International Journal of Scientific Trends- (IJST)
ISSN: 2980-4299
Volume 4, Issue 10, October - 2025
Website: https://scientifictrends.org/index.php/ijst
Open Access, Peer Reviewed, Scientific Journal

Risk of Developing Heart Rhythm Disturbances in Arterial Hypertension

Nuritdinova Nigora Batirovna
Internal Diseases and Department of Preventive Medicine Fundamentals
Associate Professor, Tashkent Medical Academy. Tashkent, 100109, Uzbekistan
nuritdinova.nigora@yandex.com
https://orcid.org/0000-0001-7236-1639

Shukurjanova Surayo Makhmudovna
Internal Diseases and Department of Preventive Medicine Fundamentals
Associate Professor, Tashkent Medical Academy. Tashkent, 100109, Uzbekistan
surayyo.vop@gmail.com
https://orcid.org/0000-0002-3989-1985

Khasanova Nelya Alibaevna
Internal Diseases and Department of Preventive Medicine Fundamentals
Specialty Cardiology 1st Year Master's Student Tashkent Medical Academy
Tashkent, 100109, Uzbekistan
nilyaxasanova1234@gmail.com

Abstract

This article highlights the problem of arterial hypertension from a scientific, theoretical and practical point of view. Arterial hypertension is diagnosed by daily blood pressure monitoring. With arterial hypertension, there is a frequent violation of the heart rhythm. The importance of Holter ECG monitoring in arterial hypertension is considered. With arterial hypertension, it is necessary to carry out daily monitoring of blood pressure to diagnose, determine the type of hypertension, and evaluate the effectiveness of hypertension treatment. In case of arterial hypertension accompanied by rhythm disturbances, Holter ECG monitoring is prescribed.

Keywords: Arterial hypertension, diagnosis, daily blood pressure monitoring, Holter ECG monitoring, cardiac arrhythmia.

Introduction

Arterial hypertension (AH) is one of the most common risk factors for cardiovascular diseases. About 45% of the world's population suffers from arterial hypertension, and the incidence of this disease increases with age[1]. According to the World Health Organization, about 1.28 billion

people worldwide have hypertension, of which about 46% are unaware of their condition[2]. In 2019, AH caused 10.8 million deaths, accounting for 19% of global deaths[3]. In Africa and South Asia, AH was at the highest level, exceeding 40% of the total population [3,4]. In developed European countries, the prevalence is relatively lower (25-30%), but with improper nutrition and lifestyle changes, high rates are noted in some regions [5]. Due to the well-developed treatment and diagnostic system in North America and Australia, mortality and morbidity rates associated with AH are decreasing [6]. According to the results of the Global Burden of Disease (GBD) study, mortality and disability associated with high blood pressure ranked high, and in 2019 AH was the main cause of death associated with cardiovascular diseases, stroke, and renal failure [7]. According to the Ministry of Health of Uzbekistan, the number of people with AH as the main cause of cardiovascular diseases in 2012 exceeded 1 million [8]. Therefore, the solution to the problem of early diagnosis of patients with AH, prevention and treatment of cardiac rhythm disorders, which are one of the most common complications caused by it, remains the most urgent among the tasks of modern cardiology. Cardiovascular diseases account for about 60% of deaths in the country, one of the main causes of which is arterial hypertension [9]. According to reports from republican medical centers, arterial hypertension was detected in 28-32% of cases among adults [10]. AH can affect the electrical activity of the heart, leading to the development of various arrhythmias. Studies conducted in various countries of the world, studying the relationship between AH and arrhythmias, have led to the following conclusions. In the Framingham study conducted in the USA, the risk of developing atrial fibrillation in patients with AH increases by 1.5-2 times. Among patients with AH, atrial fibrillation accounts for 14-20% [11]. In the Hysayama study in Japan, the risk of developing atrial fibrillation among the adult population with AH was 10% [12]. In the epidemiological study in the Russian Federation, the incidence of arrhythmias among patients with AH was 15-18% [13]. In addition, ventricular extrasystoles account for 10-15%. Ventricular tachycardia occurs in 5-10% of patients [14, 15].

Daily blood pressure is characterized by fluctuations in pressure over 24 hours. The daily (daily) rhythm of blood pressure changes in accordance with the circadian biological rhythm of a person. Usually, in healthy people, blood pressure varies depending on different times of the day, physical activity, and the level of stress [16]. However, in patients with arterial hypertension, these fluctuations can be more pronounced or have a pathological character [17].

