The effectiveness of the method of external counterpulsation in patients with chronic stable angina

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Abstract

Aim. To assess the effectiveness of the complex treatment of patients with stable angina pectoris using the method of external counterpulsation (ECP).

Methods. 92 patients with chronic stable angina were included in the comparative analysis, which divided into 2 groups: 57 patients received one course of treatment using the ECP method (main group), 35 patients received only drug treatment (control group). Before and after the therapy, a general clinical examination (including determining the functional class of angina pectoris and the need to use nitroglycerin preparations), coronary angiography, echocardiography, exercise stress test (stress ECG), and quality assessment using the Seattle Angina Questionnaire (SAQ) was performed.

Results. The decrease in the average functional class of angina was more significant in the main group compared to the control group — from 2.28±0.73 to 0.93±0.80 (p <0.05) versus from 2.34±0.73 to 1.83±0.71 (p <0.05). A decrease of at least 1 functional class more often occurred in the main group — 78.9% of patients versus 57.1% (p=0.0258). The use of nitroglycerin sharply decreased after treatment in the group, that use the ECP method (by 51.6%; p=0.002), whereas in the control group, despite a slight decrease (by 22.7%), the changes did not reach statistical significance (p=0.0736). Both groups showed similar dynamics of changes in echocardiography. The differences were obtained only for the dynamics of the ejection fraction of the left ventricle (LVEF), which was greater in the main group — an increase of 4.69±5.56% versus 1.75±5.15% (p=0.0448). The dynamics of all indicators of exercise stress test significantly differ between groups, and if for the main group it is positive, in the control group it is negative. Some indicators of quality of life change insignificant and similar for both groups (for example, on the scale for limiting physical exertion), and on some scales (stability scales for attacks and the frequency of angina attacks), the improvement in the main group is much more significant in comparing with the control group: 30–37 points versus 12–13 points, respectively (p <0.0001). The overall quality of life assessment score improved more in the group, that use the ECP method: +96.10±40.73 versus +45.31±35.06 the control group (p <0.0001).

Conclusion. The method of external counterpulsation (ECP) can be used in clinical practice as part of a comprehensive treatment of patients with stable angina pectoris; it can significantly reduce the class of angina pectoris and the need to use nitroglycerin preparations, improve a number of indicators of echocardiography, electrocardiography with exercise and quality of life indicators.

Keywords: external counterpulsation, ischemic heart disease, chronic stable angina.

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complications are factors that often complicate the treatment process. Currently, various methods are used to treat IHD [4]. Drug therapy is the main method and includes the administration of anti-anginal drugs, such as beta-adrenoceptor antagonists, calcium antagonists, and long-acting nitrates, which are referred to as first-line therapy. The use of drugs that improve disease prognosis is of no small importance. They include antiaggregants, statins, and angiotensin-converting enzyme (ACE) inhibitors.

Recently, in clinical practice for the treatment of angina pectoris, other drugs with a different mechanism of action, related to the so-called second- and third-line therapy, are being used more often. These include nicorandil, ivabradine, ranolazine, trimetazidine, perhexiline, allopurinol, molsidomine, and fasudil [5].

Despite the active use of a sufficiently broad range of drugs, drug treatment is often not effective enough for several reasons, such as low patient adherence to treatment, different individual patient sensitivity to various drugs, development of treatment tolerance, and side effects. The progression of coronary artery atherosclerosis also plays an important role, as it leads to the development of a more severe functional class (FC) of angina pectoris, despite active drug treatment.

In cases of insufficiently effective drug therapy, the use of myocardial revascularization methods leads to good results. Currently, the main methods of myocardial revascularization are endovascular treatment and coronary artery bypass grafting (CABG). Nevertheless, surgical interventions do not eliminate the cause of the disease, and the problem of IHD treatment remains relevant due to the accumulation of data from long-term follow-up of patients who underwent invasive treatment.

