20.1	114.8 ± 19.3	125.1 ± 20.8	127.3 ± 23.1†
LV mass index (g/m^2)	126.3 ± 20.1	120.7 ± 26.5	130.2 ± 31.0	133.5 ± 27.6†
Wall motion score index	2.17 ± 0.6	1.94 ± 0.5	2.08 ± 0.5	2.14 ± 0.6‡

LV, left ventricular. *$P<0.05$, time effect within group; **$P<0.01$, time effect within group; †$P<0.05$, interaction; ‡$P<0.01$, interaction.

exercise capacity obtained in the cardiopulmonary exercise test. The duration of exercise and maximal load achieved in the control test were significantly increased. Peak oxygen consumption ($V_{O_{2peak}}$) increased by about 31%. The present findings confirm the reports of other investigators. An analysis of 16 randomized studies (involving 439 patients) showed that $V_{O_{2peak}}$ increased by 12–31% depending on the duration of the exercise program, age of the study population and training protocol [25]. The VE/V_{CO_2} slope was significantly reduced from 38.4 ± 4.1 to 34.3 ± 3.9, $P < 0.05$ in the trained group. Kiilavuori *et al.* [26], Meyer *et al.* [27] and Coats *et al.* [28] obtained similar results demonstrating the beneficial effects of regular training on ventilatory efficiency and the VE/V_{CO_2} slope value. In the trained group, the oxygen consumption at anaerobic threshold ($V_{O_2}AT$) was also increased, from 10.4 ± 2.5 ml/kg per min to 12.9 ± 3.2 ml/kg per min ($P < 0.05$). It should be emphasized that 11 ml/kg per min is the cutoff value of $V_{O_2}AT$ associated with poor prognosis, as shown by Myers *et al.* [29]. Belardinelli *et al.* [11] and Wielenga *et al.* [30] also demonstrated a marked improvement of $V_{O_2}AT$ in their trained study groups.

Left ventricle

The present findings and other reports demonstrate the beneficial effects of regular physical training on exercise capacity in CHF patients. The impact of training on LV remodeling is, however, ambiguous. Concerns about harmful effects of training on cardiac remodeling resulted in discontinuation of this approach in the 1980s/1990s. Skeptical investigators reported that regular exercise may negatively influence LV parameters. In 1988 Judgutt *et al.* [13] demonstrated that global and regional LV contractility deteriorated after a 3-month training program initiated 15 weeks after myocardial infarction. Ehsani *et al.* [14] reported that end-diastolic volume in patients with coronary artery disease was increased by 9% after 12 months of training. In 2004 Kubo *et al.* [31] stated that exercise aggravates LV remodeling in patients after extensive anterior myocardial infarction.

However, most investigators do not confirm the negative effects of physical training on LV function. Dubach *et al.* [17] performed MRI to assess the effects of physical training (25 patients, training for 2 months) and did not demonstrate significant changes in LV volume and ejection fraction. The results of the EXERT study (The Exercise Rehabilitation Trial) [10] and that reported by Myers *et al.* [29] were similar. Belardinelli *et al.* [32] showed that LV contractility on dobutamine echocardiography and myocardial perfusion on thallium scintigraphy were improved in the trained group. Hambrecht *et al.* [15], in a similar population, demonstrated significantly increased LV ejection fraction and reduced dimensions. Giannuzzi *et al.* [16] obtained the most spectacular results, demonstrating significant improvements in ejection fraction, end-systolic and end-diastolic volumes. The improvement was even more important bearing in mind the significant deterioration of the same parameters in the controls.

The present findings do not confirm the negative effects of physical training on myocardial remodeling. Although the most important LV functional parameters (ejection fraction, end-diastolic volume, wall motion score index) assessed on MRI did not improve significantly in the trained group, there was a tendency towards an improvement at 6 months after training. The opposite trend was observed in the controls.

In the present study the LV was assessed by MRI. At present MRI is an accepted gold standard for LV assessment, being a more precise, reproducible and accurate technique than echocardiography [33,34]. These advantages of MRI were very important for defining the size of the population in the present study.

Another important issue in the presented trial were inclusion and exclusion criteria (for instance patients with diabetes mellitus or established atrial fibrillation were excluded), which assured the relatively high homogeneity of the study population, thus increasing the statistical reliability of the data after elimination of confounding factors. The size of the study was established in compliance with earlier recommendations. In 2004 Smart and Marwick [35] analyzed 30 randomized studies of physical training in CHF where the number of patients ranged from 16 [36] to 181 [10].

Taking into account the natural course of CHF, in which LV function gradually deteriorates due to remodeling, the

fact that this process can be interrupted argues in favor of physical training. In contrast, LV deterioration is frequently observed in untrained subjects followed for several months. In 2003 Giannuzzi *et al.* [16] analyzed the results of the exercise in left ventricular dysfunction and chronic heart failure (ELVD-CHF) trial and coined the term 'antiremodeling effect of long-term physical training'. In their opinion, available evidence suggests that, similar to certain drugs, regular physical training inhibits the process of LV remodeling in CHF. The results of the present study seem to confirm this observation.

Our data also indicate that the observed improvement of exercise tolerance can not be explained by a favorable training effect on LV remodeling. An increase of physical capacity is mainly related to peripheral rather than central mechanisms. CHF is characterized by impaired ability of peripheral vessels to vasodilation and abnormal vascular compliance [37]. Previous trials documented that regular training improves endothelial function (increase of nitric oxide production), leads to reduction of the peripheral vascular resistance and, finally, to increased skeletal muscle blood flow [15,38]. Other important mechanisms are related to changes in skeletal muscles. An increase in mitochondrial volume density, improvement in oxidative metabolism and positive structural changes may have relevant influence on exercise tolerance in CHF [39]. Regular training also attenuates excessive ergoreflex activation, leading to correction of the ventilatory response to exercise [40].

This study was performed by a single experienced center. Due to very restricted inclusion and exclusion criteria (because of the diagnostic methods used) the population in this trial may be not representative of an average population in clinical practice (patients with atrial fibrillation or with ferromagnetic elements or devices in the body were excluded). The small sample size may also be a limitation of the study.

In conclusion, outpatient physical training for 6 months in subjects with ischemic CHF significantly improves exercise tolerance assessed by a cardiopulmonary exercise test. A tendency towards an improvement of LV function in trained patients and an opposite trend observed in the controls may imply an 'anti-remodeling' effect of physical training in CHF patients. Outpatient physical training is a safe treatment modality in patients with ischemic CHF in NYHA class II and III.

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