In the 21st century, the number of patients with combined cardiovascular and respiratory system pathologies has progressively increased [9]. The association of arterial hypertension (AH) and chronic obstructive pulmonary disease (COPD) is an example of a complex disease synergy, in which these conditions are not only interconnected but also capable of complicating each other. They often manifest at the same age, share several similar pathophysiological mechanisms, and their frequency and socio-economic significance are on the rise with common risk factors.

According to data of the World Health Organization (WHO), in 2018, 2.0 million adults, or 1.6% of those ages 18 or older, had COPD. In 2020,

**Features of the Influence of Chronic Obstructive Pulmonary Disease on the Structural and Functional Parameters of the Heart and Vessels: Possibilities for Correction**

The presence of concomitant cardiac pathology exacerbates the course of chronic obstructive pulmonary disease (COPD) and worsens the prognosis for both conditions.

**Objective** — to improving the efficiency of diagnostics and treatment in patients with arterial hypertension and COPD, adding L-arginine to the complex therapy.

**Materials and methods.** A clinical examination was performed on a total of 140 patients, consisting of 82 individuals with arterial hypertension complicated by chronic obstructive pulmonary disease (COPD), and 58 patients with isolated hypertension. Each group was further subdivided into subgroups based on their treatment regimen, which included basic therapy alone, basic therapy along with co-existing pulmonary disease management, and a subgroup receiving basic therapy combined with L-arginine supplementation.

**Results and discussion.** The course of the diseases was more severe in patients with combined pathology. An acute drug test with arginine showed a decrease in blood pressure in both the small and large circles of circulation along with changes in endothelial function. Three months after the complex treatment with L-arginine resulted in the decrease of pressure in the small circle of circulation, had an effect on the structural and functional changes of both ventricles, diminished manifestations of diastolic dysfunction of the heart, increasing the physical activity and endothelium-dependent vasodilation, reduced the activity of anti-inflammatory cytokines in patients with hypertension and COPD. The inclusion of L-arginine as a functional corrector of endothelial function to the basic therapy of patients with arterial hypertension and COPD contributes to the improvement of the clinical condition of patients, normalizes blood pressure and its daily profile patients after 3 months of treatment, thereby enhancing their quality of life.

**Conclusions.** L-arginine, as a pathogenically selected donator of nitric oxide, along with basic therapy of essential hypertension and COPD improved the state of the heart and vessels and should be used for treatment both diseases.

**Keywords**

Arterial hypertension, chronic obstructive pulmonary disease, arginine, pharmacotherapy, endothelial dysfunction.
EU reach 38.6 billion euros [20, 33].

According to statistics, the incidence of COPD among Ukrainians is at the level of seven percent, that is, it covers approximately three million people, the prevalence of COPD among patients with comorbid cardiovascular pathology over the age of 60 varies in different countries from 7.8 to 19.7 % [1]. COPD not only aggravates the course of cardiac pathology, but also requires making certain corrections in the treatment process [30]. WHO attributes COPD, as well as AH, to diseases of considerable social importance due to their widespread distribution in both developed and developing countries [7].

According to the concepts of Global Initiative for chronic obstructive pulmonary disease, GOLD [7], COPD is a disease with significant extrapulmonary manifestation such as AH, metabolic syndrome, obesity, etc. They significantly complicate not only the course of the disease, but also lead to a deterioration in the quality of patient’s life. COPD becomes not only a pulmonary problem, but also a cardiac due to the frequent progressing of cardiovascular comorbid pathology in this category of patients [11]. The comorbidity of COPD and AH remains the most important, their combination ranges from 4 to 27.7 %, in the older age groups up to 62 %, it is increasing with age [39]. Recently, in the pathogenesis of pulmonary hypertension (PH) of bronchial pulmonary genesis, AH, much attention is paid to studying endothelial dysfunction as a pathogenetic link of these diseases [12, 13].

