

Pathophysiological Mechanisms of Coronary Heart Disease and Arterial Hypertension During Aging

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ABSTRACT

Cardiovascular diseases remain the leading cause of early disability, loss of working capacity, and mortality in most countries worldwide. Among them, coronary heart disease (CHD) and arterial hypertension (AH) occupy a special place, as their prevalence steadily increases with age. Due to the global aging of the population, the management of elderly and senile patients is gaining not only medical but also pronounced socio-economic significance.

Age is considered one of the key risk factors for CHD. In older age groups, coronary heart disease is a manifestation of a systemic atherosclerotic process that develops against the background of a complex of involutional changes: neurohumoral regulation, lipid and carbohydrate metabolism, hemostasis, the immune system, and vascular wall structure. These changes reduce the body's adaptive reserves, increase its sensitivity to stress, and aggravate the course of the disease.

Arterial hypertension, especially its isolated systolic form, is also an extremely common condition in older age groups. Its pathogenesis is closely related to the loss of elasticity of large arterial vessels (the aorta and its branches), impaired baroreceptor function, and compensatory cardiac hyperfunction. At the same time, a moderate increase in blood pressure in the elderly may play an adaptive role in the context of age-related decline in cardiac output and deterioration of tissue perfusion.

Thus, the management of elderly and senile patients with CHD and AH requires a comprehensive, individualized approach that takes into account both general pathogenetic patterns and the specific features of clinical presentation, diagnosis, and treatment.

Keywords: Coronary Heart Disease, Arterial Hypertension, Elderly Age, Senile Age, Atherosclerosis, Stable Angina Pectoris, Acute Myocardial Infarction, Painless Myocardial Ischemia, Atypical Course, Systolic Hypertension, Risk Factors, Dyslipidemia, Hypercoagulation, Age-Related Changes, Heart Failure, Cardiac Arrhythmias, Pharmacotherapy, Beta-Adrenoblockers, Nitrates, Calcium Antagonists, Anticoagulants, Geriatrics, Vascular Elasticity, Baroreceptors, Population Aging, Rehabilitation, Prevention

Coronary Heart Disease

Results of epidemiological studies and clinical observations conducted in our country and abroad have shown that CHD is currently one of the main causes of early disability, invalidism, and death [1]. An increase in the incidence of CHD with age is noted, which can be associated with the corresponding

progression in the frequency and severity of atherosclerosis [2]. CHD in elderly and senile individuals should be regarded as a manifestation of a systemic atherosclerotic process, as confirmed by pathomorphological studies of deceased individuals and the results of clinical research [3].

Therefore, existing concepts of the pathogenesis of CHD in older age groups must include knowledge of age-related changes predisposing to the development of atherosclerosis [2]:

1. Changes in the nervous system, consisting of weakening of inhibitory processes in the CNS, relative sympathicotonia, weakening of trophic influences of the nervous system, increased sensitivity to neurohumoral factors [4-8]. It should be noted that sclerotic vessels often respond with a spastic reaction to reflex influences, as shown in experiments on old animals [4];

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2. Decreased activity of humoral and cellular immunity, autoimmune reactions of humoral and cellular types, presence of circulating immune complexes [3];
3. Increased blood levels of β -lipoproteins, triglycerides, cholesterol, decreased activity of lipoprotein lipase, and hepatic cholesterol excretion [2];
4. Decreased carbohydrate tolerance [9];
5. Decreased function of the thyroid gland, gonads, vasopressinemia, hyperinsulinemia, hyperreactivity of the sympatho-adrenal and renin-aldosterone systems [10];
6. Chronic activation of the blood coagulation system, increased fibrinogen concentration, compensatory increase in resting fibrinolysis intensity, excessive reaction of clotting mechanisms and functional insufficiency of anticoagulant mechanisms under stress conditions, increased clotting and adhesive-aggregation properties of platelets, disruption of prostacyclin-thromboxane balance, changes in the activity of tissue coagulation and fibrinolysis factors of the vascular wall [4];
7. Progressive deterioration of nutrition of the vascular wall tissue, development of destructive and degenerative changes within it, decreased energy metabolism, intensity of lipolytic and fibrinolytic processes, increased sodium content [3].