The main problem is the development of angina recurrence, which is caused by restenosis at the sites of stent implantation or occlusion of coronary artery bypass grafts, as well as the progression of coronary artery atherosclerosis and incomplete myocardial revascularization. The results of some studies have shown that approximately 30% of patients have symptoms of angina pectoris within a year after revascularization procedures [6], and after five years, some patients require repeated myocardial revascularization. However, for one reason or another, repeated revascularization cannot be performed on these patients. First, these are patients who refuse repeated interventions, who have various contraindications for repeated surgery, with diffuse lesions of the vascular bed, diseases of small vessels, and who have a history of myocardial revascularization with a poor result.

As a result of this, there is a need to search for new therapeutic methods of IHD.

External counterpulsation (ECP) is a relatively new, non-invasive, safe, and effective method of treating various forms of IHD, including stable effort angina. The main principle of the ECP apparatus is to increase the perfusion of coronary blood flow by increasing the blood pressure in the diastole, which is implemented by compression and decompression of special cuffs placed on the patient’s legs and buttocks. Studies have shown high safety and good tolerance of the ECP method, which led to the active introduction of this method of treatment in the USA and several other countries.

As a rule, this treatment method is prescribed to patients who have already received one or more revascularization procedures, who are undergoing maximum medical treatment, and, despite this, have severe angina pectoris FC (III or IV), which is called refractory angina. Now, the ECP method is actively used to treat refractory angina. The ECP method is recommended by the European Society of Cardiology in the official recommendations of 2019 for diagnostics and treatment of stable IHD with a class of recommendations IIb at the level of evidence base B.

Despite the rather extensive experience with using this method in patients with refractory angina, it is usually not prescribed at the initial stage of treatment to patients in addition to first-line therapy. This method is prescribed, as a rule, when all other treatment methods are ineffective, including revascularization procedures, as well as first-, second-, and third-line drug therapies. It is the therapy of the last line.

It should be recognized, however, that this technique is non-invasive and usually performed on an outpatient basis. This enables this method to be used in combination with first-line therapy in patients with stable effort angina even before using revascularization procedures. Therefore, the ECP method might be able to maximize the optimization of non-invasive treatment, namely the reduction of angina pectoris FC, improvement of quality of life, reduction of the need for nitroglycerin preparations, reduction of further need for myocardial revascularization procedures which may be economically justified.

Given the above, we aimed to evaluate the efficiency of the complex treatment of patients with stable effort angina using the ECP method.

This study included 92 patients, the main group consisted of 57 patients who received at least 20 hours of ECP therapy, and the control group included 35 patients. The selection of patients for the ECP course was based on known indications and
contraindications to this method of treatment, and the personal preferences of the patients were considered.

The criteria for inclusion in the study were the following.

1. Stable effort angina of I-IV FC (according to the classification of the Canadian Association of Cardiology).
2. Relapse of angina pectoris after invasive interventions (surgical treatment or angioplasty).
3. Refusal of the patient from invasive procedures, high risk of invasive procedures, the inability to perform the invasive treatment.

Before starting treatment with the ECP method, all patients received comprehensive information about this method of treatment and its possible side effects. All patients underwent coronary angiography before treatment.

In the main group, there were 57 patients, of whom 43 (75.4%) were men and 14 (24.6%) were women. The average age of the patients was 63.07 ± 7.44 years. The mean value of angina FC before treatment was 2.28 ± 0.73. The analysis of coronary angiograms of patients of this group showed that most had the three-vessel disease of the coronary arteries (33 patients, 57.9%), in 16 patients (28.1%), the two-vessel disease was diagnosed, and eight patients (14.0%) had a single-vessel disease.

In the main group, 27 patients had a history of myocardial infarction, which amounted to 47.4% of the total number of patients. Arterial hypertension was diagnosed in 27 (47.4%) patients, and diabetes mellitus was observed in 7 (12.3%) patients. Revascularization procedures before treatment using ECP were performed in 5 (8.8%) patients, one patient (1.8%) underwent coronary artery bypass grafting, and 4 (7%) patients underwent angioplasty with stent implantation.