Normally, blood pressure increases in the morning, i.e., during daytime activity, and decreases at night, during sleep [18]. Based on daily blood pressure monitoring (DABM), several types of blood pressure changes are distinguished:

1. Normal blood pressure decreases by 10-20% at night (this condition is called "dipping"). In the morning hours (06:00-8:00), blood pressure rises again and remains high throughout the day. In this type of person, a physiological decrease in blood pressure is observed at night. Usually, the decrease is 10%-20% of the daily values. This type occurs in approximately 50-60% of people without serious cardiovascular diseases [17].

This type is less common in patients with arterial hypertension, which is associated with disruptions in the mechanisms of the circadian rhythm of pressure [17,18].

Deviations:

1. Non-dipper type: Blood pressure drops little at night (less than 10%). This type occurs in 30-40% of patients with AH. Significantly increases the risk of cardiovascular complications, stroke, and CHF. In patients of this type, the daily fluctuations in pressure are significantly reduced. The nighttime blood pressure drop is less than 10% of the daytime values. This type is common in diabetes mellitus, chronic kidney disease, and elderly people.
2. Over-dipper type: excessive dipper (decrease by more than 20%): characterized by an excessive drop in blood pressure at night (more than 20% of daytime values). Even in healthy people of this type, approximately 10% of patients with hypertension have the extreme dipper type. In some cases, excessive nighttime pressure drops can lead to impaired organ perfusion, especially in elderly patients. Excessive pressure reduction increases the risk of cerebrovascular disorders and dizziness.
3. Night-peaker type - nighttime elevation of blood pressure: this type of blood pressure does not decrease at night, but, on the contrary, increases compared to daytime values. This is the most dangerous type, occurring in 5-10% of patients with hypertension and associated with a high risk of stroke, cardiovascular diseases, and heart failure [19]. It is more common in patients with impaired renal function, obstructive sleep apnea syndrome, and severe forms of hypertension [20]. This type increases the risk of death associated with hypertension by 2-3 times [21].

Outpatient blood pressure monitoring is an important method for identifying patients with various types of daily blood pressure fluctuations. Studies show that serious cardiovascular complications are more common in patients with non-dipper and night peaker types [22]. Night-peaker type is especially dangerous: according to clinical studies, stroke and myocardial infarction occur 25-30% more often in patients of this type than in patients of the dipper type. The non-dipper type is often associated with metabolic syndrome, chronic kidney disease, and increased vascular stiffness, which increases the risk of cardiovascular events by 15-20% [23]. Thus, 24-hour blood pressure monitoring and analysis of its types for patients with arterial hypertension plays an important role in predicting complications and individualizing therapy. Examination of patients for the purpose of improving the quality of life of patients with hypertension, thereby reducing mortality, includes ECG and BP measurement, but the diagnostic value of this method for detecting arrhythmias is low.

The most informative method for studying the arrhythmogenic activity of the myocardium is conducting 24-hour ECG monitoring (Holter-ECG) and assessing its profile, identifying significant factors influencing arterial blood pressure and rhythm disturbances with 24-hour blood pressure monitoring (HBM) [24]. Arrhythmias are often associated with prolonged arterial hypertension. Arrhythmias are a common problem in patients with hypertension. The presence and complexity of supraventricular and ventricular arrhythmias can affect morbidity, mortality, as well as the quality of life of patients [25]. Effective treatment of arterial hypertension plays an important role in the prevention of structural and functional disorders of the heart. If arterial hypertension is suspected during screening examinations, the diagnosis of hypertension is recommended by repeatedly measuring blood pressure in the office during several visits or 24-hour off-office blood pressure monitoring using 24-hour NSAIDs and 24-hour ECG Holter monitoring to detect and assess heart rhythm disturbances in all patients with hypertension [26]. A significant correlation was shown between arterial hypertension, left ventricular hypertrophy,

impaired left ventricular diastolic filling, increased left atrial pressure, left atrial hypertrophy and dilation, increased atrial fibrosis, increased ectopic activity in the atrium, and arrhythmia with a slowdown in the rate of intra- and interatrial electrical conduction [27]. The authors, who studied the problem of disorders of automaticity and conduction functions in patients with arterial hypertension, came to the conclusion that supraventricular extrasystoles (SVE), ventricular extrasystoles (VE) are very common in patients with hypertension, supraventricular tachycardia (SVT), atrial fibrillation (BP, its paroxysmal form) and blockades (atrioventricular and intraventricular) [28]. Ventricular arrhythmias were detected in 62% of the examined patients with hypertension, with high-grade arrhythmias according to Lown in 50% of patients. Studies characterizing the relationship between daily BP monitoring and cardiac arrhythmias in patients with hypertension are rare, and the influence of variable BP on the types and frequency of arrhythmias is not clear [29].