All these age-related changes ultimately lead to reduced resistance to stressful situations [4].

The significance of age as a risk factor for CHD has been convincingly demonstrated in studies conducted on a population with a high level of longevity in rural Abkhazia [2]. The lifestyle in this region predisposes to healthy longevity – the incidence of CHD is low here, but it still increases with age even in this population [2].

CHD risk factors retain their significance with age, but their role changes in different age periods [3]. In older age groups, disorders of lipid and carbohydrate metabolism, excess body weight, excessive salt intake, insufficient dietary content of unsaturated fatty acids, vitamins, and biologically active substances, as well as elevated blood pressure, become more important [9]. The significance of smoking and stressful situations at work and at home decreases with age [3]. Insufficient physical activity is a risk factor for CHD in the elderly; in the senile age, its influence on the development of the disease decreases [7].

Elimination of risk factors can increase life expectancy by 5–6 years in the elderly and by 2–3 years in the senile age [9,11]. In this regard, mass preventive examinations of this population segment are necessary and advisable for the purpose of primary and secondary prevention of the disease [9].

Chronic Coronary Heart Disease

Clinical picture and diagnosis. In elderly and senile age, as well as in middle age, various clinical variants of CCHD occur [6]. Thus, its most common form in patients over 60 years of age is stable angina pectoris, characterized by the constancy of the clinical course of the disease (nature, frequency, duration of pain syndrome) [12]. Stable angina may transition to unstable angina, but this form is less common than in middle age [13-16]. Spontaneous angina pectoris, which is based on the pathogenesis of coronary artery spasm, is extremely rare in elderly and senile

age [12]. The clinical forms of the disease in older individuals are determined by the peculiarities of the pathogenesis of the disease in old age [1,16,17,10-12]. In young individuals, functional disorders of the coronary circulation play a significant role in the development of myocardial ischemia, whereas in the elderly, organic, in particular age-related and atherosclerotic, changes become of primary importance [6,15].

The pain syndrome in stable angina pectoris in elderly and senile individuals may be typical; however, the frequency of atypical manifestations of coronary insufficiency increases with age [5]. It increases from 30% at age 60–69 years to 67% at age 80–89 years [6]. Atypical manifestations include gastralgic, asthmatic manifestations, and cerebral disorders [3,18,13-15]. The frequency of painless angina increases (from 25% in patients aged 60–69 years to 45% at age 80–89 years) [6]. In 40% of patients with the painless form of the disease, cardiac arrhythmias are detected [8].

Let us dwell in more detail on the age-related features of an angina attack [5]. During an angina attack in young individuals, several symptoms of an autonomic nature can be noted: frequent shallow breathing, feeling of faintness, fear of death, pallor of the skin [11]. However, in elderly and senile individuals, the emotional coloring of the attack is less vivid, autonomic manifestations are weakly expressed and often completely absent [9,16,12,17,11,10,1,19]. Pain is often of mild intensity, having the character of pressure, tightness behind the sternum or in the heart area [6-8,2,3,18,13-15,9]. Elderly and senile individuals often note sensations of an uncertain nature, such as heaviness in the left half of the chest [20]. Unusual radiation is also a feature of angina in older age groups [10]. Often the pain radiates to the jaw, occiput, neck, or is felt only in the places of its radiation [7]. It is noteworthy that in many elderly and senile individuals, exacerbation of coronary insufficiency is associated with a rise in blood pressure [17]. In some cases, neurological symptoms come to the fore, caused by insufficiency of cerebral circulation in the basin of a particular vessel, more often in the vertebrobasilar system [1,10-12,17,19-23].

