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THE ROLE OF β-ADRENOCEPTOR BLOCKERS IN MODERN CLINICAL PRACTICE OF THE FAMILY PHYSICIAN

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Annotation. *The review article is dedicated to the modern role of beta-adrenoceptor blockers in the practice of a family physician. These drugs remain key tools in the treatment of patients with coronary heart disease, arterial hypertension, chronic heart failure, and arrhythmias. The article emphasizes the necessity of an individualized approach to the selection of beta-adrenoceptor blockers, taking into account pharmacological properties, comorbid conditions, and safety profile. Objective of the work – to analyze and evaluate the role of beta-adrenoceptor blockers in the modern clinical practice of a family physician. A retrospective analysis of literature sources on this topic was performed based on the scientometric databases PubMed, Web of Science, and Scopus. During the material processing stage, the most relevant primary sources were selected. The main selection criteria were the authority of the publication and the date of publication. By examining abstracts and full texts of the articles, 45 sources were selected for further analysis. For patients with chronic heart failure, beta-adrenoceptor blockers are a vital component of treatment that reduces mortality. Important criteria for drug selection include cardioselectivity, lipophilicity, the presence of vasodilating action, and intrinsic sympathomimetic activity. New generations of drugs have an additional vasodilating effect, which improves hemodynamics. The family physician must consider the features of pharmacokinetics in kidney and liver pathology and avoid abrupt discontinuation of therapy. The characteristics of the main representatives of this class of drugs are provided with a detailed description of their clinical benefits, dosages, and indications. The article separately considers absolute and relative contraindications for the use of beta-adrenoceptor blockers, as well as management tactics in case of overdose. Thus β-adrenoceptor blockers remain an important component in the therapy of cardiovascular diseases such as coronary heart disease, chronic heart failure, arterial hypertension, and arrhythmias, thanks to their proven ability to reduce mortality and the risk of complications. The efficacy and safety of beta-adrenoceptor blocker use are determined by their pharmacological properties and the presence of intrinsic sympathomimetic activity, which requires individualized drug selection depending on concomitant pathology. In the practice of a family physician, it is important to adhere to the principles of rational pharmacotherapy: carefully consider the indications and contraindications for prescription, avoid withdrawal syndrome, and perform gradual dose titration, which ensures the optimal clinical outcome.*
Keywords: *arterial hypertension, beta-adrenoceptor blockers, coronary heart disease.*

Introduction

Currently, β-adrenoblockers (β-blockers, BBs) remain a cornerstone in the treatment of a wide range of cardiovascular diseases, including ischemic heart disease (IHD), arterial hypertension (AH), chronic heart failure (CHF), and arrhythmias. In family medicine practice, BBs are among the most frequently prescribed classes of drugs. However, their use requires a deep understanding of pharmacological differences, particularly in the context of comorbid conditions such as chronic obstructive pulmonary disease (COPD) or diabetes [19, 28].

Secondary prevention after myocardial infarction is a classical indication where β-blockers have historically demonstrated a reduction in mortality and the risk of recurrent cardiovascular events. The choice of a specific β-blocker in the treatment of patients with ischemic heart disease should be based on its pharmacological properties, which influence tolerability and safety [5, 33].

The primary antihypertensive mechanism of β-blockers is complex and involves a reduction in cardiac output due to their negative chronotropic and inotropic effects. This leads to a decreased myocardial oxygen demand. In addition, β-blockers inhibit the release of renin from the juxtaglomerular apparatus of the kidneys, thereby contributing to a reduction in arterial blood pressure [16, 39].

For patients with symptomatic chronic heart failure, β-blockers are critically important and have a Class I recommendation supported by strong evidence. Their value for this patient cohort is also considered high from an economic perspective. The mechanism of action of β-blockers in heart failure therapy is based on counteracting the harmful effects of chronic activation of the sympathetic nervous system, which represents a compensatory yet maladaptive response to reduced cardiac output. By blocking β_1-adrenergic receptors (mainly in the heart) and β_2-adrenergic receptors (in the vasculature), β-blockers prevent the cardiotoxic effects of elevated catecholamine levels [14, 29].