The main risk factors for supraventricular, ventricular arrhythmias, and sudden death in patients with arterial hypertension are left ventricular diastolic dysfunction, left atrial volume and function, and left ventricular hypertrophy (LVH) [1-5, 30]. Left ventricular hypertrophy is common in patients with risk factors for cardiovascular diseases. Systemic arterial hypertension is the most common cause of left ventricular hypertrophy [31]. In patients with arterial hypertension and left ventricular hypertrophy, ECG and echocardiographic criteria [32], and complex ventricular arrhythmias are more common. In such patients with LVH, the risk of myocardial infarction and sudden death increases [32, 33]. According to the literature, there is no correlation between the level of arterial pressure and the mass of the left ventricular myocardium in the occurrence of ischemic episodes. According to S. Scheler et al., there is no difference in left ventricular myocardial mass and blood pressure levels in patients with arterial hypertension with or without ischemic episodes. They note that left ventricular myocardial mass does not affect the occurrence of ST-segment depression. According to the authors, the main factors leading to a decrease in the ST segment are circulatory function and structural disorders in left ventricular myocardial hypertrophy. In patients with asymptomatic left ventricular hypertrophy, MV is of questionable significance, since the risk of sudden death is uncertain in these patients with complex or frequent ventricular arrhythmias. If differences in age, sex, and other clinical factors are taken into account (relative risk 1.62; 95% confidence interval 0.98-2.68) LVH is associated with both ventricular and supraventricular arrhythmias [34]. In published studies, there was a strong correlation between left ventricular hypertrophy and left ventricular diastolic dysfunction. Also, the link between left ventricular hypertrophy and the arrhythmia index was even closer. Arrhythmogenic risk factors in patients with hypertension included late ventricular potentials, decreased heart rate variability, changes in the duration of the QRS complex and QT interval, and changes in the T wave. Assessing the risk of arrhythmia in asymptomatic patients is a difficult task. From a clinical point of view, the relationship between LVH and sudden arrhythmogenic death is well known; an important predictor of cardiovascular death and sudden arrhythmogenic death is its geometry relative to the mass of the left ventricle [34, 35].

An increase in the number of early supraventricular extrasystoles is known in arterial hypertension. Uncontrolled hypertension, worsening diastolic dysfunction, LVH, left atrial dilation, increased sympathetic activity, caffeine and alcohol abuse, smoking, and electrolyte imbalance are all factors associated with supraventricular ES. Chatterjee S. et al. in a published

meta-analysis of 10 controlled studies involving 27,141 patients reported that the frequency of supraventricular tachycardia in patients with LVH was 11.1% versus 1.1% in patients without LVH. The probability of developing supraventricular tachycardia in patients with LVH was 3.4 times higher than in patients without LVH. The frequency of ventricular arrhythmias was 1.2% versus 5.5% in patients without LVH [35]. The presence of LVH in AH patients is associated with a greater risk of persistent supraventricular and ventricular arrhythmias [36]. In addition, various types of paroxysmal supraventricular tachycardia, which can occur in patients with arterial hypertension and, most importantly, can lead to severe symptoms or even acute pulmonary edema, lead to diastolic/systolic dysfunction [34,10-14]. A 10-year observation of 253 patients with initially uncomplicated arterial hypertension confirmed that the frequency of cardiovascular complications and mortality is strictly dependent on the geometric model of the left ventricle (LV). The worst prognosis for TFA (31%) and mortality (21%) was noted in the group of patients with concentric LVH. The most optimal prognosis was fatal outcomes in the group of patients with normal LV geometry and the absence of CHD by 11% [35]. Differences in the structural-geometric model of the LV in patients with hypertension are closely related to the pathophysiology of the heart and blood circulation [37]. In patients with concentric LVH, normal end-systolic myocardial stress is characterized by an increase in normal LV volume and shape, total peripheral vascular resistance (TPVC), and a slight increase in the cardiac index (CI). Patients with concentric remodeling are also characterized by a normal level of end-systolic myocardial stress and an increase in UPVC. At the same time, a decrease in the stroke index (SI) and heart rate is observed in them. In part, this increase can be explained by a decrease in the elasticity of the arteries, which is indicated by the size of the subnormal stroke with a slight increase in HR. Patients with eccentric LVH are characterized by high HR, normal LVEF, LV expansion, and end-systolic myocardial stress. Hemodynamic prerequisites for the formation of this geometric model are the increase in venous tone or circulating blood volume. The majority of patients with hypertension have normal LV geometry and are characterized by a slight increase in BP, systolic, and diastolic pressure. As a result of an increase in hypertrophic LV pressure against the background of diastolic myocardial dysfunction, the left atrium (LV) dilates very rapidly. This leads to supraventricular extrasystoles (SVE), atrial flutter, and atrial fibrillation (AT and AF) in 25% of patients with hypertension [37]. According to the results of the Manitoba observation study, it is known that the incidence of AF in men with hypertension increases by 1.42 times [38]. Atrial fibrillation (AF) is one of the most common supraventricular arrhythmias in patients with arterial hypertension. In the presence of hypertension in men and women, the risk of developing AF increases by 1.5 and 1.4 times, respectively. With each decade, the risk of its occurrence increases [38]. Arterial hypertension is one of the risk factors for the development of such pathological conditions as atrial fibrillation (AF) and systemic thromboembolism and stroke. Combined antihypertensive therapy (using at least 3 drugs, including renin-angiotensin-aldosterone system antagonists) significantly reduces the frequency of arrhythmic episodes [31].