Objective — to improving the efficiency of diagnostics and treatment in patients with AH and COPD by adding L-arginine to the complex therapy.

Materials and methods

Study Design

This prospective clinical study was conducted in the Ivano-Frankivsk Regional Phthisio-Pulmonary Centre. The patients were enrolled between April and September 2022.

Study Population

The study was based on 140 patients with AH: the main group including 82 patients with COPD-associated arterial hypertension (AH) in the remission phase and with a III degree of bronchial obstruction. The control group comprised 58 patients with stage II first-degree/second-degree isolated AH. The average age was 59.5 ± 1.2. Among the patients with AH, 57 % were women, while among the AH with COPD patients, 65 % were men. The comparison group included apparently healthy individuals of the same gender and similar age. The number of smokers in the AH with COPD group was 82.9 %, while in the AH group, it was 40 %. The duration of AH was (5.3 ± 1.12) years, and for COPD, it was (10.2 ± 2.31) years. The frequency of COPD exacerbations was (2.5 ± 0.49) times per year. Among patients in the AH with COPD group (Group E, high risk, more symptoms), there were 82 patients. Clinical signs of heart failure (HF) were found in 75.6 % (62 individuals), while others had stage I HF with reported pulmonary insufficiency in 34.1 %.

Both groups were divided into subgroups according to the treatment. Subgroup A of the main group included patients receiving basic therapy in AH with COPD (n = 40); subgroup B of the main group — patients receiving basic therapy for AH with COPD with the addition of L-arginine (n = 42). The control group consisted of 58 patients receiving basic therapy for AH. Patients with COPD and AH were included in the study for comparison purposes, considering the goal of evaluating the effectiveness of L-arginine therapy.

All examinations were conducted before treatment, two weeks after treatment, and three months after treatment. Inclusion criteria: Stage II AH, Grade 2—3 heart failure (HF) with preserved ejection function (EF) (NYHA functional class I—III, 1994), COPD Stage III bronchial obstruction, Group E in the remission phase, with a duration of no more than 6 months, and pulmonary hypertension of Grade II (PH). Post-bronchodilatory increase in forced expiratory volume in the 1st second (FEV1) was less than 12 % compared to the initial value.

Diagnostic Statements

Traditional clinical examination methods were used: complaints, anamnestic data, physical examination, electrocardiography (ECG) and Echo-CG, daily monitoring of blood pressure (DMBP). Left ventricular hypertrophy by ECG was evaluated.
using Sokolov—Lyon and Levis methods [2]. 24-hour DMBP was performed with AVR-M04 apparatus of Meditech (Hungary). Cardiac hemodynamics was studied using the Echo-CG method on Carians-Plus 1,057 (Italia) using a 2.5-MHz sensor. The vasomotor function of the endothelium and the vascular reactivity of the peripheral vessels was determined on an ultrasonic device Cranzburger Loci-Q 500 (Germany) using a 7.5 MHz linear sensor. Endothelial dysfunction (ED) was determined on the basis of endothelium-dependent and endothelium-independent vasodilation of the brachial artery (BA), according to the method described by D. Celemajer and co-authors [14]. All patients were tested for the presence of coronavirus disease using polymerase chain reaction (ELISA test). The results were negative.

External respiratory function was evaluated by computer spiograph (Hellige EK 512E) and flow-volume curve analysis. Reversibility of bronchial obstruction was evaluated using a pharmacological test with salbutamol in a dose of 400 μg with measurement of bronchodilation response after 15 min.

The content of endothelin-1, atrial natriuretic peptide (ANP), IL-6 and TNF-α in blood plasma were determined by solid phase enzyme immunoassay using Biomedica reagent kits (Austria).

**Patient Management**

AH was diagnosed and treated according to 2013 Clinical Practice Guidelines for the Management of AH of the ESH and the ESC. COPD was managed in accordance with the guidelines of the International Congress on the «Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease» [20].

