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Effects of moderate physical training program in post-myocardial infarction patients with arterial hypertension

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Abstract

The clinical effectiveness of physical training in a Cardiac Rehabilitation Program (CRP) was assessed in hypertensive (Arterial Hypertension, AH), post-Myocardial Infarction (MI) patients. 206 patients were randomized into a physically trained group (PhTG, n=102) and an untrained, control group (CG, n=104). All patients received standard drug therapy. PhTG patients performed mild callisthenic exercises and moderately intensive bicycle exercise three times/week for one year. Compared to control patients, PhTG patients had significant changes in exercise capacity (duration +38%, $p<0.001$; total work +63.6%, $p<0.001$); rate-pressure product (-8.2%, $p<0.01$); left ventricular ejection fraction (+7.6%, $p<0.001$); left ventricular stroke volume (+5.1%, $p<0.01$). Resting BP decreased in PhTG patients (systolic BP, -3.1%, $p<0.05$; diastolic BP, -3.5%, $p<0.001$), but increased in CG patients (systolic BP, +3.1%, $p<0.05$; diastolic BP +3.4%, $p<0.05$). PhTG patients had fewer myocardial ischemic episodes, including painless ischemia during exercise, fewer angina attacks, less nitroglycerin consumption, improved quality of life, fewer cardiovascular events (-50%, $p<0.05$), and days of absence from work (-43.2%, $p<0.05$). Thus, supplementing a CRP with moderate exercise improved BP, work capacity, cardiac function, and quality of life in hypertensive, post-MI patients.

Key Words: arterial hypertension, myocardial infarction, rehabilitation, cardiovascular bioindicators, physical exercise.

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Arterial Hypertension (AH) is a well-known predisposing factor for pathological changes in various organs and tissues that lead to atherothrombotic diseases, such as, ischemic heart disease (IHD), unstable angina, Acute Myocardial Infarction (AMI), and stroke.[1] In AH patients, structural and functional disorders have been detected in all parts of the circulation, from microvessels to large vessels, including the aorta and Coronary Arteries (CA).[2,3] Among the possible mechanisms of AH's influence on atherogenesis are increased vascular permeability and adhesiveness of endotheliocytes, oxidative stress, and activation of inflammatory processes by angiotensin II (ATII). Results include endothelial dysfunction with weakening of endothelium-dependent vasorelaxation, accumulation of oxidized forms of Low Density Lipoproteins (LDL) in the subendothelial space, migration of smooth muscle cells and formation of atheroma that can be subjected to calcification and ulceration.[4-6] AH is one of the factors that destabilize atherosclerotic plaque, and sudden rupture of atherosclerotic plaque in CA or carotid arteries likely results in AMI or stroke, respectively.[7,8] Natali *et al.* reported that 41% AH and IHD patients had three-vessel CA disease according to coronary angiography data, and that their index of CA stenosis was 19% higher as compared to patients without AH.[9] In AH patients, the average cardiovascular death rate was significantly higher after 96 months.[9] In a follow-up study, the risk of developing non-fatal AMI in AH patients who underwent AMI was 21% higher than in patients without AH.[9]

The epidemiological study "ERSPECTIVE" (Perspective of antianginal therapy in Russian Federation, n=2768) carried out in 2012-2013 found that 91.3% of patients with angina pectoris had AH.[10] Notably, AH had been dia-

gnosed 4.1 ± 0.2 years before the appearance of angina pectoris. In 61.6% of AH patients, the level of average Blood Pressure (BP) remained higher (146/88 mmHg) than the recommended target ranges, despite treatment with two or more antihypertensive drugs.[10] According to the CLARIFY register (prospeCtive observational LongitudinAl RegIstry of patients with stable coronary arterY disease), which included 33 000 patients with stable IHD in 45 countries, AH occurred in 71.1%.[11]

Clearly, the degree of AH control in post AMI patients would directly affect the clinical course of the disease and its prognosis. Prior studies have shown beneficial effects of Physical Training (PhT) in patients with cardiac disease, including AH.[12-17] However, these studies did not specifically evaluate the effects of PhT in AH patients following AMI. Thus, the current study was designed to evaluate in working patients with AH, the clinical efficacy of a one-year PhT rehabilitation program.

