

## Physical rehabilitation after acute myocardial infarction: focus on body weight

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**Aim.** To study the effectiveness of 1-year exercise training (ET) after acute myocardial infarction (AMI) during outpatient cardiac rehabilitation in patients with different body mass index (BMI).

**Material and methods.** The study included 312 patients after AMI, who were randomized into four groups depending on BMI: patients who used ET program with BMI <30 kg/m<sup>2</sup> (group 1 (n=78)) and BMI ≥30 kg/m<sup>2</sup> (group 2 (n=78)); patients who did not use ET program with BMI <30 kg/m<sup>2</sup> (group 3 (n=78)) and BMI ≥30 kg/m<sup>2</sup> (group 4 (n=78)). ET of moderate intensity (60% of the threshold value) was carried out 3 times a week for a year.

**Results.** In patients with obesity, ET was associated with decrease of blood pressure by 3,3/3,6% (p<0,01 for each) and BMI by 7,7% (p<0,001), while there was an increase by 4,2/3,6% (p<0,05 for each) and 2,1% (p<0,05), respectively, in obese patients without ET. In patients without obesity, ET was associated only with BMI decrease by 3,3% (p<0,01), while in patients without obesity and ET it did not change. Daily physical activity after ET increased regardless of BMI, and without ET it decreased in obese patients. ET was associated with the increase of duration and intensity of training in non-obese patients by 39,2% (p<0,001) and 47,1% (p<0,001), respectively; in obese patients — by 23,8% (p<0,001) and 26,5% (p<0,001), respectively. In control groups it has not changed. After ET, with any BMI, the levels of low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG) decreased, and the high-density lipoprotein-cholesterol (HDL-C) increased. In the control groups, the con-

centration of TG increased, and with obesity there was also an increase in LDL-C and a decrease in HDL-C. Against the background of ET, the fibrinogen values decreased with any BMI, in contrast to the control groups. After 1-year ET, number of cardiovascular events (CVE) significantly decreased in non-obese patients by 37,5% (p<0,05) and in obese ones by 28,6% (p<0,05).

**Conclusion.** Long-term aerobic ET in patients with any BMI reduced cardiovascular risk factors and the risk of CVE. At the same time, with concomitant obesity, the maximum effect of cardiac rehabilitation was not achieved, which confirms the importance of controlling BMI in patients after AMI.

**Key words:** myocardial infarction, cardiac rehabilitation, exercise training, physical activity, obesity, body weight.

**Relationships and Activities:** none.

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Cardiovascular diseases (CVD) is the leading cause of mortality in all countries of the world, including Russia. The development of CVD is closely linked to behavioral risk factors (RF), such as smoking, unhealthy diets, low physical activity, hypertension (HTN) and obesity. In all countries, the obesity epidemic has been observed over the past decade [1]. Among patients involved in cardiac rehabilitation, approximately 80% are overweight or obese [2]. The average body mass index (BMI) of rehabilitated patients over 10 years increased from 28,5 kg/m<sup>2</sup> to 30,1 kg/m<sup>2</sup> [3].

High efficiency of the cardiac rehabilitation programs based on systematic exercise training (ET) of moderate intensity has been proven to modulate cardiovascular RF, improve the quality of life (QOL), and reduce cardiovascular and all-cause mortality in patients with coronary artery disease (CAD) [4]. The beneficial effect of cardiac rehabilitation on the disease outcomes and the survival of patients with acute myocardial infarction (AMI) remains urgent in the period of active myocardial revascularization and therapy with statins and antiplatelet agents [5].

The negative effects of obesity on the CAD progression and the mortality risk after AMI have been demonstrated in studies [6, 7]. The question of the effectiveness of cardiac rehabilitation programs and systematic ET in obese patients remains open. This is especially true in the context of the obesity paradox. According to number of studies, patients with history of AMI and/or myocardial revascularization, suffering from peripheral atherosclerosis or heart failure (HF), had a U-shaped relationship between BMI and all-cause mortality risk (with a nadir in the range of BMI 26,5–35 kg/m<sup>2</sup>) [8–10]. The protective effect of large BMI values is obviously more often manifested in elderly patients with severe CVD and undernutrition [10].

The relationship of obesity with the prognosis in obesity paradox is affected, for example, by physical activity. According to the European Prospective Investigation into Cancer and Nutrition Study (EPIC) with 334,161 men and women (12,4-year follow-up), the negative impact of physical inactivity on patient mortality was more (almost 2 times) than high BMI ( $\geq 30$  kg/m<sup>2</sup>) [11]. It should be noted that if patients after AMI and cardiac surgery are not involved in cardiac rehabilitation programs, the percentage of physically active ones among them remains low [12].

To aim was to study the effectiveness of 1-year ET after AMI during outpatient cardiac rehabilitation in patients with different BMI.