Patients received basic optimal drug therapy with nitrates, antiplatelet agents, calcium antagonists, ACE inhibitors, beta-adrenoreceptor antagonists, ivabradine, statins, and diuretics. Nitroglycerin preparations were used by 22 (62.9%) patients.

In the control group, all patients received basic optimal drug therapy, including nitrates, antiplatelet agents, calcium antagonists, ACE inhibitors, beta-adrenoreceptor antagonists, ivabradine, statins, and diuretics. Nitroglycerin preparations were used by 31 patients.

All patients underwent a general clinical examination before and after the treatment, in both the main group and the control group, including history taking, complaints taking, and complete physical examination. The quality of life was assessed using the Seattle Quality of Life Questionnaire.

A General Electric ultrasound scanner, model Vivid 4, using a 3S transthoracic sensor, was used to perform echocardiographic examinations. The following echocardiographic parameters were calculated for all patients:

- end-diastolic diameter of the left ventricle;
- end-diastolic volume of the left ventricle;
- end-systolic volume of the left ventricle;
- stroke volume of the left ventricle;
- ejection fraction of the left ventricle;
- anteroposterior diameter of the left atrium;
- volume of the left atrium;
- degree of mitral regurgitation; and,
- indicators of tissue Dopplerography — the velocity of the mitral annulus (in tissue pulsed-wave Dopplerography, determining the maximum speed of movement of the mitral annulus in the phase of rapid filling of the left ventricle, ‘e’), the ratio E/e.’

An echocardiographic study was performed for all patients in both the main group and the control group. In the main group, study 1 was performed before ECP treatment, and study 2 was performed after its completion. In the control group, study 1 was performed before the start of drug treatment, and study 2 was performed after two months.

Exercise electrocardiography (stress ECG tests) was performed at least twice in all patients in both groups. Test 1 was performed before the prescribed treatment for ECP, and Test 2 was performed immediately after termination. A Contec model 8000S treadmill, equipped with a wireless record-
Table 1. Clinical characteristics of patients in the main and control groups

| Indicator                                      | Group                          | P   |
|-----------------------------------------------|--------------------------------|-----|
|                                               | External counter-pulsation (n = 57) | Control (n = 35) |
| Age at the time of treatment, years           | 63.07±7.44                     | 61.09±5.44 | 0.2544 |
| Gender (men/women), n                         | 43/14                          | 26/9 | 0.9013 |
| Functional class of angina pectoris before treatment | 2.28±0.73                     | 2.34±0.73 | 0.5966 |
| Single-vessel disease of coronary arteries     | 8 (14.0%)                      | 7 (20.0%) | 0.4857 |
| Two-vessel disease of coronary arteries        | 16 (28.1%)                     | 10 (28.6%) | 0.7869 |
| Three-vessel disease of coronary arteries      | 33 (57.9%)                     | 18 (51.42%) | 0.4653 |
| History of myocardial infarction              | 27 (47.4%)                     | 9 (25.7%) | 0.0388 |
| History of coronary artery bypass grafting    | 1 (1.8%)                       | 0 (0.0%) | 0.4308 |
| History of angioplasty                        | 4 (7.0%)                       | 3 (8.6%) | 0.7849 |
| Diabetes mellitus                             | 7 (12.3%)                      | 6 (17.1%) | 0.5157 |
| Hypertensive disease                          | 27 (47.4%)                     | 15 (42.9%) | 0.6732 |
| Nitroglycerin intake                          | 31 (54.4%)                     | 22 (62.9%) | 0.4247 |
| Antiaggregant intake                          | 56 (98.2%)                     | 35 (100.0%) | 0.4308 |
| Calcium antagonists intake                    | 17 (29.8%)                     | 13 (37.1%) | 0.4672 |
| Angiotensin-converting enzyme inhibitors intake | 41 (71.9%)                     | 19 (54.3%) | 0.0845 |
| Beta-adrenoreceptor antagonist intake          | 52 (91.2%)                     | 31 (88.6%) | 0.6771 |
| Ivabradine intake                             | 12 (21.1%)                     | 5 (14.3%) | 0.4169 |
| Statin intake                                 | 49 (86.0%)                     | 34 (97.1%) | 0.0798 |
| Diuretic intake                               | 23 (40.4%)                     | 13 (37.1%) | 0.7595 |

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ing of the patient’s electrocardiogram during stress testing, was used to conduct stress testing.