Basic therapy in patients with AH: perindopril, amlodipine, torasemide, nebivolol, rosuvastatin and acetylsalicylic acid. Patients with AH and COPD were treated with — LAMA (tiotropium bromide), LAMA/LABA (tiotropium bromide/olodaterol) — group E according to the current protocols. Subgroup A of main group included patients receiving basic therapy in AH with COPD (n = 40); subgroup B of main group — patients receiving basic therapy in AH with COPD with adding L-arginine (n = 42).

L-arginine hydrochloride 4.2 % solution (100 ml) was administered intravenously with 100 ml within 10 days, 10 drops per minute for the first 15 minutes, then the injection rate was increased to 30 drops per minute (Tivortion-aspartat, URiA-FARM) for 10 days and continued its administration in oral dosage form — L-arginine aspartate (Tivortion-aspartat, URiA-FARM) (1-dimensional spoon 5 times a day) for 3 months.

**Statistical Analysis**

All statistical processing of the study was performed using the built-in license packages for data analysis and descriptive statistics in Microsoft Excel 2007 and Statistica 10. Quantitative data obtained in the study were first checked for the type of their distribution using Kolmogorov—Smirnov & Lilliefors test for normality and Shapiro—Wilk’s W-test. Since they all conformed to the law of normal distribution, arithmetic mean (M) and the mean square deviation (σ) were chosen. That’s why, the parametric paired Student’s t-test was used to test the null hypothesis regarding the reliability of the data difference in the two comparison groups. The Odds ratio (OR) and the Spearman’s rank correlation analysis were used. A p-value < 0.05 was considered statistically significant.

**Results**

It was found that in patients with AH and COPD, the clinical course of AH was more severe. The levels of systolic and diastolic blood pressure (SBP and DBP) were significantly higher — the SBP on average was 19.7 % higher than in the control group (p < 0.05), the average day-time — 18 % (p < 0.001), and the average night-time, when as a rule there were shortness of breath paroxysms, elevations of blood pressure (BP) in the pulmonary circulation — 39.3 % higher than in patients of the control group (p < 0.001). DBP in patients with COPD exceeded blood pressure levels in the comparison group — average day-time — by 13.4 %, and average night-time, — by 17.1 % (all p < 0.05).

We observed changes in the daily profile of BP for «Non-dipper» in 64.5 % and «Night-peeker» in 21 %, while in patients with essential AH profile «Dipper» dominated in 58.7 % and «Non-dipper» in 29.3 % of patients.

The value of the mean systolic pressure in the small circle of circulation in patients with AH without COPD corresponded to normal values — 25 [16.8—31.3] mm Hg and in patients with AH, combined with COPD, — the 1° degree of pulmonary hypertension according to the classification of Amosov (1971) — 48 [41.6—54.5] mm Hg, that was 92 % higher than the level of the comparison group (p < 0.001). The patients had shortness of breath, cyanosis and FEV, per second, which according to spiographs in patients with combined pathology was (27.6 ± 8.5) % compared with indicators in patients without COPD — (69.1 ± 4.32) % (p < 0.01).

In contrast to patients with AH, who had deviation of the electric axis of the heart to the left and the Sokolov—Lyon index RV5 > 26 mm in 100 % of cases, the Lewis index RI + SIII > 25 mm in 96.5 %, in patients of the main group, these rates were only
54.8 and 18.3 %, respectively, and the presence of P. pulmonale in 61 % and the Lewis index (R1 + SIII > 25 mm) in 58.5 % of patients. In patients with AH and COPD, unlike patients with AH, ECG clearly showed signs of hypertrophy of both ventricles and right atrium, that is a predictor of cardiovascular complications, cardiac arrhythmias, and overall mortality [3]. End-diastolic diameter of the left ventricle (LV) was 12 % higher, the end-systolic volume 13.8 % higher (p < 0.001). The diameter of the left atrial cavity in patients with COPD and AH was 43.5 [42.6—44.3] mm, which is 13 % larger than in the control group (p < 0.05). Also patients of this group, had increased early transmitral diastolic flow velocity (E) by 10.0 % (p < 0.05) and a decrease in late (atrial) transmitral diastolic flow velocity (A) by 4.3 % (p < 0.05). Thus, E/A was reduced by 7 % relatively to control (p < 0.05). In patients of main group, an increase in the size of the right ventricle (RV), the thickness of the wall of which was 46.0 [45.8—46.2] mm, and the transverse size of the right atrium — 36.0 [35.6—36.3] mm, were significantly higher than in the comparison group (p < 0.05) and, together with the clinical signs indicating RV systolic insufficiency, attached to the LV compensatory changes and its diastolic dysfunction.