In arrhythmias, the use of β-blockers is important when sympathetic tone is increased. They reduce triggered activity and prevent re-entry mechanisms that may be provoked by high adrenergic activity, commonly observed during stress, physical exertion, or acute ischemia. The primary mechanism by which β-blockers control atrial fibrillation involves reducing sympathetic tone, which leads to slowed conduction through the atrioventricular (AV) node. By blocking β_1-adrenergic receptors in the AV node, β-blockers prolong its refractory period, effectively decreasing the number of rapid atrial impulses reaching the ventricles and thereby lowering the ventricular heart rate [12, 37].

Although the prognostic benefit of β-blockers in certain subgroups of patients with heart failure and atrial fibrillation has been a subject of debate, most studies continue to support their favorable impact on prognosis. The choice of a specific β-blocker in various clinical settings should be based on evidence of its efficacy in reducing mortality. This prompted an analysis of contemporary scientific literature to determine the role of the most commonly used β-blockers in modern family medicine practice [2, 41].

Objective of the work – to analyze and evaluate the role of β-blockers in modern clinical practice of the family physician.

Materials and methods

A retrospective analysis of literature sources from 2014 to 2024 on this topic was conducted using the scientometric databases PubMed, Web of Science, and Scopus. To identify information regarding the role of β-blockers in modern clinical practice, various combinations of the following keywords were used: «β-blockers», «absolute and relative contraindications», «clinical efficacy», «safety profile», «Atenolol», «Betaxolol», «Bisoprolol», «Esmolol», «Carvedilol», «Labetalol», «Metoprolol», «Nebivolol», «Propranolol», and «Sotalol». During the material processing stage, the most relevant primary sources were selected. The main selection criteria included the authority of the journal and the date of publication. Through the review of abstracts and full-text articles, 45 sources were selected for further analysis. A detailed analysis of the selected original articles related to the studied issue is presented below.

Results. Discussion

The clinical efficacy and safety profile of β-blockers largely depend on their pharmacological properties. It is important for family physicians to distinguish these drugs based on their cardioselectivity, lipophilicity, and additional pharmacodynamic activities.

Cardioselectivity (from *cardio* — heart, and *selective* — specific) refers to the selective action of a drug predominantly on β_1-adrenergic receptors in the heart, with minimal effect on β_2-adrenergic receptors located in the bronchi, blood vessels, and uterus. Cardioselective β-blockers (e.g., Bisoprolol, Metoprolol) are the preferred choice for patients with concomitant chronic obstructive pulmonary disease (COPD) or diabetes mellitus.

It should be noted that cardioselectivity is relative — at higher doses, even these agents may lose their selectivity and begin to affect β_2-receptors as well.

Cardioselective β_1-blockers:

- ↓ heart rate (*negative chronotropic effect*);
- ↓ myocardial contractility (*negative inotropic effect*);
- ↓ myocardial oxygen demand;
- ↓ blood pressure through reduced cardiac output and inhibition of the renin –angiotensin system [42, 45].

According to their lipophilicity, β-blockers are classified as lipophilic, hydrophilic, or amphophilic. Lipophilic β-blockers (e.g., Propranolol, Metoprolol, Carvedilol) are fat-soluble, easily penetrate cell membranes — including the blood–brain barrier — and are primarily metabolized and eliminated by the liver. Hydrophilic β-blockers (e.g., Atenolol, Sotalol) are water-soluble, have limited penetration into the central nervous system, and are mainly excreted by the kidneys. Amphophilic β-blockers (e.g., Bisoprolol, Betaxolol) possess moderate solubility in both lipids and water. Consequently, they are well absorbed from the gastrointestinal tract, partially metabolized in the liver, and partially excreted by the kidneys. Clinical considerations regarding lipophilicity are as follows: in hepatic impairment, hydrophilic β-blockers (e.g., Atenolol, Sotalol) are preferred. In renal impairment, lipophilic β-blockers (e.g., Metoprolol, Carvedilol) are more appropriate. In patients with depression, insomnia, or fatigue, lipophilic β-blockers should be avoided [11, 17].

The vasodilatory activity of β-adrenoblockers is an important property of newer generations of these drugs, which not only reduce heart rate and blood pressure but also dilate blood vessels, thereby decreasing peripheral resistance. Classic β-blockers (e.g., propranolol, atenolol) constrict blood vessels (through β_2-receptor blockade), which can lead to peripheral ischemia, cold extremities, and increased afterload.