## Material and methods

The study included 312 patients after AMI ( $>3$  weeks) and percutaneous coronary interventions (PCI) at the age of  $<60$  years for men and  $<55$  years for women (mean age  $52,1 \pm 3,9$  years). All patients signed informed consent. There were following exclusion criteria: inadequately controlled HTN, aortic or left ventricular (LV) aneurysm with thrombosis, serious arrhythmias, NYHA class III–IV HF, BMI  $\geq 40$  kg/m<sup>2</sup>, moderate/severe diabetes and other severe comorbidities. This study was performed in accordance with the Helsinki declaration and Good Clinical Practice standards. The ethics committee approved this study.

Patients received standard drug therapy. They were randomized into four groups depending on BMI: experimental — patients who used ET program with BMI  $<30$  kg/m<sup>2</sup> (group 1 (n=78)) and BMI  $\geq 30$  kg/m<sup>2</sup> (group 2 (n=78)); control — patients who did not use ET program with BMI  $<30$  kg/m<sup>2</sup> (group 3 (n=78)) and BMI  $\geq 30$  kg/m<sup>2</sup> (group 4 (n=78)). To dynamically analyze certain parameters during rehabilitation, patients of the experimental group without obesity were divided into two subgroups depending on BMI: BMI  $<25$  kg/m<sup>2</sup> (n=32) and 25,0–29,9 kg/m<sup>2</sup> (n=46). The follow-up period lasted 12 months.

**Physical rehabilitation program** included a set of gymnastic exercises of moderate intensity (60% of the threshold value according to cycle ergometer test (CET) by Aronov DM). There were group exercise classes (up to 10–12 people) lasting 60 minutes 3 times/week.

Clinical examination included collecting medical history, physical exam, measurement of blood pressure (BP), heart rate (HR) and BMI. Resting electrocardiography (ECG) was performed. CET using cycle ergometer (General Electric, USA) was performed until the generally accepted clinical or ECG stopping criteria (WHO, 1973, Aronov DM, 1995), or submaximal HR (Andersen KL, 1971) with the analysis of the duration (min) and power (W) of physical activity, resting and maximum HR (max) during CET (beats/min), resting double product (DP, CU) and DPmax. (Heart rate  $\times$  systolic BP (SBP)/100), HR (beats  $\times$  min) and DP (CU  $\times$  min) growth during CET, the total physical work performed (A, kJ). Echocardiography was performed according to a standard method (Agilent, USA) with an assessment of the maximum anterior-posterior dimension of left atrium (LA, cm), LV end-diastolic dimension (EDD) and end-systolic dimension (ESD), LV ejection fraction (EF) by the Simpson's biplane method.

We determined levels of blood lipids (mmol/L): total cholesterol (TC), triglycerides (TG), high

Table 1

## Characteristics of groups at the beginning of the study

| Parameter                    | Experimental groups           |                               | Control groups                |                               | P                     |                       |
|------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-----------------------|-----------------------|
|                              | Group 1<br>Obesity-<br>(n=78) | Group 2<br>Obesity+<br>(n=78) | Group 3<br>Obesity-<br>(n=78) | Group 4<br>Obesity+<br>(n=78) | Group 1 vs<br>Group 3 | Group 2 vs<br>Group 4 |
| Age, years (M±m)             | 51,9±7,9                      | 51,7±6,8                      | 52,2±7,2                      | 52,6±6,7                      | NS                    | NS                    |
| Men/Women, n (%)             | 73 (93,6)/5 (6,4)             | 75 (96,2)/3 (3,8)             | 74 (94,9)/4 (5,1)             | 73 (93,6)/5 (6,4)             | NS                    | NS                    |
| History of AMI, n (%)        | 40 (51,3)                     | 42 (53,8)                     | 41 (52,6)                     | 42 (53,8)                     | NS                    | NS                    |
| Hypertension, n (%)          | 35 (44,9)                     | 52 (66,7)*                    | 37 (47,4)                     | 51 (65,4)*                    | NS                    | NS                    |
| Class I-II HF, n (%)         | 35 (44,9)                     | 31 (39,7)                     | 35 (44,9)                     | 29 (37,2)                     | NS                    | NS                    |
| Type 2 diabetes, n (%)       | 3 (3,8)                       | 11 (14,1)*                    | 2 (2,6)                       | 10 (12,8)*                    | NS                    | NS                    |
| SBP, mm Hg (M±m)             | 124,2±17,1                    | 129,1±16,9                    | 123,5±15,3                    | 127,4±16,8                    | NS                    | NS                    |
| DBP, mm Hg (M±m)             | 82,1±11,0                     | 85,3±10,3                     | 80,2±9,1                      | 84,2±10,4                     | NS                    | NS                    |
| Heart rate, bpm (M±m)        | 73,3±8,1                      | 75,5±9,2                      | 73,8±9,7                      | 76,4±10,7                     | NS                    | NS                    |
| BMI, kg/m <sup>2</sup> (M±m) | 26,6±1,6                      | 32,2±1,6*                     | 26,9±2,1                      | 32,4±1,8*                     | NS                    | NS                    |
| LVEF, % (M±m)                | 57,5±8,8                      | 55,6±9,4                      | 57,3±8,7                      | 56,7±9,0                      | NS                    | NS                    |

**Note:** NS — not significant, \* —  $p < 0,05$  — significance of differences between the groups “group 1 — group 2” and “group 3 — group 4”.