Materials and Methods

The study was carried out in accordance with Good Clinical Practice standards and with the Helsinki Declaration principles. Written informed consent was obtained from all participants of this study. Ethics Committee of the National Medical Research Center for Therapy and Preventive Medicine, Moscow, Russian Federation, approved the Study Protocol on March 19, 2015 (Protocol #3).

206 working patients, men<60 and women <55 years old, who had had ST-elevated AMI, and performed thrombolysis, between three and eight weeks previously were included in this randomized clinical study involving 10 medical centers from 10 Russian Federation cities. Patients were randomized prospectively into two groups: a physically trained group (PhTG, n=102), who participated in a PhT rehabilitation program in addition to standard medical therapy, and a control group (CG, n=104), who received only standard medical therapy. Medical therapy included b-Adrenergic Blockers (bAB), antiplatelet drugs, Angiotensin-Converting Enzyme (ACE) inhibitors, statins, diuretics and nitrates. Patient records revealed no systematic difference in drug therapy between the groups. The study lasted for one year.

Three times per week, PhTG patients performed mild callisthenic exercises and trained on a stationary bicycle at an intensity of 50-60% of their respective maximal power value (MxP, W), as determined pre-study by a cycle ergometry test (CE-test). The CE-test was performed on a bicycle ergometer with an increase of 25 W every 3 min from the initial exercise power of 25 W until clinical or ECG criteria for stopping the exercise was observed, or until submaximal Heart Rate (HR)[18] was reached, *i.e.*, the maximal CT-test power value (MxP, W). Other CE-test values included i) exercise time (ET; min); ii) Threshold Power (ThP, W); iii) total work (ToW, kJ) computed by ThP × ET × 60/1000; iv) Rate-Pressure Product (RPP, units) computed by (HR× systolic BP)/100. The CE-test was performed on all subjects at the beginning of the study and at one year. Prior to the CE-test, BAB and nitro-drugs were withheld 48 and 24 hours, respectively. The potential therapeutic benefits of the physical rehabilitation program were assessed by comparing clinical, instrumental, and laboratory data at 1 year with pre-study data and with respective CG patient data.

Clinical data included medical history, physical examination, measurements of BP and HR, and calculation of body mass index (BMI, kg/m^2). Instrumental examination included standard ECG during and after the CE-test to determine rhythm disturbances and the number of ST-segment depressions (symptomatic or silent) ≥1 min. While clinical variables were monitored during the study, only values recorded before and at the conclusion are presented in this report.

Echocardiography (EchoCG) was performed to determine cardiac linear and volume parameters: maximum transverse Left Atrium (LA) dimension, LV End-Diastolic Volume (EDV) and End-Systolic Volume (ESV), Interventricular Septum Thickness (IST) at end-diastole, LV end-diastolic and systolic posterior wall thickness, LV ejection fraction (EF, Simpson method), LV ventricular ejection (VE), maximal transmitral flow at beginning of P wave (E peak) and during atrial systole (A peak) and the E/A ratio. Stroke volume was computed from the difference between LV EDV and LV ESV and then multiplied by heart rate to compute cardiac output. Total Peripheral Resistance (TPR) was calculated from mean arterial pressure divided by cardiac output. Cardiac index was computed by dividing cardiac output by estimated body surface area.

Laboratory testing was performed after 12-14 hr fasting. These tests included determination of total cholesterol (TC, mmol/L) and triglycerides (TG, mmol/L) using a "Mars" autoanalyzer (Korea) with enzymatic diagnostic kits. High-density lipoprotein cholesterol (HDL-C, mmol/L) was detected by the same method as for TC measurement in the supernatant after LDL and very low-density lipoproteins (VLDL, mmol/L) precipitation with mix of sodium phosphotungstate and 0.5 M magnesium chloride. LDL content (LDL-C, mmol/L) was calculated according to Friedewald *et al*.[19]

Daily physical activity (PA) of patients was evaluated using the National Medical Research Center of Preventive Medicine motion activity questionnaire QPHA23+[20] Less than 62 questionnaire QPHA23+ points indicated a low level of PA, 62-84 points, a moderate level of PA, and more than 84 points, a high level of activity of PA. Patient Quality Of Life (QoL) was also analyzed.[21] In addition, patient diaries were analyzed to determine of the number of angina attacks and use of Nitroglycerin (NTG) for their relief.