New β-blockers possess additional vasodilatory effects that improve hemodynamics and reduce adverse effects. This provides an additional advantage in the treatment of concomitant arterial hypertension or peripheral vascular disease and contributes to better hemodynamic tolerance in chronic heart failure (CHF).

Several mechanisms underlie vasodilation.

- Direct blockade of vasoconstrictive α_1-adrenoceptors leads to dilation of arteries and veins (carvedilol, labetalol).
- Stimulation of nitric oxide (NO) synthesis in the endothelium causes vasodilation (nebivolol).
- Partial stimulation of β_2-receptors results in vascular relaxation (celiprolol).
- Blockade of Ca^{2+} influx into cells causes relaxation of vascular smooth muscle and membrane-stabilizing effects (betaxolol) [20, 38].

Intrinsic sympathomimetic activity (ISA) is the ability of certain β-adrenoblockers to partially stimulate β-adrenoceptors while simultaneously blocking them from the action of endogenous catecholamines (adrenaline, noradrenaline). In other words, the drug is not a «pure antagonist» but acts as a partial agonist.

Partial stimulation of β-receptors leads to a moderate reduction in heart rate while maintaining baseline cardiac tone. Such β-blockers are generally better tolerated and do not cause severe bradycardia or vasospasm.

The clinical significance of using β-blockers with intrinsic sympathomimetic activity includes:

- Arterial hypertension in patients with bradycardia (pindolol);
- Arterial hypertension in elderly patients (acebutolol);
- Patients with COPD or asthma (celiprolol — has vasodilatory properties and reduces bronchospasm).

However, such β-blockers are not recommended in cases of:

- Heart failure (they may weaken the therapeutic effect);
- Ischemic heart disease without tachycardia (less pronounced decrease in heart rate and myocardial oxygen demand);
- Post-infarction period (reduced cardioprotective effect) [26, 32].

Atenolol is a synthetic β_1-selective adrenoblocker of the second generation that exerts antagonism toward adrenoceptors predominantly located in the myocardium. A key feature of atenolol, of particular relevance to family physicians, is its pharmacokinetic profile. It undergoes minimal hepatic metabolism and is primarily excreted via the kidneys, which necessitates careful monitoring and dosage adjustment in patients with impaired renal function.

Atenolol is approved by the U.S. Food and Drug Administration (FDA) for the treatment of arterial hypertension, angina pectoris, and acute myocardial infarction. It is also used in the management of endocrine disorders such as thyrotoxicosis, where it effectively controls symptomatic tachycardia and tremor.

The elimination half-life ($T^1/_2$) is 6–9 hours. The initial dose is 25–50 mg once daily, and the maintenance dose is 50–100 mg once daily [4, 7].

Betaxolol is a cardioselective adrenoceptor blocker. The drug is approved by the U.S. Food and Drug Administration (FDA) and is available in two formulations: oral tablets (10 mg and 20 mg) and an ophthalmic solution (0.5%). Indications for use include arterial hypertension, stable angina pectoris, and secondary prevention after myocardial infarction. Its ophthalmic formulation is used to reduce intraocular pressure in open-angle glaucoma. Betaxolol has high bioavailability (approximately 90%) and a long elimination half-life ($T^1/_2$ up to 20 hours), which allows for once-daily dosing. This dosing regimen is an important factor in improving patient adherence to long-term therapy. One of the most significant advantages of betaxolol is its favorable central nervous system profile. This property is clinically valuable, particularly in geriatric populations, where patients often experience comorbid depressive disorders and sleep disturbances. The initial dose is 5 mg once daily, preferably in the morning, and the maintenance dose is 10–20 mg once daily [1, 15].