**Abbreviation:** DBP — diastolic blood pressure, BMI — body mass index, LVEF — left ventricular ejection fraction, AMI — acute myocardial infarction, SBP — systolic blood pressure, HF — heart failure.

density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) by Friedwald formula (WT Friedwald, et al. (1972)) with  $TG \leq 4,5$  mmol/L. Using ACL coagulation analyzer (Instrumentation Laboratory, Italy), concentration of fibrinogen (g/L) and prothrombin index (%) was identified. Glucose levels (mmol/L) were assessed on an Airon-200 analyzer (Italy).

QOL was assessed was evaluated using questionnaire of Aronov DM and Zaitsev VP (1982) [13]. Daily physical activity was assessed using the ODA23+ questionnaire: <62 points — low physical activity; 62–84 points — moderate activity; >84 points — high activity. Patients kept diaries recording angina episodes.

We analyzed the total number of temporary disability days during the year. The primary endpoint included sudden cardiac death, recurrent AMI, and stroke.

**Statistics.** Analysis of the results was performed using the Statistical Analysis System (SAS, version 6.12). The mean and standard error (M±m) were calculated. For indicators measured on a nominal or ordinal scale, the frequency of detecting various gradations in percent was estimated. The significance of differences between groups was evaluated by Student's t-test for independent samples or paired t-test for dependent variables. For comparative analysis of more than two groups, analysis

of variance (ANOVA) was used. Different proportions were compared using the chi-squared test. Differences were considered significant at  $p < 0,05$ .

## Results

Obese patients in both groups (groups 2 and 4) were initially more likely to suffer from HTN and type 2 diabetes than non-obese patients (groups 1 and 3) (Table 1). There were no significant differences between the experimental and control groups without (groups 1-3) and with obesity (groups 2-4).

**Changes of cardiovascular RF.** Office BP in patients prior to inclusion in the study were within <140/90 mm Hg. After 12 months in non-obese patients of the experimental and control groups, BP levels did not change. In obese patients of the experimental group, SBP significantly decreased by 3,3% ( $p < 0,01$ ) and diastolic BP (DBP) by 3,6% ( $p < 0,01$ ), while patients of the control group, on the contrary, had increase by 4,2% ( $p < 0,05$ ) and 3,6% ( $p < 0,05$ ), respectively.

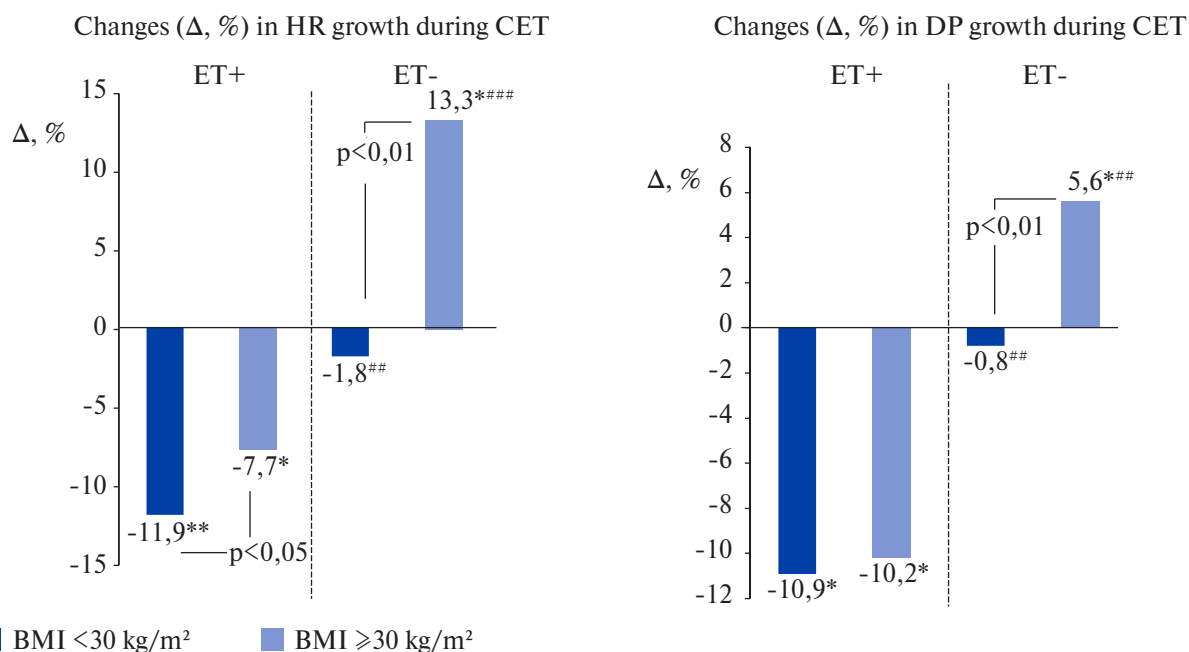
With ET, a significant decrease in BMI was observed in patients with (by 7,7%,  $p < 0,001$ ) and without obesity (by 3,3%,  $p < 0,01$ ). Moreover, BMI did not change in trained patients with normal body weight ( $BMI < 25,0$  kg/m<sup>2</sup>) and significantly decreased by 2,9% (from  $27,1 \pm 1,4$  to  $26,3 \pm 1,5$  kg/m<sup>2</sup>,  $p < 0,001$ ) in trained patients with