Statistics. The SAS software package (Statistical Analysis Systems, SAS Institute, USA) was used to analyze the study results. For each quantitative scale indicator, the mean group value (M) and the standard error (SE) of the mean were determined. For nominal variables, the corresponding frequencies of different gradations were expressed as percentages. Ratios were evaluated with a chi-square test. The significance of differences between variables, including percentages, was assessed by Student's t-test for independent data or by a paired t-test for data from the same patients. The level of significance was set at $p<0.05$.

Results

Table 1 shows that most characteristics of subjects in the two group were similar, including elevated systolic and diastolic BP. 42-44% of patients had symptoms of mild to moderate heart failure (HF). The percentage of patients with diabetes mellitus and with angina pectoris was greater in the PhTG. Men predominated in the study populations, but the proportion of men in the trained and control groups did not differ significantly. Subjects were of working age, and about 50% had had at least one MI prior to a recent acute MI. The majority of study subjects (68%) had ST-Elevation Myocardial Infarction (STEMI); the remainder (32%) had Non-ST-Elevation Myocardial Infarction (NSTEMI).

During the one-year duration of the study, no patients withdrew for non-medical reasons. In the PhTG, two patients did not complete the study, one due to stroke and one due to cardiovascular death. In the CG, six patients did not complete the study, four due to stroke, one due to pulmonary embolism, and one due to cardiovascular cause (p=0.17).

Table 1. *Pre-study characteristics of study patients with arterial hypertension and recent AMI.*

Variable	PhTG (n=102)	CG (n=104)	p
Men, % / Women, %	94.1 / 5.9	91.0 / 9.0	NS
Age, years	52.7±0.7	53.2±0.7	NS
Angina pectoris, %	85.7	64.0	<0.05
Angina, CCS class	1.4±1.0	1.2±1.1	NS
Heart failure NYHA class I-II, %	41.9	44.2	NS
Previous MI, %	51.9	48.5	NS
Diabetes mellitus, %	7.0	3.4	<0.05
SBP, mmHg	134±2	133±2	NS
DBP, mmHg	85±1	84±1	NS
HR, beats/min	75±1	76±1	NS
Ejection fraction of the left ventricle (LV EF), %	56.0±10.0	58.0±9.0	NS
Body mass index (BMI)	27.7±3.3	27.8±3.6	NS
Total cholesterol (TC), mmol/l	5.5±1.2	5.7±3.2	NS
Low density lipoprotein cholesterol (LDL-C), mmol/l	3.7±1.2	3.8±1.3	NS
Triglycerides, mmol/l NS		1.8±0.9	1.8±0.9
High density lipoprotein cholesterol (HDL-C), mmol/l	1.06±0.35	1.09±0.31	NS
b-adrenergic blockers (bAB), %	92.8	92.7	NS
i-ACE, %	77.1	80.5	NS
Antiplatelet, %	95.1	98.8	NS
Lipid lowering drugs, %	74.6	71.0	NS

Values are percentages of the total (%) or means±SE; PhTG, patients in physical trained group; CG, patients in untrained, control group; CCS, Canadian Cardiovascular Society; NYHA, New York Heart Association; HF, heart failure; MI, myocardial infarction; Previous MI, MI prior to recent acute MI; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; p, probability of a statistically significant difference, PhTG vs CG; NS, p ≥0.05.

Dynamics of office BP

Initially, the patients of the two groups had similar systolic BP (SBP) and diastolic BP (DBP). After one year in the CRP, SBP of PhTG patients decreased by 3.0% from 134±2 mm Hg initially to 130±2 mm Hg ($p<0.05$), and DBP decreased by 3.5% from 85±1 mm Hg to 82±1 mm Hg ($p<0.001$). Over the same interval, SBP of CG patients increased by 3.1% from 133±2 mm Hg initially to 137±2 mm Hg ($p<0.05$), and DBP increased by 3.4% from 84±1 to 87±1 mm Hg ($p<0.05$).