Bisoprolol stands out among β-adrenoblockers for its high cardioselectivity. Its pharmacological profile ensures targeted action on β_1-receptors, which are primarily located in the heart, thereby minimizing the impact on β_2-receptors, which could otherwise cause undesirable effects in the bronchial system or peripheral vessels. Bisoprolol remains one of the three β-adrenoblockers recommended as an essential component of pharmacotherapy for patients with heart failure (HF). Current clinical guidelines clearly state that bisoprolol, along with metoprolol succinate and carvedilol, represents the preferred choice among β-blockers for this patient population. Its use is recommended to reduce both overall mortality and the rate of hospitalizations. The therapeutic dose range of bisoprolol is 2.5–10 mg once daily. It has an elimination half-life of approximately 10–12 hours in healthy adults. The prescription of bisoprolol for arterial hypertension (AH) should be clearly justified by concomitant indications, such as heart rate control in atrial fibrillation or management of coexisting ischemic heart disease (IHD). When administering bisoprolol, slow and gradual dose titration is essential, as this approach improves patient tolerance and, consequently, adherence to therapy [8, 30].

Esmolol is an ultrashort-acting, selective β-adrenoblocker designed for intravenous administration in situations requiring rapid control of heart rate or blood pressure. Its pharmacological profile is characterized by predominant selectivity for β_1-adrenergic receptors, which are primarily located in the myocardium. With increasing doses, esmolol begins to inhibit β_2-receptors. This dose-dependent loss of cardioselectivity is clinically important, as it increases the risk of adverse effects such as bronchospasm or exacerbation of peripheral circulatory disorders. Esmolol has a short distribution half-life of approximately 2 minutes and an elimination half-life of about 9 minutes. Esmolol hydrochloride is administered exclusively intravenously. Due to its rapid kinetics, plasma concentrations can be quickly adjusted by modifying the infusion rate, allowing for precise hemodynamic control. The drug is available in ready-to-use iso-osmotic solutions, including 10 mg/mL formulations in 10 mL, 50 mL, and 250 mL vials. All administration protocols require careful dose titration with continuous monitoring of vital signs [31, 36].

Carvedilol is a unique representative of the β-adrenoblocker class due to its broad spectrum of action. It is a non-selective β-adrenoblocker that affects both β_1- and β_2-adrenoceptors, while also exhibiting significant additional α_1-blocking activity. The presence of α_1-blockade leads to peripheral vasodilation and consequently to a more pronounced reduction in peripheral vascular resistance and arterial pressure compared with purely cardioselective agents such as bisoprolol or metoprolol. In family medicine practice, an important aspect of carvedilol therapy is its pharmacokinetic profile, particularly the need to take the drug with food. Failure to follow this simple instruction may result in intolerance symptoms, which could lead to therapy discontinuation. Carvedilol is a racemate, with enantiomers that differ in both activity and metabolism. The S-enantiomer blocks both α_1- and β-adrenoceptors, whereas the R-enantiomer blocks only α_1-adrenoceptors. Their elimination half-lives also differ: approximately 5–9 hours for R-carvedilol and 7–11 hours for S-carvedilol. In family practice, carvedilol is considered an appropriate choice for patients with arterial hypertension (AH), particularly in those with diabetic cardiomyopathy, given its favorable metabolic profile. The initial dose for hypertension is typically 6.25 mg once daily, while the maximum daily dose usually does not exceed 25 mg [27, 34].

Labetalol is a combined adrenoblocker distinguished by its dual mechanism of action, as it blocks α_1-, β_1-, and β_2-adrenoceptors (the latter two non-selectively). The α-to-β activity ratio of approximately 1:7 defines its specific hemodynamic profile, which is particularly important for its current clinical applications. Blockade of β_1-adrenoceptors

produces the classical effects of β-blockade — reduced heart rate and decreased myocardial contractility (negative chronotropic and inotropic effects) — as well as suppression of renin release. However, the presence of α_1-blocking activity is a key advantage, as it leads to peripheral vasodilation and reduction of total peripheral vascular resistance. This dual mechanism allows labetalol to effectively lower arterial pressure without causing reflex tachycardia, a common issue with pure vasodilators, while also preventing the increase in peripheral resistance typically seen with non-selective β-blockers lacking α_1-blocking properties. The clinical applications of labetalol, where its unique pharmacology is most valuable, include hypertensive crisis and the management of hypertension associated with pregnancy, including gestational hypertension and preeclampsia. Its pharmacological profile establishes labetalol as an important, though specialized, option in emergency and obstetric settings. Family physicians should be aware of common drug interactions that can influence labetalol's effectiveness: long-term use of nonsteroidal anti-inflammatory drugs (NSAIDs) such as indomethacin or ibuprofen may reduce its antihypertensive effect through pharmacodynamic antagonism. Conversely, concomitant use with certain antidepressants, nitrates, or other antihypertensive agents may lead to excessive hypotension. The initial dose for adults, including pregnant patients, is typically 100 mg twice daily. The usual total daily dose for most adults ranges from 400 to 800 mg, divided into two doses. Doses above 1200 mg per day are rarely required in outpatient practice [6, 23].