Before the start of treatment, all patients of both the main and control groups underwent selective coronary angiography, which was performed using angiographic devices from various manufacturers, such as the Axiom Artis system by Siemens (Germany), Innova 2100 by GE (USA), and Allura FD20 by Philips (USA).

For catheterization of the aorta followed by catheterization of the coronary arteries, the right or left transfemoral approach was used according to the Judkins method. Access to the femoral artery was provided by the Seldinger method under local anesthesia, namely with 20 ml of 0.5% procaine solution (procaine hydrochloride). Selective coronary angiography was performed using Judkins or Amplatz catheters. In contrast, the contrast agent Omnipack 350 was used. To contrast the left and right coronary arteries, standard projections, and in some cases, additional projections were used.

Coronary angiogram analysis was performed on monitors using programs integrated into the system. The analysis determined the type of blood supply to the heart, the lesion location in the coronary arteries by segments, the condition of the main trunk of the left coronary artery, the number of affected arteries, their diameter, and the degree of development of collateral blood flow.

Treatment with ECP was performed using a device of the Vasomedical company, model TS-4. Before the start of the treatment session, blood pressure was measured in all patients, and the clinical status was assessed. After changing clothes (the patient put on special underwear that reduced the likelihood of the skin irritation), the patient laid on the treatment bed of the ECP apparatus. Special cuffs were placed on the patient’s legs and buttocks. An electrocardiogram was taken using three electrodes that were applied to the patient’s skin and transmitted signals to the control screen of the ECP apparatus. A plethysmograph sensor was placed on the patient’s finger, the signals from which were also transmitted to the ECP apparatus screen so that the patient’s plethysmogram was displayed under its signal from the electrocardiograph (in single-channel mode).

After turning on the device, the air is supplied from the compressor to the cuffs strictly during diastole, and before the next systole, air from the
cuffs is quickly blown off. Systole is determined based on the R wave of the patient’s electrocardiogram. The physician regulates the compression and decompression times based on the patient’s plethysmogram in order to achieve optimal diastolic blood flow enhancement. The increment of adjusting the compression/decompression time is 10 ms, and the minimum time from the previous R wave is 150 ms.

The pressure of ECP was within the range of 200–300 mm Hg. ECP pressure was selected for each patient individually, as well as compression and decompression times. During treatment, the total treatment time was calculated; if necessary, patients received additional ECP sessions so that the total accumulated treatment time was 35 hours.

The two groups were compared according to quantitative scales based on the nonparametric Mann–Whitney test. A comparison of three or more groups on quantitative scales was performed based on the nonparametric Kruskal-Wallis test. To describe the quantitative indicators, the mean value and standard deviation in the $M \pm S$ format were used. An analysis of changes in indicators in

### Table 2. Comparison of the indicators before treatment and after treatment for the group of external counterpulsation (ECP) and the control group.