If the dominant type of LV remodeling in patients with AH was concentric hypertrophy of the LV (in 69 %), then patients with AH and COPD had concentric remodeling (in 70.7 % of cases), and concentric hypertrophy was observed only in 18.3 % of patients and twice as much in patients with eccentric hypertrophy (11 %).

Vascular movement of the endothelium during the Zellermeier—Sorenson test with reactive hyperemia showed a decrease in the diameter of the BA in ultrasound imaging in patients of both groups with AH and in patients with AH and COPD compared with healthy people (Figure). Accordingly, it was 0.39 [0.33—0.45] cm, 0.37 [0.31—0.43] cm and in the group of healthy individuals < 0.53 [0.43—0.62] cm, which was significantly higher than in patients of both groups (p < 0.05), and indicated ED in patients in both study groups. In response to sublingual nitroglycerin intake, the meaning of diameters of the BA in patients of both groups and healthy patients increased, respectively, by 5, 19, and 13 % (p < 0.05).

The cytokine system, a participant in inflammatory processes, has been found to be most active in patients with combined pathology, even in the absence of a COPD exacerbation phase (Table). The concentration of IL-6 in plasma was 5.8 times higher in patients with AH (p < 0.05) than in healthy patients, and 1.4 times higher in patients with combined pathology compared to the control group (p < 0.001). Similarly, the activity of TNF-α increased. Plasma ANP was 3.63 times higher in

### Table. Effect of L-arginine on the cytokine profile and indicators of neurohumoral regulation in patients with AH and COPD (М ± σ)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Healthy (n = 30)</th>
<th>The patients with AH and COPD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>The patients with AH and COPD (n = 40)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Before treatment</td>
</tr>
<tr>
<td>ANP, nmol/ml</td>
<td>1.46 [1.2—2.3]</td>
<td>8.8* [6.7—10.9]</td>
</tr>
<tr>
<td>ET-1, fmol/ml</td>
<td>0.26 [0.2—0.5]</td>
<td>3.8* [1.2—6.0]</td>
</tr>
</tbody>
</table>

Note. BT — basic therapy; * statistical significance of differences compared to healthy; ** statistical significance of differences compared to pre-treatment rates, (p < 0.05).
patients with AH and 6 times higher than in healthy patients (p < 0.001). The vasoconstrictor ET-1 was in 11.0 times and 14.6 times higher, respectively (p < 0.001) than in the healthy. All this indicates about ED — reduction of vasodilators and increase of vasoconstrictors, which in turn changes the structure and function of both ventricles of the heart, HF. This is confirmed by the direct correlation between ET-1 and mean BP in the pulmonary artery (r = +0.41, p < 0.05).

Using the L-arginine drug as a pathogenetic factor in the acute drug Zellermeier—Sorenson’s test instead of nitroglycerin [24] showed that during the test, the expansion of the BA diameter in response to the infusion of L-arginine was 22 % in patients in the main group, 10 % in patients with AH and 17 % in healthy people (p < 0.05), indicating the nitrate-like properties of L-arginine and allowed its use in the complex therapy of AH and COPD for functional correction of endothelial dysfunction. Moreover, we found during this acute test DMAT have shown that L-arginine had a pronounced antihypertensive effect in the large circulatory system during the infusion and the day after, indicating its vasodilatory capabilities. Since endothelium-dependent vasodilation is due to the action of NO [28, 35, 37], the obtained expansion of the diameter of the BA testified the donor NO properties of L-arginine and confirmed the pathogenetic direction of the suggested treatment.