Dynamics of exercise capacity and BP

Initially, the patients of the two groups did not differ in indicators of exercise capacity: ET, ThP, and ToW. After one year in the CRP, Figure 1 shows that ET of PhTG patients increased by 38% ($p<0.001$), and ToW increased by 64% ($p<0.001$). These variables had not changed significantly in CG patients at one year. Thus, at the conclusion of the study, ET and ToW were sigificantly greater in the PhTG ($p<0.001$) compared to CG. At baseline, ThP was 80±2 W in PhTG patients and 87±2 W in CG patients. After one year, ThP had increased to 107±3 W ($p<0.001$), whereas ThP of CG patients had not changed significantly (89±2 W, $p>0.05$). Thus, following CRP, ThP was significantly greater for PhTG patients than for CG patients ($p<0.001$).

BP, HR and RPP of the two groups at MxP of the CE-test and at the 5th minute following its termination were similar before the CRP (Table 2). After one year, this was no longer the case. At MxP, patients SBP of PhTG patients had decreased by 4.6% ($p<0.05$), DBP by 4.4% ($p<0.05$), HR by 4.7% ($p<0.05$), and RPP by 8.2% ($p<0.01$) from initial, pre-CPR values. In contrast, these variables did not change significantly in CG patients. Between group comparisons of these variables after one year showed that DBP, HR, and RPP, were significantly reduced in the PhTG (Table 2). At the 5th minute after termination of the CT-test, DBP of CG patients was significantly higher than recorded one year earlier, whereas in PhTG patients values of all C-test variables were similar to those recorded pre-CRP. Between group comparisons found DBP and HR to be significantly less in the PhTG at one year.

Dynamics of EchoCG indicators of LV function

At the beginning of the study, the values of LV EDV and ESV were significantly higher in PhTG patients than in CG patients: EDV, 142±3 ml vs 132±3 ml ($p<0.05$); ESV, 63±2 ml vs 56±2 ml ($p<0.02$). Other EchoCG parameters did not significantly between the groups. After one year of CRP, PhTG patients had improved LV function. Their LV ESV significantly decreased from 63±2 ml to 55±2 ml (7.8%, $p<0.001$); LV EF increased from 56±1% to 60±1% (7.6%, $p<0.001$); in control group there were no changes in LV EF, 58±8%; systolic volume increased from 79±2 ml to 81±2 ml (5.1%, $p<0.01$). These changes were accompanied by an increased cardiac index (5.9%, $p<0.001$) and decreased TPR (6.4%, $p<0.05$). LA size remained stable (3.8±0.1 cm at baseline and 3.8±0.1 at one year, $p>0.05$).

After one year of observation, EchoCG parameters of CG patients either did not change or worsened: LA size increased (4.3%, $p<0.05$). Between group comparisons at one year found significantly favorable improvements in LV EDV, LV ESV, LV EF, size of LA and TPR of PhTG patients. The E/A ratio did not differ between the groups either at baseline (1.0±0.1 and 1.2±0.1, $p>0.05$) or at one year (0.9±0.1 and 1.1±0.1, $p>0.05$).