| Indicator                                      | Main group (ECP. n = 57) | Control group (n = 35) |
|------------------------------------------------|--------------------------|------------------------|
|                                                | Before treatment         | After treatment        | p          | Before treatment         | After treatment        | p          |
| Left ventricular end-diastolic dimension. cm   | 5.32±0.75                | 5.25±0.67              | 0.3047     | 5.27±0.48                | 5.21±0.47              | 0.0411     |
| Left ventricular end-diastolic volume. ml      | 80.21±26.99              | 79.82±26.21            | 0.6028     | 93.07±19.48              | 90.41±27.88            | 0.4414     |
| Stroke volume of the left ventricle. ml        | 44.48±11.04              | 48.00±11.32            | 0.0018     | 54.44±12.10              | 54.86±19.54            | 0.8314     |
| Left ventricular ejection fraction. %          | 57.46±9.99               | 62.15±9.00             | <0.0001    | 58.70±6.53               | 60.45±9.55             | 0.0711     |
| Left atrium dimension. cm                      | 3.85±0.42                | 3.82±0.40              | 0.4590     | 3.98±0.43                | 3.95±0.48              | 0.2845     |
| Left atrium volume. ml                        | 61.91±22.08              | 57.11±17.20            | 0.0071     | 65.15±20.06              | 64.33±22.53            | 0.2343     |
| Degree of mitral regurgitation. severity       | 0.88±0.48                | 0.82±0.44              | 0.1240     | 0.77±0.33                | 0.73±0.31              | 0.1088     |
| Mitral annular early velocity (e’). cm/s       | 7.02±1.57                | 7.31±1.85              | 0.0016     | 7.19±1.92                | 7.36±2.03              | 0.0543     |
| E/e’ ratio. n                                  | 12.10±4.72               | 11.63±4.46             | 0.1050     | 13.34±3.91               | 13.07±3.80             | 0.2028     |
| Total exercise performance time. s            | 266.81±108.09            | 370.00±116.63          | <0.0001    | 302.57±131.18            | 297.14±127.38          | 0.3391     |
| Exercise performed. metabolic equivalent       | 5.52±1.85                | 7.15±1.91              | <0.0001    | 5.87±2.15                | 5.78±2.10              | 0.3575     |
| Time before the onset of ST-segment depression by 1 mm.s | 174.91±89.71            | 258.35±101.97          | <0.0001    | 204.57±105.95            | 200.29±104.92          | 0.4321     |
| Angina class                                   | 2.28±0.73                | 0.93±0.80              | <0.0001    | 2.34±0.73                | 1.83±0.71              | 0.0030     |
| Scale of physical activity restrictions. %     | 67.42±6.15               | 65.02±5.68             | 0.0319     | 64.07±6.98               | 63.35±8.31             | 0.5614     |
| Scale of stability of angina attacks. %        | 63.16±15.02              | 93.33±10.91            | <0.0001    | 53.71±15.92              | 65.71±13.35            | <0.0001    |
| Scale of frequency of angina attacks. %        | 56.67±20.64              | 93.33±8.09             | <0.0001    | 51.43±15.74              | 64.29±12.67            | 0.0007     |
| Scale of satisfaction with treatment. %        | 66.21±15.30              | 83.97±10.77            | <0.0001    | 64.80±14.47              | 76.92±8.86             | 0.0003     |
| Scale of attitude to the disease. %            | 45.91±15.03              | 59.80±15.03            | <0.0001    | 46.90±21.58              | 55.96±16.61            | 0.0394     |
| Overall quality of life score                  | 299.36±34.26             | 395.45±27.27           | <0.0001    | 280.92±33.16             | 326.23±26.90           | <0.0001    |
the case of comparing two periods was conducted based on the nonparametric Wilcoxon test, and in the case of comparing three or more periods, it was performed based on the nonparametric Friedman test. Statistical data processing was performed using the Statistica 10 and SASJMP 11 application software packages.

This work was approved by the ethics committee of the scientific council of the National Academy of Sciences of Azerbaijan.

Table 2 presents the results of the statistical analysis of changes in indicators for the periods “Before treatment” and “After treatment” for the main group (ECP), and the control group for the categories “Echocardiography data,” “Exercise electrocardiography data,” “Change in the class of angina pectoris,” and “Quality of life assessment.”

Table 3 presents the comparative analysis of the dynamics of changes in these parameters.

Fig. 1 presents the results of a statistical analysis of nitroglycerin intake by patients for the periods before treatment and after treatment for the ECP group and the control group. In a comparative analysis for the period before treatment, there was no statistically significant difference in the use of nitroglycerin between both groups (54.4 and 62.9%, respectively; \( p = .4247 \)). However, in the period after treatment, the difference was significant; patients in the ECP group were less likely to use nitroglycerin than those in the control group (26.3% and 48.6%, respectively; \( p = .0296 \)).