Metoprolol is one of the most widely used β-adrenoblockers in cardiology and primary care. Its therapeutic efficacy is based on selective inhibition of β_1-adrenergic receptors, leading to reduced myocardial contractility, slowed heart rate (HR), and lowered arterial pressure (AP). Consequently, metoprolol remains a key drug for the treatment of various cardiovascular conditions, including arterial hypertension (AH), angina pectoris, chronic heart failure (CHF), and various types of arrhythmias. The clinical role of metoprolol is closely tied to its pharmacokinetic profile, which differs depending on the salt form. Two main formulations are available: metoprolol tartrate (immediate-release) and metoprolol succinate (extended-release). For the treatment of chronic cardiac conditions such as CHF or AH, the extended-release formulation is not merely a matter of convenience but a therapeutic necessity. Mortality reduction studies in CHF were conducted exclusively with metoprolol succinate. Insufficient blockade caused by tartrate may compromise the patient's long-term prognosis. Moreover, the once-daily dosing regimen of the succinate form significantly improves patient adherence. Therefore, when prescribing metoprolol for long-term disease management, family physicians should always select and explicitly indicate metoprolol succinate. The usual initial dose of metoprolol succinate is 25–100 mg per day, and the maintenance dose ranges from 100 to 400 mg per day [43, 44].

Nebivolol is a modern, third-generation cardioselective β-adrenoblocker. Its pharmacological uniqueness is based on its chemical structure as a racemate, consisting of two different enantiomers: R-nebivolol (D-nebivolol) and S-nebivolol (L-nebivolol). Each enantiomer has a specific pharmacological profile. The D-enantiomer is responsible for the primary mechanism of action, providing competitive and highly selective β_1-adrenoceptor blockade. This effect underlies nebivolol's ability to reduce heart rate and myocardial contractility. The L-enantiomer, in contrast, is responsible for mild vasodilatory properties. This vasodilatory effect is mediated via metabolic interaction with the L-arginine/nitric oxide (NO) system. This dual mechanism of action distinguishes nebivolol from classical β-blockers, which lack NO-mediated vasodilatory activity. Nebivolol is an effective agent for the treatment of arterial hypertension. For most adult patients, the recommended dose is 5 mg once daily. The tablet is taken orally, regardless of food intake, preferably at the same time each day to maintain a stable plasma concentration [10, 13].

Propranolol is a synthetic β-adrenoblocker that was historically the first drug developed for the treatment of angina pectoris, representing a major breakthrough in cardiology. It is considered a prototype against which all other β-blockers are compared. Pharmacologically, propranolol is a non-selective competitive antagonist of β-adrenergic receptors and importantly, it lacks intrinsic sympathomimetic activity. Propranolol is rapidly absorbed after oral administration, reaching peak plasma concentration (Cmax) in approximately 2 hours. Clinically significant is its extensive first-pass metabolism in the liver, and taking the drug with food may increase its bioavailability, which is a practical recommendation for achieving more stable and predictable plasma concentrations. In patients with severe hepatic impairment, such as cirrhosis, propranolol metabolism and elimination are significantly impaired. Studies show that in these patients, the elimination half-life increases to about 11 hours, compared with 4 hours in healthy individuals. Propranolol is included in the first-line list of evidence-based medications for the prevention of episodic migraine. Prophylactic therapy is recommended when a patient experiences four or more migraine attacks per month or eight or more headache days per month. The recommended daily dose range for migraine prophylaxis is 80–240 mg per day. Additionally, propranolol is used to manage symptoms of social anxiety disorder, particularly performance anxiety, by reducing the physical manifestations of sympathetic activation such as tachycardia, sweating, and tremor. In endocrinology, it is employed to control symptoms of thyrotoxicosis. Due to its non-selective mechanism of action, propranolol carries significant risks, requiring strict adherence to contraindications and continuous patient monitoring [21, 40].