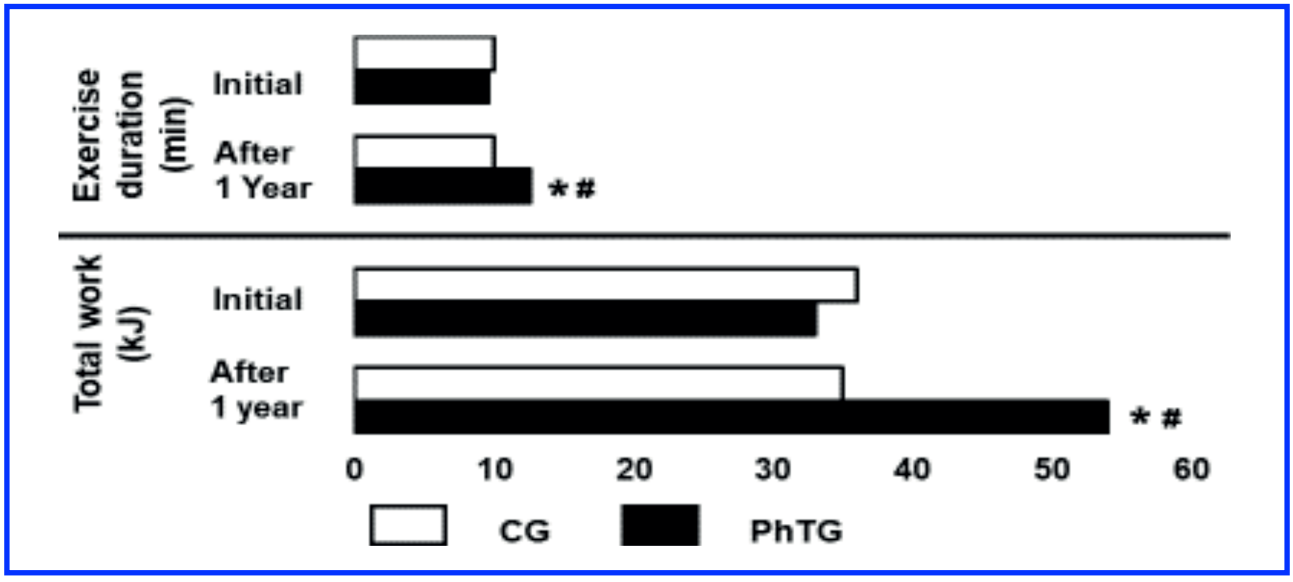

Figure 1. *Exercise duration and total work performed during a cycle ergometry test performed prior to the study (Initial) and at conclusion of the study (After 1 year). CG, patients in the untreated control group; PhTG, patients in the physically trained group. *$p<0.001$vs Initial value $^{\#}p<0.001$ vs respective between group value.*

Table 2. *Initial and one-year, post-rehabilitation CE-test values of systemic arterial blood pressure, heart rate, and rate pressure product of PhTG and control patients recorded at maximum power of the CE test and at 5 min after cessation of the CE-test.*

Variable	Groups						PhTG *vs* CG	
	PhTG (n=100)			CG (n=98)			Initial	1 year
	Initial	1 year	p	Before	1 year	p	p	p
At MxP of CE-test								
SBP, mmHg	173±3	166±3	<0.05	175±3	173±3	NS	NS	NS
DBP, mm Hg	97±1	93±1	<0.05	96±1	97±1	NS	NS	<0.02
HR, beats/min	113±2	109±2	<0.05	116±2	116±2	NS	NS	<0.05
RPP, units	199±6	184±6	<0.01	206±6	203±6	NS	NS	<0.02
5 min after cessation of CE-test								
SBP, mm Hg	136±2	134±2	NS	138±2	137±2	NS	NS	NS
DBP, mm Hg	87±1	84±1	NS	84±2	87±1	<0.01	NS	<0.01
HR, beats/min	83±2	82±2	NS	86±2	88±1	NS	NS	<0.05
RPP, units	115±3	110±3	NS	120±3	120±3	NS	NS	<0.05

Values are means±SE; PhTG, patients in the physical trained group; CG, patients in the untrained, control group; CE-test, cycle ergometry test; MxP, maximal CT-test power; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; RPP, rate pressure product; NS, p ≥0.05.

Dynamics of 24-h ECG monitoring and frequency of angina episodes

Data of 24-hour ECG monitoring are presented in Table 3. After one year of CRP, the average number of episodes of ischemic ST depression during monitoring in PhTG patients decreased by 51.3% (p <0.01), the incidence of painless ischemia decreased by 55.6% (p <0.05) and the episodes of silent myocardial ischemia by 46.8% (p <0.01). No positive changes were observed in the CG patients. Numbers of supraventricular and ventricular extrasystoles were not affected by CRP.