Table 2

**Changes of exercise tolerance before and after 1-year rehabilitation program  
in patients after AMI with different BMI**

| Parameter<br>(M±m)                    | Точка<br>исследования | Experimental groups           |                               |       | Control groups                |                               |    |
|---------------------------------------|-----------------------|-------------------------------|-------------------------------|-------|-------------------------------|-------------------------------|----|
|                                       |                       | Group 1<br>Obesity-<br>(n=78) | Group 1<br>Obesity-<br>(n=78) | P     | Group 3<br>Obesity-<br>(n=78) | Group 4<br>Obesity+<br>(n=78) | P  |
| Physical activity,<br>points          | Initially             | 55,1±9,4                      | 52,8±10,2                     | NS    | 53,2±11,4                     | 51,5±11,3                     | NS |
|                                       | 12 months after       | 72,9±8,2                      | 62,2±7,1                      | <0,05 | 57,1±9,5**                    | 48,1±11,8*                    | NS |
| P                                     |                       | <0,001                        | <0,05                         |       | NS                            | <0,05                         |    |
| Walking, km/day                       | Initially             | 4,0±1,8                       | 3,6±1,3                       | NS    | 3,8±1,5                       | 3,5±1,3                       | NS |
|                                       | 12 months after       | 5,8±1,1                       | 4,4±1,3                       | <0,05 | 3,8±0,9*                      | 2,8±1,1*                      | NS |
| P                                     |                       | <0,01                         | <0,05                         |       | NS                            | <0,05                         |    |
| Power of physical<br>activity, W      | Initially             | 76,3±18,1                     | 83,4±18,7                     | NS    | 80,2±20,1                     | 87,1±17,9                     | NS |
|                                       | 12 months after       | 112,4±16,2                    | 105,2±20,3                    | NS    | 85±18,9*                      | 87±19,8*                      | NS |
| P                                     |                       | <0,001                        | <0,001                        |       | NS                            | NS                            |    |
| Duration of physical<br>activity, min | Initially             | 9,4±2,9                       | 10,2±3,3                      | NS    | 9,8±2,9                       | 10,6±3,1                      | NS |
|                                       | 12 months after       | 13,1±3,9                      | 12,6±3,1                      | NS    | 10,4±3,9**                    | 10,6±3,3*                     | NS |
| P                                     |                       | <0,001                        | <0,001                        |       | NS                            | NS                            |    |

**Note:** NS — not significant, \* —  $p < 0,01$  — significance of differences between the groups “group 1 — group 3” and “group 2 — group 4”.