Sotalol is a unique drug that combines the properties of a non-selective β-adrenoblocker with a potent potassium channel-blocking effect. Its clinical antiarrhythmic effect is manifested through prolongation of the action potential and the effective refractory period, which makes it effective in suppressing and preventing supraventricular and ventricular ar-

rhythmias. The main approved indications for sotalol include maintenance of sinus rhythm in patients with paroxysmal supraventricular tachycardia and paroxysmal atrial fibrillation, as well as the treatment of hemodynamically stable ventricular tachycardia. Treatment typically starts at 80 mg per day (administered as 40 mg twice daily with a 12-hour interval between doses). In some patients, the daily dose may need to be increased to 160–320 mg, also divided into two doses. Structural heart disease is the main risk factor for proarrhythmia, which complicates the use of sotalol in patients with heart failure. The most serious adverse effect of sotalol is QT interval prolongation, which increases the risk of Torsade de Pointes, necessitating strict adherence to safety monitoring protocols. Sotalol is safest in patients with minimal or no structural heart disease [3, 18].

Table 1. Properties of β-Adrenergic Blockers.

Medication	Cardioselectivity	Elimination half-life	Initial dose
Atenolol	Yes	6-9 hours	25–50 mg once daily
Betaxolol	Yes	20 hours	5 mg per day
Bisoprolol	Yes	10-12 hours	2.5 mg once daily
Esmolol	Yes	9 minutes	500 mcg/kg intravenously
Carvedilol	No	5-9 hours	6.25 mg per day
Labetalol	No	5-8 hours	100 mg per day
Metoprolol	Yes	3-7 hours	25–50 mg per day
Nebivolol	Yes	10-12 hours	5 mg once daily
Propranolol	No	3-6 hours	10–40 mg
Sotalol	No	10-20 hours	40 mg twice daily

Absolute and relative contraindications for β-adrenoblockers (β-blockers). Family physicians should be well aware of conditions in which β-blockers are absolutely contraindicated: cardiogenic shock; decompensated chronic heart failure (CHF) – therapy should be discontinued; severe bradycardia (HR < 50 bpm)*;* second- or third-degree atrioventricular (AV) block; sick sinus syndrome (without a pacemaker); bronchial asthma; atrial fibrillation in the context of Wolff–Parkinson–White (WPW) syndrome. Relative contraindications include: chronic obstructive pulmonary disease (COPD) (requires use of highly selective β-blockers); diabetes mellitus (risk of masking hypoglycemia symptoms, necessitating patient education and careful monitoring); Raynaud's disease or other peripheral vascular disorders (condition may worsen with non-selective β-blockers) [24, 25].

Poisoning with β-adrenoblockers is a potentially life-threatening condition that requires immediate intervention. The family physician must be familiar with the primary measures for stabilizing the patient's condition. The most severe cases of poisoning are caused by non-cardioselective β-blockers without intrinsic sympathomimetic activity, particularly those with membrane-stabilizing properties, primarily Propranolol. Sotalol is also dangerous, as being a Class III antiarrhythmic, it carries a risk of QTc interval prolongation and the development of Torsade de Pointes-type tachycardia. The classical triad of β-blocker poisoning includes: hypotension, bradycardia, cardiogenic shock. Characteristic ECG findings include: bradycardia, QTc prolongation (especially with Sotalol), widened QRS complex (particularly with β-blockers possessing membrane-stabilizing activity). It is also important to remember that hypoglycemia is a typical feature of β-blocker poisoning and requires immediate correction. Before hospitalization and the provision of specialized care, the family physician should: establish reliable intravenous access and initiate adequate crystalloid infusion therapy in cases of hypotension; administer glucagon as a specific antidote; use vasopressors (such as epinephrine or dopamine) in cases of refractory hypotension [9, 35].