Based on Tables 2 and 3, we can conclude that more statistically significant changes occurred in the ECP group. Some indicators changed significantly both in the ECP group and in the control group. Namely, angina FC decreased in both groups, all indicators of quality of life, except for restriction of physical activity, increased in both groups. Assessments on a physical activity scale did not change in the control group but decreased in the ECP group. The stroke volume of the left ventricle, the ejection fraction of the left ventricle,
and the early velocity of the mitral annulus increased in the ECP group, although there were no changes in these parameters in the control group. Despite a decrease in the volume of the left atrium after treatment in the ECP group and the control group, the differences were not significant.

Based on Table 3, we can conclude that when comparing changes in time in echocardiography, the changes were approximately the same in both groups. Differences were obtained only for changes in the ejection fraction of the left ventricle, which was greater in the ECP group, in which the fraction changed by almost 5%, whereas in the control group, it was only 2%. The changes in the time of all indicators of the stress ECG test differed in the main and control groups. In addition, the dynamics in the ECP group were positive, whereas, in the control group, they were negative. Thus, improvements are noted for the following:

- total exercise time (+103.19 ± 56.13 s versus –5.43 ± 38.68 s, p = .0001);
- exercise performed in metabolic equivalents (+1.63 ± 0.89 versus –0.09 ± 0.59, p = .0001); and
- time before the onset of ST-segment depression by 1 mm (+83.44 ± 57.78 s versus –4.29 ± 30.13 s, p = .0001).

When comparing the indicators of the quality of life, we can conclude that, on some scales, the dynamics were insignificant and equal for both groups. For example, according to the scale of physical activity restriction, the changes do not exceed 3 points. According to some scales (attack stability scales and the frequency of angina attacks), the changes in the ECP group were much higher than in the control group (30–39 points versus 12–13 points, respectively). The total score also varied much more in the ECP group than in the control group (average, about two times). A decrease in angina pectoris FC by 1 FC is also more pronounced in the ECP group than in the control group. The use of nitroglycerin sharply decreased after treatment in the ECP group (by more than 50%), and in the control group, despite a slight decrease, the changes did not reach statistical significance.

Treatment for stable effort angina has two main aims. The first is to reduce the frequency and intensity of angina attacks, increase in physical load tolerance and, therefore, improve the patient’s quality of life. The second aim is to improve prognosis and prevent the occurrence of acute myocardial infarction, unstable angina, and sudden cardiac death in this category of patients [7]. For this purpose, drug therapy is used, which is recommended for all patients, and myocardial revascularization procedures [8].

Along with drug therapy, which has been firmly established in the treatment of this pathology, new invasive therapeutic methods have been introduced, namely CABG and various methods of percutaneous coronary interventions, where the leading role is played by the procedure of implanting an endoprosthesis (stent) in the affected segment of the coronary artery. Invasive methods of treating angina increased the effectiveness of treating patients with IHD significantly, as they improved the quality of life and prognosis in this category of patients. CABG also improved the prognosis and quality of life in patients with lesions in the trunk of the left coronary artery, with three-vessel disease, and in patients with diabetes mellitus. Emergency coronary angioplasty performed in the first hours of an acute coronary syndrome with ST-segment elevation significantly reduces the mortality rate in this category of patients. These positive outcomes have led to the widespread use of invasive treatment methods for patients with IHD.

The significant positive aspects of the above invasive treatment methods, however, were accompanied by several factors that limited their use in a certain category of patients. The existing data indicate that, compared with drug therapy, percutaneous coronary interventions with stent implantation in the affected segment of the coronary artery do not lead to significant improvement in the prognosis of patients with stable angina. According to the latest results of the Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation study, the probability of death and nonfatal myocardial infarction upon follow-up for an average of 11.9 years did not statistically differ in the groups of patients who initially underwent coronary angioplasty with stenting and received aggressive drug therapy, compared with the group of patients who received only intensive drug treatment [9].
Despite the use of medical treatment and revascularization of the myocardium in clinical practice, the problem of treating angina pectoris in some patients does not have a solution. In these cases, when angina pectoris FC III or IV is established according to the classification of the Canadian Cardiovascular Society despite providing optimal drug therapy, prescribing lifestyle modifications, and performing revascularization procedures, angina pectoris is considered as refractory.