The diaries of the PhTG patients showed that during one year of CRP, the average frequency of angina pectoris attacks decreased by 47.6% (p <0.001), and the average number of NTG tablets taken by patients to treat attacks of angina pectoris decreased by 53.8% (p <0.01) (Figure 2). There were no changes in the number of angina attacks or the amount of NTG taken by CG patients. Also, among the PhTG patients, the number of taken long-acting nitrates significantly decreased by 25% (p <0.001).

Dynamics of lipid transport indicators

At one year of the CRP, an antiatherogenic response in the PhTG patients was evident with an increased serum HDL-C (13.1%, p <0.001). There was a significant decrease in TC/HDL-C (-9.7%, p <0.01) and in LDL-C/HDL-C (-11.2%, p <0.05). In contrast, atherogenicity indices of CG patients increased: TC/HDL-C (13.1%, p <0.05) and LDL-C/HDL-C (16.7%, p <0.05). In the CG group, HDL-C remained unchanged (1.07±0.03 mmol/l at baseline and 1.06±0.03 mmol/l in one year, p>0.05).

Dynamics of PA. The daily PA of the CG patients scored 53 ± 1 points on the QPHA-23 + questionnaire, initially and 52 ± 3 points one year later. In contrast, PhTG patients increased their PA score by 13.1% (p <0.01) to 67.9 points. Thus, during the CRP, the PhTG patients transitioned from low to moderate daily PA.[21]

Clinical characteristics and disease outcome

Positive changes of EchoCG indicators of LV function, the increase in exercise capacity, and daily PA in PhTG patients were reflected in their clinical improvement. The BMI in the main group decreased to 26.9±3.2 (decrease by 3.1%) and there were no changes in the control group -27.9±3.5. The number of revascularizations (PCI or CABG) in current year in the main group was 1 (1.6%) and 6 (5.3%), in the control group. Also, their quality of life significantly improved (30.8%, p <0.001) as compared with no change in the CG patients. The PhTG patients reported an average 1.9 days of temporary disability per person during the year compared to 4.9 days per person for CG patients (p <0.05).

Table 3. *Initial and one-year, post-rehabilitation results of 24-hour ECG monitoring.*

Variable	Groups						p PhTG *vs* CG	
	PhTG			CG				
	Initialn =102	1 year n=100	p	Initial n=104	1 year n=98	p	Initial	1 year
Duration of ECG monitoring, h	22.3±0.3	22.0±0.3	NS	23.0±0.1	22.6±0.2	NS	NS	NS
Supraventricular extrasystoles, n	118±4	70±4	Eq	105±5	89±5	Eq	Eq	Eq
Ventricular extrasystoles, n	256±7	231±6	Eq	272±7	205±7	Eq	Eq	Eq
Ischemic ST depression, n episodes	4.4±0.6	2.1±0.3	<0.01	3.5±0.5	3.4±0.6	NS	NS	<0.05
Silent myocardial ischemia, n episodes	3.5±0.5	1.9±0.3	<0.01	2.5±0.4	3.2±0.6	NS	NS	<0.02
Painful myocardial ischemia, n episodes	0.9±0.2	0.4±0.1	<0.05	0.9±0.1	0.5±0.1	NS	NS	NS

Values are means±SE; PhTG, patients in the physical trained group; CG, patients in the untrained, control group; NS = p ≥0.05.