At the same time, it should be noted that there are practically no regular studies on the influence of blood pressure on the incidence of AF and the severity of the condition. In this regard, determining the relationship between blood pressure levels, antihypertensive therapy, and the incidence of arrhythmias is of great importance in the development of effective therapeutic regimens. The main pathophysiological mechanisms of AF associated with arterial hypertension

include hypertension-induced LVH and subsequent left atrial dilation and restructuring, while the activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system also contributes to the development of AF [32]. Arterial hypertension and AF increase the risk of thromboembolic complications and heart failure. In the last decade, atrial fibrillation has been increasingly prevalent, which is associated with population aging and increased risk factors such as arterial hypertension, obesity, dyslipidemia, smoking, diabetes mellitus, heart disease, and chronic obstructive pulmonary disease [32]. The presence of arterial hypertension in patients with AF additionally increases the risk of acute cerebrovascular accident by 2-3 times. BP increases the risk of cardiovascular diseases and mortality. In patients with arterial hypertension, the risk of developing AF increases to 42%. Effective treatment of hypertension in patients with AF can eliminate systemic changes in the heart, reduce thromboembolic complications, and delay or prevent the occurrence of atrial fibrillation [34]. Patients with LVH had supraventricular tachycardia (SVT) in 27.3% of cases ($p<0.05$), and the number of LVH was associated with LV volume [35]. Over 16 years, the observation of 2,482 patients with endocrine or other non-CVD hypertension revealed that in untreated patients with sinus rhythm hypertension, age and LVH are independent prognoses for the development of AF, and the volume of the LV is an additional risk factor for AF [34,35]. A wide spectrum of ventricular arrhythmias can manifest in patients with arterial hypertension. It has long been recognized that premature ventricular palpitations or unstable ventricular tachycardia (VTA) complicate the profile of arterial hypertension. LVH increases the risk of developing cardiovascular mortality and stroke in patients [36]. The relationship between LVH and the frequency and severity of ventricular arrhythmias depends on the degree of hypertrophy, and the presence of LVH can be the most important factor in the development of ventricular arrhythmias in hypertension. The Framingham study shows that premature ventricular contractions increase the risk of sudden death by 2.9 times in men and 1.6 times in women [26,18]. Improved control of arterial hypertension can significantly reduce mortality from cardiovascular diseases [27]. The relationship between LVH and spontaneous ventricular arrhythmia has been confirmed in well-controlled, experimental studies [16,25,34]. There is evidence that LVH regression leads to the restoration of normal electrical and structural properties of the myocardium, a decrease in the frequency and complexity of ventricular arrhythmias [4,11,21,33,38,]. Part of the risk is associated with the presence of an electrophysiological phenotype of hypertrophic myocardium associated with proarrhythmogenicity, as well as myocardial ischemia and a number of other factors [38].