β-Blocker withdrawal syndrome is critically important, so sudden discontinuation of β-blockers is strictly prohibited. The withdrawal syndrome manifests as: rebound increase in arterial blood pressure (BP), tachycardia, in patients with ischemic heart disease (IHD) — it may lead to exacerbation of angina attacks. Discontinuation should occur by gradually tapering the dose over several weeks [22].

Thus, the modern role of β-adrenoblockers in family medicine practice is based not only on traditional indications, but also on the integration of new evidence regarding their efficacy and the need for careful management of comorbidities. A key factor is the selection of the drug considering its pharmacological profile, which ensures both effectiveness and safety.

Conclusion and prospects for further developments

1. β-Adrenoblockers remain an important component of therapy for cardiovascular diseases such as ischemic heart disease, chronic heart failure, arterial hypertension, and arrhythmias due to their proven ability to reduce mortality and the risk of complications.

2. The efficacy and safety of β-adrenoblockers are determined by their pharmacological properties and the presence of intrinsic sympathomimetic activity, which requires an individualized selection of the drug depending on the patient's comorbid conditions.

3. In family medicine practice, it is important to adhere to the principles of rational pharmacotherapy: carefully consider the indications and contraindications for prescribing, avoid β-blocker withdrawal syndrome, and implement gradual dose titration to ensure an optimal clinical outcome.

The relevance of future research lies in the development of unified protocols and international guidelines on the use of β-blockers in the treatment of hypertension and coronary heart disease, particularly in the practice of internists and family physicians.

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МІСЦЕ β-АДРЕНОБЛОКАТОРІВ У СУЧАСНІЙ КЛІНІЧНІЙ ПРАКТИЦІ СІМЕЙНОГО ЛІКАРЯ

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Анотація. *Оглядова стаття присвячена сучасній ролі β-адреноблокаторів у практиці сімейного лікаря. Ці препарати залишаються ключовими засобами лікування хворих на ішемічну хворобу серця, артеріальну гіпертензію, хронічну серцеву недостатність та аритмії. У статті підкреслена необхідність індивідуалізованого підходу до вибору β-адреноблокаторів з урахуванням фармакологічних властивостей, коморбідних станів і профілю безпеки. Мета роботи – проаналізувати й оцінити місце β-адреноблокаторів у сучасній клінічній практиці сімейного лікаря. Ретроспективний аналіз літературних джерел з даної проблематики виконано на основі наукометричних баз даних PubMed, Web of Science та Scopus. На етапі опрацювання матеріалів здійснювався відбір найбільш релевантних першоджерел. Основними критеріями відбору були авторитетність видання та дата публікації. Шляхом вивчення анотацій та повних текстів статей, було відібрано 45 джерел для подальшого аналізу. Для пацієнтів із хронічною серцевою недостатністю β-адреноблокатори є життєво необхідним компонентом лікування, що знижує смертність. Важливими критеріями вибору препарату є кардіоселективність, ліпофільність, наявність вазодилатуючої дії та внутрішня симпатоміметична активність. Нові покоління препаратів мають додаткову вазодилатуючу дію, що покращує гемодинаміку. Сімейний лікар повинен враховувати особливості фармакокінетики при патології нирок і печінки та уникати раптового припинення терапії. Наведено характеристику основних представників цього класу препаратів з детальним описом їхніх клінічних переваг, дозувань і показань. У статті окремо розглянуто абсолютні й відносні протипоказання до застосування β-адреноблокаторів, а також тактику ведення при передозуванні. Отже β-адреноблокатори залишаються важливим компонентом терапії таких серцево-судинних захворювань як ішемічна хвороба серця, хронічна серцева недостатність, артеріальна гіпертензія та аритмії завдяки доведеній здатності знижувати смертність і ризик ускладнень. Ефективність і безпечність застосування β-адреноблокаторів визначаються їх фармакологічними властивостями та наявністю внутрішньої симпатоміметичної дії, що вимагає індивідуалізованого вибору препарату залежно від супутньої*

патології. У практиці сімейного лікаря важливо дотримуватись принципів раціональної фармакотерапії: ретельно враховувати показання й протипоказання до призначення, уникати синдрому відміни та здійснювати поступову титрацію дози, що забезпечує оптимальний клінічний результат.
Ключові слова: *артеріальна гіпертензія, β-адреноблокатори, ішемічна хвороба серця.*

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