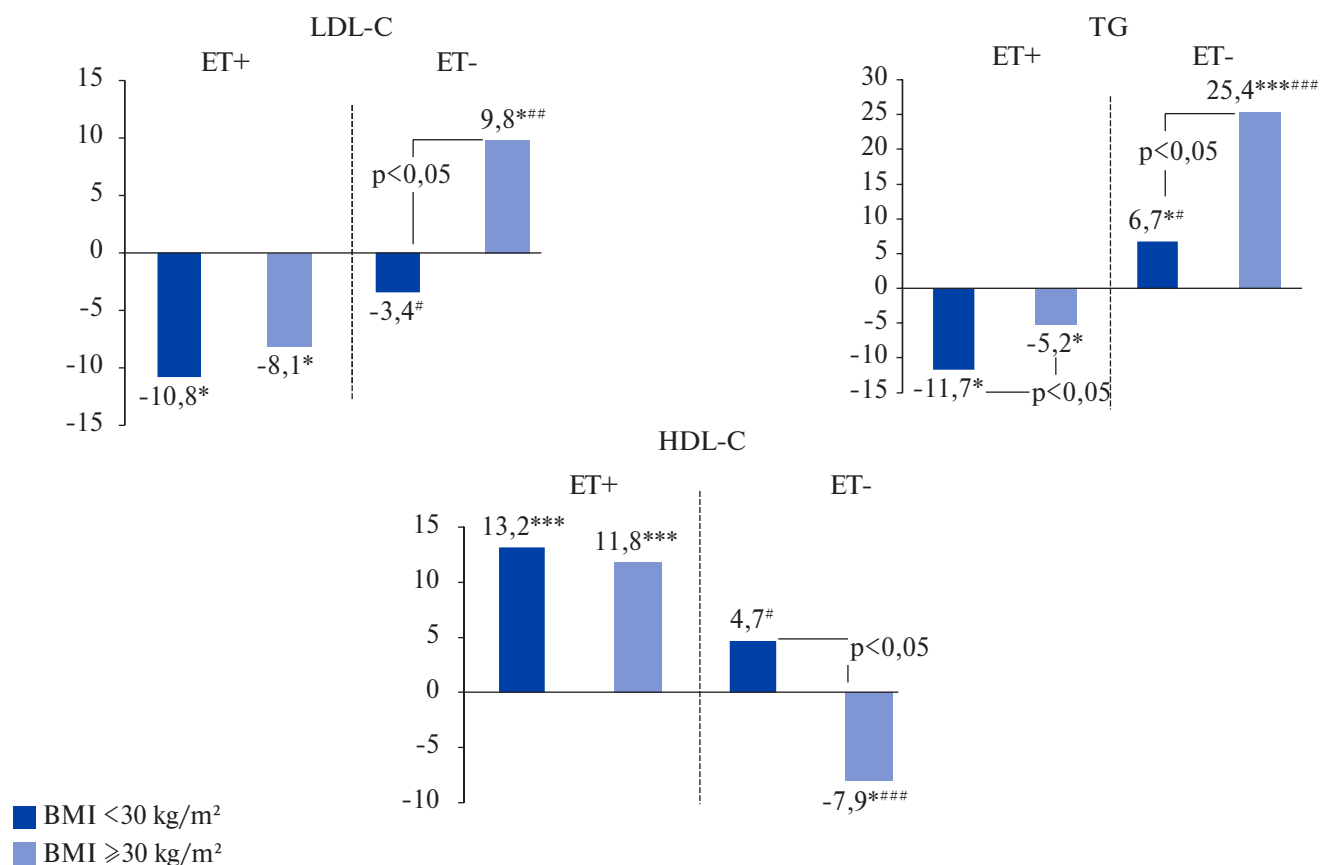


**Figure 1.** Changes (Δ, %) in HR and DP growth during CET in patients after AMI with/without 1-year regular ET with different BMI.

**Note:** \* —  $P < 0,05$ , \*\* —  $P < 0,01$  — in relation to initial value, # —  $P < 0,05$ , ## —  $P < 0,01$ , \*\*\* —  $P < 0,001$  — comparison of the experimental (ET+) group and the control group (ET-) among patients without obesity (BMI < 30 kg/m<sup>2</sup>) or with obesity (BMI ≥ 30 kg/m<sup>2</sup>);  $P < 0,05$ ,  $P < 0,01$  — intragroup comparison of non-obese (BMI < 30 kg/m<sup>2</sup>) and obese patients (BMI ≥ 30 kg/m<sup>2</sup>).

overweight (BMI 25,0-29,9 kg/m<sup>2</sup>). In the control groups, BMI did not change in patients without obesity and significantly increased (by 2,1%,  $p < 0,05$ , to 33,1±2,2 kg/m<sup>2</sup>) in obese patients.

Initially, all patients had a low level of daily physical activity (Table 2). After 12 months of ET, it increased to an average level in obese patients (by 16,5%,  $p < 0,05$ ) and to a greater extent in non-



**Figure 2.** Changes ( $\Delta$ , %) in blood lipids and lipoproteins in patients after AMI with/without 1-year regular ET with different BMI.

**Note:** \* —  $P < 0,05$ , \*\* —  $P < 0,01$  — in relation to initial value, # —  $P < 0,05$ , ## —  $P < 0,01$ , ### —  $P < 0,001$  — comparison of the experimental (ET+) group and the control group (ET-) among patients without obesity (BMI < 30 kg/m<sup>2</sup>) or with obesity (BMI ≥ 30 kg/m<sup>2</sup>);  $P < 0,05$ ,  $P < 0,01$  — intragroup comparison of non-obese (BMI < 30 kg/m<sup>2</sup>) and obese patients (BMI ≥ 30 kg/m<sup>2</sup>).

obese patients (by 32,3%,  $p < 0,001$ ). In control groups with obese patients, the level of physical activity did not change, while in obese patients it significantly decreased (6,1%,  $p < 0,05$ ). The distance traveled increased in non-obese patients by 1,8 km ( $p < 0,01$ ) and to a lesser extent in obese patients — by 0,8 km ( $p < 0,05$ ), whereas in control groups it did not change in non-obese patients and significantly decreased by 0,7 km ( $p < 0,05$ ) in obese patients.