In the etiology of a painful attack in old age, the significance of negative emotions decreases, which is apparently explained by the weakening of extracardiac regulation and a general decrease in the intensity of reflex reactions [4]. Conversely, the factor of physical overexertion acquires great importance [7]. This is because, against the background of age-altered myocardial metabolism leading to reduced efficiency of energy production, even a slight increase in demands on the heart quickly leads to a mismatch between coronary blood flow and myocardial oxygen demand [2]. This is significantly facilitated by atherosclerotic and age-related changes in the coronary arteries, since due to the loss of their elasticity, adequate blood supply to the myocardium cannot be ensured [10].

Meteorological factors, such as significant changes in atmospheric pressure or humidity, can provoke an attack of coronary insufficiency in elderly and senile individuals [5]. Abundant food, causing distension of the digestive tract and flatulence, is also a common provoking factor for angina pectoris and myocardial infarction [6].

Fat load, causing alimentary hyperlipidemia, as already indicated, activates the blood coagulation system in elderly and senile persons [4]. The greatest tendency to hypercoagulation is observed 3–5 hours after ingestion of fatty food [2]. Therefore, even after consuming a small amount of it, attacks of angina pectoris or myocardial infarction may occur, especially at night [9].

In the natural course of chronic coronary ischemia, a high risk of myocardial infarction and a general tendency to increase the symptoms of coronary insufficiency should be noted [5]. The result of coronary insufficiency is dystrophy of muscle fibers, followed by their atrophy with replacement by connective tissue; atherosclerotic cardiosclerosis develops, which leads to heart failure and various cardiac rhythm disorders [6].

Rhythm and conduction disturbances – extrasystole, atrial fibrillation, blocks of varying degrees and of various parts of the conduction system – are frequent and significant symptoms of atherosclerotic cardiosclerosis in elderly patients with CHD [1-3,8-20].

The frequent occurrence of these disorders is due to the fact that with age, even during physiological aging, conditions arise in the heart that contribute to rhythm disturbances, among which the following should be highlighted: decreased lability and automaticity of the sinus node; decreased neural influences on the heart; increased sensitivity of the myocardium to humoral substances, in particular to vasopressin, catecholamines; heterogeneity of the myocardium and conduction system of a morphological and metabolic nature; hypertrophy and dilatation of the left ventricle; decreased energy metabolism; increased glycolytic processes; changes in electrolyte content, especially potassium ion content; delayed recovery of metabolic shifts in the myocardium; decreased coronary circulation; microcirculation disorders; hypoxia [4].

Cardiac rhythm disorders (ventricular fibrillation, asystole) are potential factors of fatal outcome; reduce the efficiency of the heart, lead to heart failure; cause thromboembolism [2].

Against the background of chronic circulatory failure due to atherosclerotic cardiosclerosis, acute left ventricular failure often occurs in elderly individuals [16,12,17,11,10,1,19-21]. Easier and faster development of heart failure in elderly and senile individuals, unlike in young individuals, is facilitated by age-related changes in the cardiovascular system, primarily a decrease in myocardial contractility [20-22]. Therefore, the main hemodynamic form of heart failure in older patients with CCHD is low cardiac output failure [17].

The trigger for decompensation in elderly and senile patients with CCHD is often bronchopulmonary diseases, physical and alimentary loads, psycho-emotional stress, acute infections, inflammatory processes in the urinary tract, attacks of paroxysmal tachycardia, atrial tachycardia, surgical interventions, and even irrational therapy, etc [6].

More frequent cerebral circulation disorders in heart failure in elderly patients are associated both with age-related changes in cerebral hemodynamics (decreased intensity of

cerebral circulation) and with atherosclerotic vascular lesions [2,3,18,9,12-16].

A particularly high frequency of thromboembolic lesions of the pulmonary trunk in heart failure should be noted [5]. Due to the decrease in compensatory capabilities of the cardiovascular system with age, the prognosis for heart failure in older patients is worse than in young individuals [6].