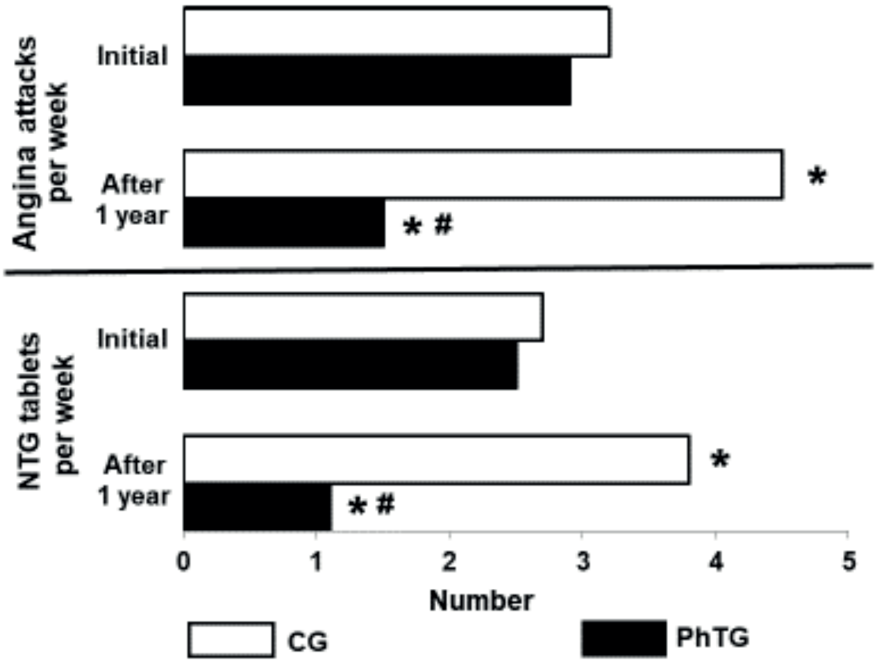

Figure 2. *The number of angina attacks and nitroglycerin tablets taken by patients per week prior to the study (Initial) and at conclusion of the study (After 1 year). CG, patients in the untreated control group; PhTG, patients in the physically trained group. *p< 0.05 within group comparison. #p< 0.001 between-group comparison.*

During the study, 12 of the PhTG and 29 of the CG had cardiovascular events that required medical evaluation ($p < 0.05$). All of the patients in the PhTG with cardiovascular events were able to meet the criteria of at least 85% of scheduled exercise sessions and were also able to participate in the post-study evaluations. Likewise, all CG patients who experienced cardiovascular events participated in the post-study evaluations.

Discussion

The study demonstrated positive, complex benefits of a one-year cardiac rehabilitation program that included systematic exercise training and standard drug therapy for post-AMI patients with AH. Not only were cardiac variables improved by this program, there were concurrent reductions in these patients' arterial blood pressure compared to control patients treated with standard drug therapy alone. In addition, there was a positive influence of exercise training in restraining the pathological remodeling of myocardium post-AMI.[22] These new findings in patients with AH extend our earlier research that demonstrated benefits of a comprehensive rehabilitation program with systematic exercise training.[23] As in our previously described "School for patients after AMI",[23] patients in the present study were highly motivated; no patients withdrew from the study for non-medical reasons. Furthermore, safety of the present CRP was evident from the very low number of patients who withdrew from the study for medical reasons. Although these numbers were small for both PhTG and control group, significantly more patients withdrew from the control group than from the exercise trained group.

Published meta-analyses data of randomized controlled clinical trials confirm the ability of moderate dynamic exercise training to decrease BP levels, both systolic (-3.4 to -7.4 mm Hg) and diastolic (-2.4 to -5.8 mm Hg) pressures.[24, 25] In the current study, the one-year exercise training program decreased office SBP by 4 mmHg and DBP by 3 mmHg. BPs at MxP of the CE-test were also reduced. Meta-analysis of epidemiological and clinical data has shown that a decline in SBP or DBP as small as 2 mmHg is associated with a marked reduction in mortality due to ischemic heart disease (-7%) and to stroke (-10%).[26]

A decrease in TPR, which occurred the PhTG patients, is the primary mechanism that decreases BP after systematic exercise training.[27] This is facilitated by positive neurohumoral, vascular and structural adaptive changes in the background of exercise training. This decrease in systemic vasoconstriction reflects neutralization of excessive sympathetic stimulation of vascular smooth muscle.[28] Also, systematic exercise training improved endothelium functional activity by increasing the release of the endothelial vasodilator, Nitric Oxide (NO), and reducing release of the vasoconstrictive agent, endothelin-1.[28-30] This vascular remodeling induced by systematic exercise training in AH patients likely contributed to antihypertensive effect observed in this study.