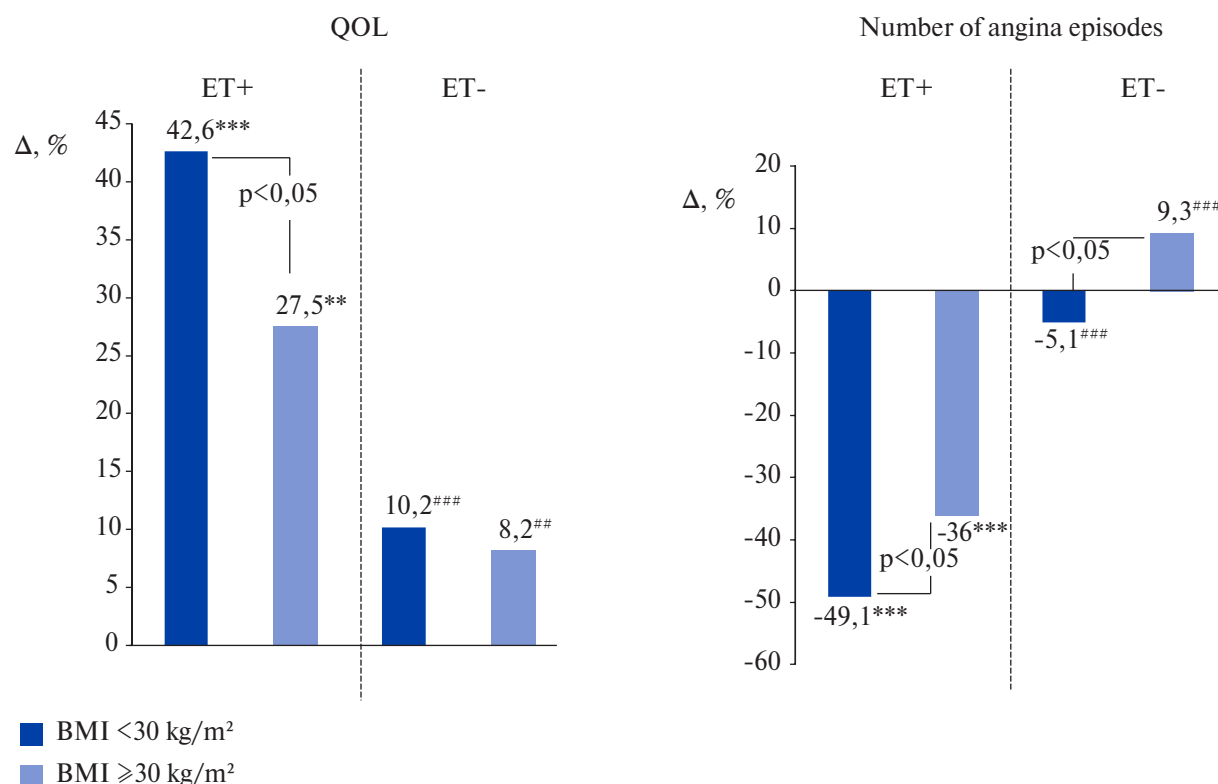
**Exercise tolerance changes.** Patients included in the study did not initially differ in exercise tolerance having moderate results on CET (Table 2). After 12 months of ET, patients with both BMI < 30 kg/m<sup>2</sup> and BMI ≥ 30 kg/m<sup>2</sup> had a significant increase in exercise tolerance: power of physical activity increased by 47,1% ( $p < 0,001$ ) and 26,5% ( $p < 0,001$ ), respectively ( $p < 0,005$  between groups), and the duration by 39,2% ( $p < 0,001$ ) and 23,8% ( $p < 0,001$ ;  $p < 0,01$  between groups). This was accompanied by a significant increase in the total physical work in non-obese patients by 63,3%

( $p < 0,001$ ) and in obese ones by 46,8% ( $p < 0,001$ ;  $p < 0,05$  between groups).

There was a decrease in average HR and DP growth during CET (Figure 1). Load threshold on CET was increased in non-obese patients by 13,7% ( $p < 0,05$ ) and obese ones by 10,6% ( $p < 0,05$ ) without significant differences between them.

In untrained patients without obesity, the studied indicators of exercise tolerance did not change (Figure 1). However, obese patients, on the contrary, needed more oxygen and an increase in hemodynamic load (HR and DP growth during CET) to achieve initial values.

**Changes of echocardiography.** No initial differences in echocardiographic parameters between groups were found. With 1-year ET, there was a small but significant decrease in LV ESD in patients without obesity by 3,9% ( $p < 0,05$ ) and with obesity by 3,2% ( $p < 0,05$ ). LVEF increased by 7,3% ( $p < 0,01$ ) and 5,7% ( $p < 0,01$ ), respectively. EDD and LA dimension did not change. In trained patients with a BMI < 25 kg/m<sup>2</sup>, LVEF increased by



**Figure 3.** Changes (Δ, %) in QOL and number of angina episodes in patients after AMI with/without 1-year regular ET with different BMI. **Note:** \* —  $P < 0,05$ , \*\* —  $P < 0,01$  — in relation to initial value, # —  $P < 0,05$ , ## —  $P < 0,01$ , ### —  $P < 0,001$  — comparison of the experimental (ET+) group and the control group (ET-) among patients without obesity (BMI <30 kg/m<sup>2</sup>) or with obesity (BMI ≥30 kg/m<sup>2</sup>);  $P < 0,05$ ,  $P < 0,01$  — intragroup comparison of non-obese (BMI <30 kg/m<sup>2</sup>) and obese patients (BMI ≥30 kg/m<sup>2</sup>).