The inclusion of L-arginine to the treatment of main group patients showed within 2 weeks of therapy a decrease in the average daily SBP and DBP, in drug-resistant patients with COPD on 33 % (p < 0.01), while without L-arginine this decrease was 7.5 % (p > 0.05). Targeted levels of SBP were maintained at the end of 3 months of therapy.

The clinical status of patients also significantly changed. All the changes described were due to an improvement in endothelial function under the influence of L-arginine. This is evidenced by the increase in the diameter of BA for reactive hyperemia to 0.47 [0.44—0.51] cm (p < 0.001) in the main group of patients, unlike in patients who received standard therapy, the diameter of the vessel increased by 0.01 cm (p < 0.1). In patients with AH the diameter of BA during reactive hyperemia significantly increased to 0.46 [0.37—0.48] cm (p < 0.05) (see Figure). Changes in peripheral hemodynamics under the influence of L-arginine were also reflected in the pressure in the small circulation, which at the end of treatment decreased to 31.5 [29.7—34.2] mm Hg (p < 0.01).

L-arginine therapy led to a change in the types of circadian BP profiles. In patients with AH without aggravating disease, 59 % of patients had a «Dipper» profile, 29 % had «Non-dipper» and 12 % had «Over-dipper»; in patients with COPD the number of patients with the «Non-dipper» profile was 64 %, with a steady increase of BP at night («Night-peaker») — 21 %, the «Dipper» profile share was only 10 %, and the «Over-dipper» was 5 %. 3 months after the complex treatment with L-arginine, the daily BP profiles underwent significant changes and the main group of patients was 45 % with «Dipper» profile, 40 % with «Non-dipper», 10 % with «Night-peaker» and 5 % with «Over-dipper».

The decrease in BP in the small circle of circulation under the influence of treatment contributed to the increase of ventilation and gas exchange, as evidenced by the improvement of vital capacity and especially the increase of Tiffino index, which after 3 months of treatment approached 71.2 [50.4—90.0] %, (p < 0.05). Positive reliable correlation of moderate degree was found between the Tiffino index value and SBP daily (r = 0.70, p < 0.05), between the systolic pressure in the small circulatory system and the daytime DBP (r = 0.67, p < 0.05).

Therapy with the inclusion of L-arginine had an impact on the structural and functional changes of both ventricles — decreased manifestations of its diastolic dysfunction in patients with AH and COPD, the size and volume of the ventricles, which was reliable and prognostically favorable for these patients in terms of diseases and complications that accompany ventricular remodeling — HF, fatal cardiac arrhythmias and more. Thus, in patients with AH and COPD, the time of isovolumic relaxation of the LV increased by 34 % (p < 0.05), which increased E/A to 1.22 [0.90—1.54], p < 0.05 and indicates a better diastolic function of the LV. The final diastolic size of RV decreased after 3 months of L-arginine treatment from 3.23 [2.85—3.63] cm to 2.65 [2.46—2.84] cm (p < 0.001). The size of the left atrium and ventricle, the thickness of the posterior wall of the LV and its myocardial mass index were statistically significantly decreased. This also optimized the contractile capacity of the LV — its stroke volume increased by 21.1 % (p < 0.05).

Long-term treatment with L-arginine for 3 months, with baseline therapy, also improved the functional state of the vessels, as indicated by the corrected endothelial function of the BA in the Zellermeier trial: the diameter of the BA increased by 27.7 % versus 7.7 % in patients treated with basic therapy and 6 % in patients with AH. This indicates a higher efficacy of L-arginine in hypoxia and hypercapnia.