PhTG patients had fewer attacks of angina during the study and had more favorable findings on 24-hr ECG monitoring post-test. These benefits may have accrued from recognized impacts of moderate, systematic exercise training that include of decrease of HR and BP, improvement of the gas transport, and restoration of coronary endothelial function and the resulting expansion of the coronary reserve.[31] Exercise training stimulates coronary angiogenesis and increases collateralization.[31-34] In this regard, exercise training increases Vascular Endothelial Growth Factor (VEGF) and the expression of its receptors.[35]

Moderate dynamic exercise has previously been demonstrated to increase HDL-C and its apoprotein (apo) AI.[36] The present study extends these antiatherogenic findings to hypertensive, exercising patients recovering from AMI. With aerobic exercise, the activity of peripheral lipoprotein lipase increases, which leads to an increased rate of apo B-containing lipoproteins utilization and activation of the cholesterol reverse transport mechanism, so HDL-C and its apo AI are increased. HDL is a modulating factor for the activity of endothelial NO synthase, the main source of NO.[37]

Cardiac rehabilitation programs based on systematic exercise training results have been shown to significantly reduce risk of recurrent MI, cardiovascular mortality and total mortality.[38] These findings are consistent with observations that long-term exercise attenuates the progression of coronary artery disease and may even reduce atherosclerosis lesions.[39,40] The lower mortality of PhTG patients is consistent with these reports.

The current study showed that the CRP reduced the number days of temporary disability. This benefits not only the patient, but the economy as well. The lesser incidence of cardiovascular events among the PhTG is consistent with previously described clinical benefits of CRP in non-AH subjects.[41,42]

Finally, it should be recognized that involving AMI patients in a cardiac rehabilitation program is an important factor in increasing their adherence to other therapy.[43] The continued contact with medical personnel motivates patients to correct their lifestyle, to take prescribed medication, and to continue participation in the rehabilitation program.

List of abbreviations

CRP, cardiac rehabilitation program
MI, myocardial infarction
AMI, acute myocardial infarction
AH, arterial hypertension
PhT, physical training
PhTG, physically trained group
CG, control group
MxP, maximal power
CE-test, cycle ergometry test
ET, exercise time
ThP, threshold power
ToW, total work
RPP, rate-pressure product
BAB, beta-adrenoblockers
ATII, angiotensin II
IHD, ischemic heart disease

CA, coronary artery
BP, blood pressure
LDL, low density lipoprotein
HR, heart rate
BMI, body mass index
LA -left atrium
EDV, end-diastolic volume
ESV, end-systolic volume
IST, interventricular septum thickness
EF, ejection fraction
VE, ventricular ejection
TPR, total peripheral resistance
TC, total cholesterol
TG, triglycerides
HDL-C, high-density lipoprotein cholesterol
VLDL, very low-density lipoproteins
PA, physical activity
QoL, quality of life
NTG, nitroglycerin
SE, standard error
HF, heart failure
STEMI, ST-elevation myocardial infarction
NSTEMI, non-ST-elevation myocardial infarction
SBP, systolic blood pressure
DBP, diastolic blood pressure
EchoCG, echocardiogram
CABG, coronary artery bypass grafting
PCI, percutaneous coronary intervention
NO, nitric oxide
VEGF, vascular endothelial growth factor

Contribution

DMA conceived the study, designed the study and were in charge of overall direction and planning; MGB contributed to the design and implementation of the research, to the analysis of the results and to the writing of the manuscript, carried out the experiments; NPL carried out the experiments, contributed to the design and implementation of the research, to the analysis of the results and to the writing of the manuscript; HFD, EBM processed the experimental data, performed the analysis, the results and worked on the manuscript, authors contributed to the final version of the manuscript; SVL wrote the manuscript with input from all authors. All authors discussed the results and contributed to the final manuscript.

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Conflict of interest

The authors declare no conflict of interest.

Ethics approval and consent to participate

The study was carried out in accordance with Good Clinical Practice standards and with the Helsinki Declaration principles. Written informed consent was obtained from all participants of this study. Ethics Committee of the National Medical Research Center for Therapy and Preventive Medicine, Moscow, Russian Federation, approved the Study Protocol on March 19, 2015 (Protocol #3).

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