7,8% ( $p < 0,05$ ) and with a BMI of 25,0-29,9 kg/m<sup>2</sup> — by 6,7% ( $p < 0,05$ ).

Without ET, patients without and with obesity did not have favorable echocardiographic changes after 12 months. On the contrary, obese patients had a slight increase in LA dimension (by 4,1%,  $p < 0,05$ ).

**Changes of atherothrombogenic parameters.** Blood glucose levels in all groups was within normal limits and did not changed for 12 months. After ET, the fibrinogen concentration significantly decreased: in patients without obesity — by 18,9% (from  $3,7 \pm 1,1$  g/L to  $3,0 \pm 0,4$  g/L,  $p < 0,05$ ) and with obesity — by 21,6% (from  $3,7 \pm 1,3$  g/L to  $2,9 \pm 0,5$  g/L,  $p < 0,05$ ). In control groups, such positive dynamics were not recorded (without obesity — from  $3,7 \pm 1,2$  g/L to  $3,5 \pm 0,8$  g/L; with obesity — from  $3,6 \pm 0,8$  g/L to  $3,8 \pm 0,6$  g/L, respectively). The prothrombin index increased only in the control groups equally: in patients without obesity by 8,1% ( $p < 0,05$ ) and with obesity by 8,6% ( $p < 0,01$ ).

All trained patients had a favorable change in lipid panel: LDL-C and TG levels significantly decreased; HDL-C concentrations increased (Fi-

gure 2). In control group with non-obese patients, only the TG concentration increased moderately, and with obese patients, the levels of LDL-C and TG increased and HDL-C decreased (Figure 2).

**Changes of QOL and clinical state.** In both experimental groups, QOL parameters improved, especially significant in patients with BMI <30 kg/m<sup>2</sup> (Figure 3). In two control groups, QOL was not changed.

In trained patients with any BMI, a decrease in the number of angina episodes was observed, but was more pronounced in non-obese subjects (Figure 3). Moreover, in trained patients with normal body weight, the number of angina episodes decreased by 54,6% ( $p < 0,001$ ) and with overweight — by 43,5% ( $p < 0,001$ ). In the control groups, obese patients had significantly more angina episodes than non-obese ones.

The total number of temporary disability days in trained non-obese patients was 162 (2,1 per 1 patient) and obese ones — 250 (3,2 per 1 patient); in the control groups — 258 (3,3 per 1 patient) and 384 days (4,9 per 1 patient), respectively. With ET, the number of temporary disability days decreased in patients without obesity by 96 days

(by 1,1 per 1 patient,  $p < 0,05$ ) and with obesity by 134 days (by 1,7 per 1 patient,  $p < 0,05$ ). Moreover, patients with obesity had higher total number of temporary disability days than non-obese patients in experimental (by 88 days totally or 1,1 per 1 patient) and control groups (by 126 days totally or 1,6 per 1 patient). The total number of temporary disability days in trained patients with BMI  $< 25$  kg/m<sup>2</sup> was 187 days (2,4 per 1 patient) and with BMI of 25,0-29,9 kg/m<sup>2</sup> — 132 days (1,7 per 1 patient).

After 12-month ET, the primary endpoint in patients without obesity was recorded in 2 cases and with obesity — in 4 cases, and in the control groups, respectively, in 8 and 9 cases. Due to an exacerbation of CAD, there were 8 hospitalizations in trained patients without obesity and 11 in obese patients, and 8 and 12 hospitalizations in non-trained patients, respectively. The total number of all cardiovascular events (CVE) (primary endpoint + hospitalization) was 10 in trained patients without obesity and 15 with obesity; in the control groups, 16 and 21, respectively. With ET, there were reduction of all CVE in patients without obesity (comparison of the experimental and control groups) by 37,5% ( $p < 0,05$ ) and in patients with obesity by 28,6% ( $p < 0,05$ ). The total number of all CVE in trained patients with a BMI  $< 25$  kg/m<sup>2</sup> amounted to 7 events and with a BMI of 25,0-29,9 kg/m<sup>2</sup>, only 3 events.

### Discussion

This randomized clinical trial demonstrated the beneficial effects of 1-year outpatient physical rehabilitation program in patients after AMI/PCI with different body mass. At the same time, the study revealed some features of this results.

The involvement of obese patients in the ET program made it possible to effectively control cardiovascular RF. Thus, in patients with obesity, the levels of SBP (by 4,2 mm Hg) and DBP (by 3,1 mm Hg) significantly decreased against their increase in non-trained patients. This is consistent with a meta-analysis which showed that aerobic ET in sedentary obese people is able to lower SBP by an average of 3,4-7,4 mm Hg, and DBP by 2,4-5,8 mm Hg [14].