For the treatment of refractory angina, many drug and non-drug treatment methods have been proposed [10]. For drug treatment of refractory angina, in addition to first-line therapy, new drugs with various mechanisms of action have been developed. Among the main non-drug methods of treatment for refractory angina, the following treatment methods have been proposed and studied over recent years [11]:

- enhanced external counterpulsation;
- neurostimulation methods, namely percutaneous electoneurostimulation and spinal cord stimulation;
- shock wave therapy of the heart;
- transmyocardial and percutaneous laser revascularization;
- implantation of the coronary sinus reducer; and,
- various methods of therapy using stem cells and genetic engineering.

Most non-drug methods of treatment, despite some optimism at the initial stages, did not later receive a sufficiently high class of recommendations from the European Society of Cardiology. Thus, based on the recommendations of this society of 2013, percutaneous electroneurostimulation and spinal cord stimulation have a class of recommendations IIb, and transmyocardial revascularization has a class of recommendations III [12, 13]. At that, ECP has established itself as an effective method of treating refractory angina, which is specified in the recommendations of the European Society of Cardiology of 2013 and 2019.

The ECP method has been studied comprehensively and continuously to be studied in various clinics around the world [14–17]. The accumulated data demonstrated the possibility of the successful therapeutic use of the method in patients with chronic IHD and systolic heart failure of ischemic origin [18–20].

The positive effective effect of ECP therapy is due to various mechanisms of action [21]. An increase in coronary blood flow during the ECP procedure leads to an increase in shear stress, which has a positive effect in the form of increased collateral circulation, as through neangiogenesis (arteriogenesis) existing collaterals open and/or expand [22]. Endothelial function is improved, as well as the contractile ability of the myocardium. The clinical efficiency of one course of ECP remains for a long time after the end of the treatment course.

It should be recognized that the therapeutic effectiveness of ECP is not limited only by its effectiveness in patients with refractory angina, and this method can also be used for several other diseases. In our work, we demonstrated the effectiveness of this technique in patients with stable effort angina who received basic drug treatment. These results enable us to recommend this method for broader application in cardiology practice to treat patients with stable effort angina.

**CONCLUSIONS**

The method of external counterpulsation can be used in clinical practice as part of the comprehensive treatment of patients with stable effort angina. The use of this technique compared with the control group was able to reduce significantly the functional class of angina pectoris and the need to use nitroglycerin preparations and improve several indicators of echocardiography, exercise electrocardiography, and quality of life.

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**REFERENCES**

1. American Heart Association. Heart disease and stroke statistics — 2011 update. Circulation. 2011; 123 (4): e18–e209. DOI: 10.1161/CIR.0b013e3182009701.

2. Zaher C., Goldberg G.A., Kadlubek P. Estimating angina prevalence in a managed care population. *Am. J. Manag. Care*. 2004; 10 (11 suppl.): S339–S346. PMID: 15603243.

3. Hilton T.C., Chaitman B.R. The prognosis in stable and unstable angina. *Cardiol. Clin.* 1991; 9 (1): 27–38. DOI: 10.1016/S0733-8651(18)30315-1.

4. Kloner R.A., Chaitman B. Angina and its management. *J. Cardiovasc. Pharmacol. Therap.* 2016; 22 (3): 199–209. DOI: 10.1177/1074284416679733.

5. Cheng K., Sainsbury P., Fisher M., de Silva R. Management of refractory angina pectoris. *Eur. Cardiol. Rev*. 2016; 11 (2): 69–76. DOI: 10.15420/ecr.2016:26:1.

6. Holubkov R., Laskey W.K., Haviland A. Angina 1 year after percutaneous coronary intervention: a report from the NHLBI Dynamic Registry. *Am. Heart J*. 2002; 144 (5): 826–833. DOI: 10.1067/mhj.2002.125505.

7. Abrams J., Thadani U. Therapy of stable angina pectoris: The uncomplicated patient. *Am. J. Manag. Care*. 2005; 11 (12 suppl.): e255–e259. DOI: 10.1161/CIRCULATIONAHA.104.526609.