Due to the atypical course of CCHD in the elderly, instrumental research methods should be more widely used for diagnosing the disease, in particular electrocardiography, both at rest and during exercise, and topoelectrocardiography [5]. Vectorcardiography significantly complements the possibilities of electrocardiography, especially in cases of conduction dysfunction and left ventricular hypertrophy [6]. X-ray contrast and radionuclide studies are also used [18].

Treatment. In all cases, treatment of patients should be comprehensive [21]. It is necessary to identify CHD risk factors, possibly eliminate stressful situations, normalize the work and rest schedule, physical activity, and nutrition [7-9,2,3,12-18]. When treating elderly patients with CHD, it should be taken into account that age-related changes in the pharmacokinetics and pharmacodynamics of drugs determine a specific approach to their prescription [5-8,2]. It is important to determine the optimal dosage of the drug, starting with small doses, as they are often sufficient for effective treatment [4-8]. Large doses in elderly and senile individuals may be ineffective, and sometimes even cause complications [15]. To assess the effectiveness of a drug, acute loading tests with different doses should be used, studying changes in hemodynamic parameters, contractile and bioelectrical activity of the myocardium [2-9,12-17]. The choice of antianginal drug should be made based on the above indicators [17,11,10,1].

The main objectives of pharmacotherapy for chronic forms of CHD are to ensure the correspondence between oxygen delivery to the myocardium and its demand in order to prevent recurrent angina attacks, to combat rhythm disturbances, the progression of atherosclerosis and cardiosclerosis, as well as the development of heart failure [15].

The severity of organic, age-related, and atherosclerotic changes in the coronary vessels in older individuals makes it inappropriate to use antianginal drugs whose main mechanism of action is a coronary vasodilatory effect (e.g., chimes (synonym: dipyridamole), carbocromene, etc.) [17]. The most promising is the use of drugs that reduce myocardial oxygen demand and reduce cardiac work (β -adrenoblockers, nitrates, etc.) [19-21].

For severe pain syndrome, fast-acting drugs should be used, for example, nitroglycerin, which belongs to the nitrate group [15]. The main mechanism of action of drugs in this group is to reduce cardiac work due to peripheral vasodilation [12,17,11,10]. The effect occurs within 5–10 minutes [16,12,17,11,10,1]. Nitroglycerin is rapidly destroyed in the body, so its administration can be repeated after 4–5 minutes, and for a repeated painful attack a little later – after 15–30 minutes [15]. Many elderly and senile patients with CHD and severe atherosclerotic lesions of

the cardiovascular system tolerate usual doses of nitroglycerin poorly, which cause them general weakness, headache, and dizziness [9]. According to some researchers, the reason is a decrease in blood pressure to a level at which, due to severe coronary atherosclerosis, blood supply to the heart becomes insufficient; according to others, nitrates, by dilating peripheral vessels, increase the tone of cerebral vessels [4-8]. Often, half doses of nitroglycerin stop the attack and do not cause unpleasant sensations [9,16,12,17,11]. The combination proposed by B. E. Votchal can also be recommended: 9 ml of 3% menthol alcohol and 1 ml of 1% alcohol solution of nitroglycerin [14-17,9]. Amyl nitrite can also be used to stop angina attacks [15]. To relieve pain syndrome, mustard plasters on the heart area, chloroethyl blocks, and intracutaneous novocaine block are used [16,12,17,11,10,1]. In the presence of clinical and electrocardiographic indications, narcotic analgesics (morphine, promedol) should be prescribed in combination with analgin, antihistamines (diphenhydramine, suprastin, pipolphen, etc.); neuroleptics and inhalation of nitrous oxide are also prescribed [15]. Validol is generally ineffective for relieving pain syndrome in elderly and senile individuals [17]. Along with the listed drugs, long-acting drugs are prescribed for pain syndrome: nitrates, β -adrenoblockers, calcium antagonists, etc [15]. A protracted attack of angina should be differentiated from acute myocardial infarction [13].