Studies with patients involved in ET program demonstrated a decrease in body mass already by 12-24 weeks [2, 15]. In our obese patients, aerobic ET reduced BMI by an average of 7,7%, and in patients without obesity by 3,3%. The extent of ET reduction depends on the load level: the higher this level, the more a decrease in BMI [16]. It has been proven that weight loss during ET in patients with overweight or obesity occurs due to a decrease in

white (visceral) adipose tissue with an increase in lean mass [17]. If patients after AMI are not involved in cardiac rehabilitation programs, it is difficult to effectively control body weight and the opposite effect can be observed, as in the current study.

Patients' participation in ET programs increased their daily physical activity in obese patients by 16,5% and in non-obese patients by 32,3%. In non-trained obese patients, the level of daily physical activity continued to decline. Since sedentary lifestyle adversely affects the survival of patients after AMI, close attention should be paid to increasing the level of daily physical activity [18].

A positive aspect of ET was an increase in exercise tolerance with economical heart operation and lower energy consumption due to a decrease in the hemodynamic response to the load and myocardial oxygen demand (HR and DP growth during CET). Moreover, the training effect in patients without obesity was more pronounced. Clinically, ischemic threshold elevation was expressed by a reduction in angina episodes during daily living activities in obese patients (by 36%) and to a greater extent in patients without obesity (by 49,1%). Regular ET has been shown to improve the coronary endothelial function with an increase in the coronary and collateral flow reserve due to the stimulation of angiogenesis [19]. Such changes can be noticeable after 1-3 months, depending on the intensity of training.

An increase in exercise tolerance, as well as a decrease in the number of angina episodes with regular ET in patients after AMI, is of great importance for improving survival. So, a 15% (1 standard deviation) increase in aerobic fitness was associated with a ~18% lower risk of MI 30 years later [20]. In the present study, the increase in power of physical activity during CET after a 1-year ET was 47,1% in patients without obesity ( $p < 0,001$ ) and 26,5% with obesity ( $p < 0,001$ ). According to the Prospective Observational Longitudinal Registry of Patients With Stable Coronary Artery Disease (CLARIFY) with 20,400 subjects, persistence of angina episodes in patients with CAD can increase the risk of cardiovascular death and nonfatal MI by 66% and AMI by 66% [21].

After a 1-year period without ET, the initial physical performance required a higher oxygen-cost hemodynamic reaction, clinically manifested by the larger number of angina episodes.

Regular 1-year ET is able to restrain LV remodeling processes with increase in its contractile function regardless of the initial body weight, in contrast to the control groups.

Regular ET, causing positive metabolic changes in skeletal muscle and adipose tissue, eliminates the vis-

ceral fat negative effects, which increase cardiometabolic risk. After the completion of a 1-year physical rehabilitation, patients with any BMI had lower levels of LDL-C and TG, and higher HDL-C levels. Such positive effects in lipid metabolism with weight loss and improved exercise tolerance after cardiac rehabilitation/regular ET in obese patients were reported in other studies [2, 22]. A decrease in fibrinogen concentration after ET in patients with any BMI is a favorable fact, since hyperfibrinogenemia is a marker of thrombus formation and inflammation. In contrast, obese patients not involving in cardiac rehabilitation programs had atherothrombogenic changes in blood.

This study included working-age patients. Therefore, the number of temporary disability days was assessed. The involvement of patients after AMI in a rehabilitation program allowed reducing the number of temporary disability days: with obesity ( $\text{BMI} \geq 30 \text{ kg/m}^2$ ) by 49 days; without obesity ( $\text{BMI} < 30 \text{ kg/m}^2$ ) by 90 days.

In patients with obesity of both groups, CVE developed more often than in patients without obesity. Participation of non-obese patients after AMI in a 1-year rehabilitation program reduced the incidence of all CVE (primary endpoint + hospitalization) by 1,6 times, and in patients with

obesity — by 1,4 times. According to study by Doimo S, et al., the participation of patients after AMI/PCI ( $n=839$ ) in cardiac rehabilitation programs led to a 1,7-fold decrease in the risk of CVE + hospitalization compared to non-participants ( $p<0,001$ ) [23].

### Conclusion

Outpatient cardiac rehabilitation program, based on regular ET of moderate intensity, showed a comprehensive positive effect on obese patients after AMI. This proves the need for more active involvement in the rehabilitation programs of obese patients with high atherothrombogenic risk. It has been shown that with concomitant obesity it is not possible to achieve the maximum effect of cardiac rehabilitation. Since concomitant obesity can worsen the rehabilitation prognosis, control of body weight is a priority for patients with CAD. Obese patients should be assigned to a special group, and their rehabilitation should be based on an individually selected loads. Moreover, the duration of rehabilitation programs should be long in order to obtain the best clinical effect.

**Relationships and Activities:** none.

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