8. Jain A., Elgendy I., Al-Ani M. et al. Advances in pharmacotherapy for angina. *Expert Opin Pharmacother*. 2017; 18 (5): 457–469. DOI: 10.1080/14656566.2017.1303483.

9. Sedlis S.P., Hartigan P.M., Teo K.K. Effect of PCI on long-term survival in patients with stable ischemic
heart disease. *N. Engl. J. Med.* 2015; 373: 1937–1946. DOI: 10.1056/NEJMoai1505532.

10. Kocyigit D., Gurses K.M., Yalcin M.U., Tokgozoglu L. Traditional and alternative therapies for refractory angina. *Curr. Pharmaceut. Design.* 2017; 23 (7): 1098–1111. DOI: 10.2174/138161282366612123145148.

11. Abdelwahab A.A., Elsaied A.M. Can enhanced external counterpulsion as a non-invasive modality be useful in patients with ischemic cardiomyopathy after coronary artery bypass grafting? *Egypt Heart J.* 2018; 70 (2): 119–123. DOI: 10.1016/j.ehj.2018.01.002.

12. Montalescot G., Sechtem U., Achenbach S. et al. 2013 ESC guidelines on the management of stable coronary artery disease. *Eur. Heart J.* 2013; 34: 2949–3003. DOI: 10.1093/eurheartj/eht296.

13. Soran O. Alternative therapy for medically refractory angina: Enhanced external counterpulsation and transmyocardial laser revascularization. *Heart Fail. Clin.* 2016; 12 (1): 107–116. DOI: 10.1016/j.hfc.2015.08.009.

14. Ahlborn M., Hagerman I., Ståhlberg M. et al. Increases in cardiac output and oxygen consumption during enhanced external counterpulsation. *Heart Lung Circ.* 2016; 25 (1): 1133–1136. DOI: 10.1016/j.hlc.2016.04.013.

15. Raza A., Steinberg K., Tartaglia J. et al. Enhanced external counterpulsion therapy: Past, present, and future. *Cardiol. Rev.* 2017; 25 (2): 59–67. DOI: 10.1097/CRD.000000000000022.

16. Sardari A., Hosseini S.K., Bozorgi A. et al. Effects of enhanced external counterpulsion on heart rate recovery in patients with coronary artery disease. *J. Tehran. Heart Cent.* 2018; 13 (1): 13–17. PMID: 29997665.

17. Valenzuela P.L., Sánchez-Martínez G., Torrontegi E. et al. Enhanced external counterpulsion and short-term recovery from high-intensity interval training. *Int. J. Sports Physiol. Perform.* 2018; 13 (8): 1100–1106. DOI: 10.1123/ijsspp.2017-0792.

18. Linnemeier G. Enhanced external counterpulsion — a therapeutic option for patients with chronic cardiovascular problems. *J. Cardiovasc. Man.* 2002; 13: 20–25. PMID: 12500419.

19. Melin M., Montelius A., Rydén L. et al. Effects of enhanced external counterpulsion on skeletal muscle gene expression in patients with severe heart failure. *Clin. Physiol. Funct. Imaging.* 2018; 38 (1): 118–127. DOI: 10.1111/cpf.12392.

20. Ranitya R. Enhanced external counterpulsion in chronic heart failure: Where do we stand? *Acta. Med. Indones.* 2015; 47 (4): 273–274. PMID: 26932694.

21. Du J., Wang L. Enhanced external counterpulsion treatment may intervene the advanced atherosclerotic plaque progression by inducing the variations of mechanical factors: A 3D FSI study based on in vivo animal experiment. *Mol. Cell Biomech.* 2015; 12 (4): 249–263. PMID: 27263260.

22. Degen A., Millenaar D., Schirmer S.H. Therapeutic approaches in the stimulation of the coronary collateral circulation. *Curr. Cardiol. Rev.* 2014; 10 (1): 65–72. DOI: 10.2174/1573403X113099990027.