The approach to choosing a drug for long-term antianginal treatment should be differentiated [17]. In geriatrics, this is of particular importance due to pronounced organic changes in the myocardium and coronary vessels, weakening of neuro-reflex influences, and a decrease in the adaptation reserve of the circulatory system [4].

β -Adrenoblockers are often the drugs of choice in geriatric practice [15]. They reduce myocardial oxygen demand due to negative chronotropic and inotropic effects [17]. β -Adrenoblockers without sympathomimetic activity (anaprilin, etc.) should be prescribed in the absence of bradycardia [15]. Treatment should be started with a small dose, as large doses can cause undesirable side effects – signs of heart failure in the form of hypotension, severe dyspnea [9]. If it is necessary to prescribe β -adrenoblockers to patients with bradycardia, drugs with intrinsic sympathomimetic activity (visken, trasicor) are recommended [17]. They have a less pronounced negative chronotropic effect [6]. The peripheral vasodilating effect of these drugs allows them to be used also in elevated blood pressure in patients with CHD [4].

Despite differences in mechanisms of action, the indications for prescribing cordarone are the same as for visken, since ultimately these drugs lead to similar results: the α -adrenoblocking effect of cordarone on adrenoreceptors of peripheral vessels leads to a decrease in peripheral vascular resistance and blood pressure, while the β -adrenoblocking effect leads to a negative chronotropic effect [15]. The absence of a cardiodepressive action in cordarone distinguishes it favorably from β -adrenoblockers and makes it most indicated in geriatric practice [4].

Prescription of drugs whose antianginal action is mainly associated with a peripheral vasodilating effect is justified in cases of not far-advanced atherosclerosis of coronary and

peripheral vessels (nitrates, corinfar, finoptin, isoptin, corvaton) [15,9,16,12,17,11,10,1,19,20]. In hypotension, they are ineffective [15]. Moreover, with an even greater decrease in blood pressure in these patients, complications may develop: dyspnea, dizziness, increased frequency of cardiac complications [4-7]. The likelihood of their development increases with age [2].

Drugs with a predominantly coronary vasodilatory effect are effective in mild coronary atherosclerosis, when CHD is based on coronary spasm (chimes, oxyfedrine, etc.) [15]. When combined with β -adrenoblockers, they become effective [19]. In geriatric practice, the combination of small doses of β -adrenoblockers with nitrates, calcium antagonists, and glycosides is rational more often than in the treatment of middle-aged patients [4-7]. When combining β -adrenoblockers with isoptin, finoptin, or glycosides, careful monitoring of the magnitude of the negative chronotropic action is necessary [19]. All of the listed drugs possess this property, and therefore, when they are used together, the likelihood of developing pronounced bradycardia increases [15,9,16,12,17,11,10]. Combination of calcium antagonists with glycosides is possible [17].

In unstable angina, the appropriateness of prescribing anticoagulants, antiplatelet agents, and antithrombotic drugs is beyond doubt [15]. It should be emphasized that adequately selected drug therapy should be used as a basis for physical rehabilitation of elderly patients with CCHD [7]. When conducting physical training in elderly individuals, it is necessary to consider that the adaptive capabilities of the circulatory system decrease with age [17]. Physical rehabilitation is less effective for elderly patients than in middle age, but can be successfully used in a complex of rehabilitation measures [7].

A special place in the treatment of patients with CCHD should be given to the correction of cardiac rhythm disorders, the frequency of which increases in elderly patients with CCHD [6]. Treatment of CHD patients with circulatory failure requires a special approach and special discussion [5].

Sanatorium-resort treatment for elderly patients is more indicated in local cardiological sanatoriums [7]. The issue of surgical treatment is decided strictly individually [14]. This is because old age is accompanied by a large number of diseases with a high risk of complications and death [13]. It should be noted, however, that due to widespread atherosclerosis, the need for surgery in elderly patients occurs less frequently [14].