L-arginine is a pathogenetically selected donor of NO, which significantly reduces the content of IL-6 in the serum of patients with AH and COPD by 1.9 times, TNF-α — by 2.3 times (see Table). Extremely important is the relationship of these
indicators and changes in geometric patterns of the left ventricle. IL-6 production decreased the most during eccentric LV hypertrophy (1.6 times, p < 0.05). The development of HF was slowed by concentric hypertrophy (decrease in the amount of ANP by 2.6 times, p < 0.05), as well as apoptosis of the LV myocardium (decrease in TNF-α content by 2.5 times, p < 0.05).

Therapy with L-arginine increased the physical activity of patients with AH with COPD by 20%, their general health status was rated higher by 31.6% (p < 0.05), while basic therapy did not cause such an assessment (tendency to improve). The quality of life of patients in the main group (combination of AH with COPD) was assessed using the St. George questionnaire (SGRQ).

**Discussion**

We believe that AH is obviously an independent disease, since AH occurred after the age of 40, as essential hypertension typically begins, in patients with impaired heredity in 89% of cases, has a stable course and is not associated with exacerbations of COPD [9, 15]. And if some authors [10, 20, 21] believe that the rise in blood pressure in patients with COPD is associated with attacks of bronchospasm and hypoxia, this also denies the existence of pulmonary hypertension in this category of patients [13].

The presence of COPD and pulmonary hypertension, hypoxia and hypercapnia in patients with AH contributed to a change in the daily profile of BP for «Non-dipper» and «Night-peakers», while in patients with essential AH profile «Dipper» dominated in 58.7% of patients. This provided the conditions for a permanent hemodynamic load, the formation of dysfunction of both ventricles of the heart and hypertension in the small circulatory system in patients with AH and COPD III degree of bronchial obstruction [13, 30].

Structural and functional parameters are defined by us of both ventricles differed significantly in patients of the main group in comparison to patients with AH due to the severity of hypertension in comorbid pathology, due to hypoxia and dystrophic changes in the myocardium due to bronchial obstructive syndrome [10, 20]. There is a close relationship between ED and left ventricular hypertrophy (LVH), in patients with AH ED is an important component in the structure and function of the heart that is defined by us [11, 17, 21].

ED is accompanied by increased secretion of vasoconstrictors and neurotransmitters of the inflammatory response [19, 37]. The cytokine system, as a participant of inflammatory processes [6], has shown itself to be in the most active state in patients with combined pathology, despite the absence of an exacerbation phase of COPD (see Table). Regulation of vascular tone and atrial status, which affects vascular filling and is regulated by ANP, clearly demonstrates the changes which were observed in patients with AH and COPD and pulmonary hypertension. This is confirmed by the direct correlation between ET-1 and mean BP in the pulmonary artery (r = +0.41, p < 0.05) and by the data of other authors [26, 31].

Since we have found out that the most important and common pathogenetic link in the development of AH and COPD is an imbalance between vasoconstrictors of different origin and endothelium-releasing factor, we used as a pathogenetic factor arginine drug. Moreover, at high pressure in large and small circles of circulation the work of eNO-synthase is disrupted, availability of arginine reserves for its work is reduced [18, 34]. And in the body, NO is synthesized from the aminoacid arginine by the enzyme NO synthetase by attaching molecular oxygen to the terminal nitrogen atom in the guanidine group of arginine [18, 23].

To confirm the validity of such thoughts, we conducted an acute drug test with the drug L-arginine during the Zellermeier—Sorenson test [14], where the investigational drug was used instead of nitroglycerin [24]. Since endothelium-dependent vasodilation is due to the action of NO [5, 28], the obtained expansion of the diameter of the BA testified the donor NO properties of L-arginine and confirmed the pathogenetic direction of the suggested treatment.