Arrhythmogenic risk factors in patients with hypertension with LVH:

- Heart rate variability (HRV)
- Increased duration of action potential
- Increased variability of the QT interval (extension of the QT interval)
- T wave exchange
- Late ventricular potentials detection

The change in the amplitude of the T wave from contraction to contraction is one of the very new signs of risk of arrhythmia in patients with AMI or cardiomyopathy. The presence of microalternation of the T wave reflects the loss of synchronicity of ventricular repolarization, i.e., the electrophysiological predisposition to the occurrence of ventricular re-entry reflects the occurrence of arrhythmia [45]. It has been established that structural changes caused by

myocardial hypertrophy affect repolarization processes [13,22]. Patients with hypertension with T-wave microalternations can be identified as a high-risk group for the development of ventricular arrhythmias, especially QT torsade de pointe. Torsade de pointe ventricular arrhythmia is usually due to the frequency of ventricular contractions, the association between QT and RR intervals after a short and long RR interval, or the drug-induced increased QT/RR ratio. This phenomenon is also present in LVH. The same is observed in congenital QT elongation syndrome, and as recently discovered, it serves as a prognosis for CHD in post-infarction patients [38]. The risk of arrhythmias is closely related to the structure of the myocardium, therefore treatment that slows down remodeling also affects the restoration of the electrophysiological uniformity of the heart muscle. Antihypertensive therapy reduces LVH, prolongs repolarization time, and reduces the frequency and severity of ventricular arrhythmias and the development of cardiac arrhythmias [27,28,32].

Assessing the risk of arrhythmia in asymptomatic patients is a difficult task. After a thorough questioning and physical examination of patients, it is necessary to check the systolic and diastolic functions of the heart and assess the BMI. The next important point is the exclusion of painless myocardial ischemia - the absence of persistent or unstable ventricular tachycardia (VT), measured by Holter ECG monitoring, indicates that these patients are at very low risk. In patients with frequent HE, a decrease in ejection fraction (EF), and LVH, it is necessary to conduct a detailed examination with a study of the arrhythmogenic substrate and the profile of the autonomic nervous system [29]. For this purpose, non-invasive methods can be used: analysis of micro-T waves and changes in heart rate variability, as well as intracardial electrophysiological studies with the recorded QT.

Conclusion:

The origin of many arrhythmias is associated with prolonged arterial hypertension. The presence of structural changes in the atria and ventricles at various levels of hypertension can explain the high frequency of AF and ventricular arrhythmias in these patients. For the correct assessment of arrhythmogenic risk in patients with hypertension, Holter ECG monitoring to slow down structural changes that create an ideal substrate for myocardial electrical instability, 24-hour blood pressure monitoring are leading methods, it is necessary to select the most effective antihypertensive drugs in patients with hypertension, analyze their effectiveness. ECG monitoring in patients with hypertension contributes to the early and reliable detection of various arrhythmias, which allows for the timely prescription of correct antihypertensive and antiarrhythmic therapy.

References

1. Wold Health Organization (WHO). Global status report on noncommunicable diseases. Geneva: WHO; 2021.
2. Forouzanfar M.H., et al. Global burden of hypertension and associated risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020;396(10258):1204–1222.
3. NCD Risk Factors Collaboration (NCD-RiskC). Worldwide trends in hypertension prevalence and progress in treatment and control. Lancet. 2021;398(10299):957–980.