Acute Myocardial Infarction

Myocardial infarction is understood as a focus of necrosis caused by a mismatch between the blood supply to a specific area of the myocardium and its demands at that moment [13]. The probability of myocardial infarction in elderly and senile individuals is higher than in middle age [5]. The following factors underlie this phenomenon [4]:

1. Age-related changes in the physicochemical and biochemical properties of blood: changes in electrokinetic processes in the vascular bed, increased adhesive and aggregation properties of platelets, increased hypercoagulation potential (hypercoagulation), decreased functional capabilities of the

fibrinolytic system, disruption of regulatory mechanisms of the hemocoagulation system, decreased lipoprotein lipase activity, dyslipoproteinemia, increased content of coarse proteins [2];

2. Age-related changes in the vascular wall: hyperplastic and destructive processes (age-related sclerosis), changes in coagulation-fibrinolytic potential, inhibition of lipolytic processes, tortuosity and aneurysmal dilation of capillaries and arterioles [10].

Neuroleptanalgesia with the synthetic analgesic fentanyl (0.05–0.1 ml) and the neuroleptic droperidol (5 mg) is effective [15]. A ready-made mixture of these drugs, thalamonal (1 ml contains 2.5 mg droperidol and 0.05 mg fentanyl), is often used [9,16,12,17]. To combat an intractable painful attack, a narcotic mixture of nitrous oxide (60%) with oxygen is used [15]. Relief of pain syndrome can be achieved by administering sodium hydroxybutyrate (120–150 mg/kg intramuscularly) [4-8]. To improve the function of the peri-infarction zone and limit the size of necrosis, fibrinolytic and anticoagulant therapy is prescribed [13]. For the same purpose, a glucose-potassium-insulin mixture and corticosteroids are used [15]. To improve myocardial trophism and metabolic processes, vitamins, potassium preparations, anabolic steroids, and other drugs are prescribed [17]. These measures may also be useful for preventing arrhythmias [9,11,12,14-17]. Improved oxygenation of ischemic myocardium can be achieved through oxygen therapy, which should be carried out in the first days of the disease at oxygen concentrations of 40–60% hourly for 10–15 minutes [6-8,2,3,18,13]. In recent years, hyperbaric oxygenation has been increasingly used in the treatment of patients with myocardial infarction [15].

Treatment with anticoagulants and thrombolytic drugs should be started from the onset of the disease [13]. In the absence of contraindications, intravenous administration of heparin 10,000 units is recommended, followed by intravenous or intramuscular administration of 5000 units at intervals not exceeding 6 hours, under the control of main coagulogram parameters and blood clotting [15]. The effective dose is determined – blood clotting should decrease by half [17]. Treatment with heparin is carried out for 5-7 days, but in some cases the duration may be extended [13]. Toward the end of treatment, indirect anticoagulants (phenilin, pelentan, syncumar) are added so that their effect is already manifest by the time heparin is discontinued [11]. When prescribing indirect anticoagulants, a decrease in the prothrombin index to 60-65% is achieved [17]. When prescribing anticoagulants to older patients, it should be taken into account that impaired liver and kidney function alter sensitivity to anticoagulants; moreover, these patients often have reduced activity of the prothrombin complex of the blood [9].

The antagonist of heparin is protamine sulfate [15]. The drug is administered slowly intravenously as a bolus: 0.1–0.2 ml of a 1% solution of the drug is required to neutralize 100 units of heparin [17].

Fibrinolytic agents (fibrinolysin, streptolyase, streptase, urokinase) are also indicated in the treatment of older patients with myocardial infarction [13]. Age over 70 years is a relative contraindication to their prescription [15].

For the prevention of arrhythmias, lidocaine, mexiletine, novocainamide, β -adrenoblockers, cordarone, and the administration of a “polarizing mixture” are indicated for patients [17].

In cardiogenic shock, it is necessary to relieve pain syndrome and prescribe vasotonic agents: cordiamine, corazole, mezaton [17]. To increase myocardial contractility, glycosides and pressor amines (norepinephrine, mezaton) are used intravenously as a drip [15]. β -Adrenostimulants (isadrin, isoproterenol, novodrin, izuprel) and glucocorticosteroids have a positive inotropic effect on the myocardium [9,16,12,17,11]. To increase blood pressure, pressor agents – angiotensin II – are used [17].