Long-term treatment with L-arginine for 3 months, with baseline therapy, also improved the functional state of the vessels, as indicated by the corrected endothelial function of the BA in the Zellermeier trial. This indicates a higher efficacy of L-arginine in hypoxia and hypercapnia. This judgment is confirmed by the information of other authors [5, 22, 28] and confirms the close dependence of BP on the severity of ventilatory disorders and believe that AH contributes to the frequent exacerbation of COPD, the development of its resistance to treatment, more severe bronchial obstruction [6, 32].

Therefore, L-arginine therapy with NO stimulation at the beginning of remodeling has a positive effect; the eccentrically altered LV improves its structure and function. Hence, L-arginine has an additional effect, apparently, through the regulation of endothelial function, on the intensity of immunoinflammatory mechanisms [28, 35, 37].

The correlated relationships between the condition of the cardiovascular and the respiratory system reveal the mutual encumbrance of diseases and the high dependence of systemic hypertension on endo-
Conclusions

Patients with comorbid pathology: AH II with COPD of III stage bronchial obstruction in the remission phase have chronic HF with preserved LV systolic function, diastolic dysfunction of the left and right ventricles with impaired relaxation, prevalence of concentric LV remodeling and concentric hypertrophy, dilatation of the RA and RV. Test with reactive hyperemia indicates endothelial dysfunction. Indicators of systemic overall response and factors of neurohumoral activation of blood are directly dependent on the severity of the pathological process and from the geometry of the heart.

The inclusion of L-arginine as a functional corrector of endothelial function to the basic therapy of patients with AH and COPD contributes to the improvement of the clinical condition of patients, normalizes BP and its daily profile patients after 3 months of treatment, affecting quality of life. L-arginine therapy improved the state of systolic and functions of both ventricles, final systolic and diastolic size of the LV, increased E/A index of the LV, reduced the thickness of the posterior wall of the LV and caused decrease in mean BP in the pulmonary artery; had a positive impact on the processes of myocardial remodeling, optimized endothelial function. L-arginine improves the volumetric and rapid rates of ventilatory function of the lungs, significantly reduced the content of vasoconstrictors ET-1, ANP and IL in 3 months.

References

9. Ethical Statement & Informed Consent. The research was approved by the Bioethical Committee of Ivanо-Frankivsk National Medical University. A consent form was signed by each prospective participant before recruitment into the study. All the procedures in the study met biotechnical standards according to the Helsinki Declaration.
10. Data Availability. The data that support the findings of this study are available from the corresponding author upon reasonable request.
11. The authors declare that no conflicts exist. The authors declared no financial support.
Особливості впливу хронічного обструктивного захворювання легень на структурно-функціональні параметри серця і судин: можливості їхньої корекції

Наявність супутньої серцевої патології обтяжує перебіг хронічного обструктивного захворювання легень і погіршує прогноз обох захворювань.

Мета роботи — корекція порушень структурно-функціонального стану серця і судин, системної запальної відповіді у хворих на артеріальну гіпертензію, асоційовану з хронічним обструктивним захворюванням легень, зокрема в рамках комплексної терапії з L-аргініном.

Матеріали та методи. Проведено клінічне обстеження 140 хворих, з них 82 хворих на артеріальну гіпертензію. Обидві групи розділили на підгрупи відповідно до лікування (базова терапія та лікування з L-аргініном).

Результати та обговорення. У пацієнтів з артеріальним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіпертензійним гіперт...
ції ендотелію до базисної терапії хворих на артеріальну гіпертензію та хронічне обструктивне захворювання легень сприяє поліпшенню клінічного стану пацієнтів, нормалізації артеріального тиску та його добового профілю, а отже, підвищує якість життя.

**Висновки.** L-аргінін, як патогенетично детермінований донатор оксиду азоту, разом із базовою терапією есенціальної гіпертензії та хронічного обструктивного захворювання легень поліпшує стан серця та судин. Його слід використовувати для лікування обох захворювань.

**Ключові слова:** артеріальна гіпертензія, хронічне обструктивне захворювання легень, аргінін, фармакотерапія, ендотеліальна дисфункція.

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