4. Zhou B., Perel P., Mensah G.A., Ezzati M. Global epidemiology, health burden, and effective interventions for elevated blood pressure and hypertension. Nature Reviews Cardiology. 2021;365(9455):217–223.
5. Kearney P.M., Whelton M., Reynolds K., et al. Global burden of hypertension: analysis of worldwide data. Lancet. 2005;365(9455):217–223.
6. Ezzati M., Riboli E. Behavioral and dietary risk factors for noncommunicable diseases. New England Journal of Medicine. 2013;369:954–964.s:
7. Ministry of Health of the Republic of Uzbekistan. Program for reducing cardiovascular diseases. Tashkent, 2023.
8. 8. Soliev I.B. Epidemiological studies of arterial hypertension. Medical Journal. 2022; (15): 12-18.
9. Levy D., Anderson K.M., Savage D.D., et al. Risk of atrial fibrillation in hypertensive patients: the Framingham Heart Study. New England Journal of Medicine. 1994;331(6):329–334.
10. Kiyohara Y., Kubo M., Nakagawa S., et al. Risk factors for atrial fibrillation in a general population: the Hisayama Study. Stroke. 2003;34(7):1827–1831.
11. Shlyakhto E.V., Konradi A.O. Hypertension and ventricular arrhythmias. Journal of Hypertension. 2018;36(12):2342–2350.
12. Levy D., Garrison R.J., Savage D.D., et al. Left ventricular hypertrophy and risk of ventricular tachycardia. Circulation. 1990;81(3):814–821.
13. O'Brien E., Parati G. Ambulatory blood pressure monitoring in the management of hypertension. Journal of Hypertension. 2013;31(9):1731–1768.
14. Hermida R.C., Ayala D.E., Mojon A., et al. Ambulatory blood pressure monitoring: normality and abnormalities. Hypertension Research. 2008;31(3):521–528.
15. Kario K., Pickering T.G., Umeda Y., et al. Nighttime blood pressure and cardiovascular events. Hypertension. 2003;42(3):286–292.
16. Williams B., Mancia G., Spiering W., et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. European Heart Journal. 2018;39(33):3021–3104.
17. Healey J.S., Connolly S.J. Atrial fibrillation: hypertension as a causative agent, risk factor for complications, and potential therapeutic target. Am J Cardiol. 2003;91(10A):9G–14G.
18. Palmiero P., Maiello M. Ventricular arrhythmias and left ventricular hypertrophy in essential hypertension. Minerva Cardioangiol. 2000;48(12):427–434.
19. Yiu K.H., Tse H.F. Hypertension and cardiac arrhythmias: a review of the epidemiology, pathophysiology and clinical implications. J Hum Hypertens. 2008;22:380–388.
20. Yildirir A., Batur M.K., Oto A. Hypertension and arrhythmia: blood pressure control and beyond. Europace. 2002;4(2):175–182.
21. Elias M.F., Sullivan L.M., Elias P.K., et al. Left ventricular mass, blood pressure, and lowered cognitive performance in the Framingham offspring. Hypertension. 2007;49:439.
22. Ang D., Lang C. The prognostic value of the ECG in hypertension: where are we now? J Hum Hypertens. 2008;22:460.
23. Sultana R., Sultana N., Rashid A., et al. Cardiac arrhythmias and left ventricular hypertrophy in systemic hypertension. J Ayub Med Coll Abbottabad. 2010;22(4):155–158.

24. Chatterjee S., Bavishi C., Sardar P., et al. Meta-analysis of left ventricular hypertrophy and sustained arrhythmias. Am J Cardiol. 2014;114:1049.
25. Naser N., Dilic M., Durak A., et al. The Impact of Risk Factors and Comorbidities on The Incidence of Atrial Fibrillation. Mater Sociomed. 2017;29(4):231–236.
26. Naser N., Kulic M., Dilic M., et al. The Cumulative Incidence of Stroke, Myocardial infarction, Heart Failure and Sudden Cardiac Death in Patients with Atrial Fibrillation. Med Arch. 2017;71(5):316–319.
27. Poulimenos L., et al. Cardiac arrhythmias in arterial hypertension. J Clin Hypertens. 2020;22:1371–1378.
28. Whelton P.K., Carey R.M., Aronow W.S., et al. 2017 ACC/AHA/APA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the management of high blood pressure in adults. Hypertension. 2018;71(6):1269–1324.
29. Dan G.A., Badila E., Weiss E., et al. Arterial hypertension in patients with atrial fibrillation in Europe. Int J Cardiol. 2018;254:136–141.
30. Bohm M., Reil J.C., Deedwania P., et al. Resting heart rate: risk indicator and emerging risk factor in cardiovascular disease. Am J Med. 2015;128:219–228.
31. Kolloch R., Legler U.F., Champion A., et al. Impact of resting heart rate on outcomes in hypertensive patients with coronary artery disease: findings from the INVEST study. Eur Heart J. 2008;29:1327–1334.
32. Verdecchia P., Reboldi G., Gattobigio R., et al. Atrial fibrillation in hypertension: predictors and outcome. Hypertension. 2003;41:218.
33. Miller O.N. Updated recommendations for the treatment of patients with atrial fibrillation. Methodical manual. Novosibirsk, 2013. - 75 p. 34. Ferri C., Croce G., Desideri G. Role of combination therapy in the treatment of hypertension: focus on valsartan plus amlodipine. Adv Ther. 2008;25:300–320.
34. Kovalik I., Dabrowski R., Borowiec A., et al. Combined hypotensive treatment in patients with recurrent atrial fibrillation and arterial hypertension. Kardiol Pol. 2012;70:659–666.