The presence of hypovolemia is an indication for the administration of plasma expanders [15]. Due to the metabolic acidosis that occurs in such patients, correction of the acid-base state should be included in therapy [15,9,16]. All of the listed drugs should also be used starting with small – 50% of the average therapeutic – doses [9]. Treatment of pulmonary edema involves immediate relief of pain syndrome, lowering of blood pressure (if elevated), correction of acid-base state and electrolyte balance [15]. In case of rhythm disturbances, antiarrhythmic therapy is indicated [15]. It is also necessary to improve the contractile function of the myocardium and reduce venous return of blood to the heart [9-12,14-17]. Narcotics, glucocorticosteroids, ganglionic blockers, diuretics, peripheral vasodilators, tourniquets on the extremities, hot foot baths, venesection, oxygen inhalation, and antifoam agents are used [14]. The effectiveness of treatment in older age groups depends on the correct decision regarding the duration of strict bed rest [7]. This issue is decided individually, based on the extent of myocardial damage and the clinical course of the disease [13].

Usually, bed rest is gradually discontinued by the end of the 2nd–3rd week of the disease [7]. Light massage and respiratory therapeutic exercises as early as the 3rd–4th day of the disease are of great importance in returning to the usual motor regimen [15]. For the prevention of hypostatic pneumonia, therapeutic exercises are carried out under the control of objective research methods [17]. Physical rehabilitation should occupy an important place in the treatment of patients with myocardial infarction [7]. With prolonged bed rest, it is necessary to exclude the possibility of pressure ulcers and urinary tract infections; regulating bowel function is very important [9]. A patient with myocardial infarction in the acute phase of the disease should be prescribed a diet with salt restriction [7]. Subsequently, the patient is subject to mental and social rehabilitation [15]. It is necessary to carry out measures aimed at preventing the development of recurrent infarction, for which it is advisable to use antianginal drugs, especially β -adrenoblockers, antiatherosclerotic drugs that improve metabolic-trophic processes in the heart muscle [17]. For long-term prevention of coronary thrombosis, the use of indirect anticoagulants and fibrinolysis stimulants is indicated; however, when prescribing them, it is necessary to take into account the high sensitivity and low tolerance of the elderly and senile organism to pharmacological agents [9].

Arterial Hypertension

Arterial hypertension occurs in various diseases: endocrine (thyrotoxicosis, pheochromocytoma, Itsenko-Cushing's disease,

acromegaly), renal and urological (glomerulonephritis and pyelonephritis, urolithiasis), cardiovascular (atherosclerosis of specific vascular zones, circulatory insufficiency, coarctation of the aorta), anemias, certain brain tumors, and other types of pathology [16]. However, in all these diseases, elevated blood pressure is only one of the symptoms, often far from the leading one [12]. The most common conditions in elderly and senile age are essential hypertension and so-called atherosclerotic or isolated systolic hypertension [18].

Blood pressure is an integral indicator of the activity of the cardiovascular system; its level depends on many factors, primarily on the magnitude and speed of cardiac output and the vascular resistance of the arterial system [16]. The participation of various structural and functional components of the arterial system in the formation of a certain level of blood pressure is unequal [12]. The greatest role belongs to muscular-type vessels – arterioles, the least – to large elastic-type arterial vessels: the aorta and its main branches [4-8,2,3]. The functional state of the arterioles – muscular-type vessels – and their ability to respond to neurohumoral pressor and depressor impulses determine the level of blood pressure [16]. At the same time, the presence of clusters of baroreceptors in the region of the aortic arch and carotid sinuses completely changes the concept of the role of large arterial vessels in the formation of blood pressure levels [12]. This manifests itself especially clearly with age [2]. It is the large arterial vessels that, losing elasticity and distensibility with aging, are the first to lose the function of an active accumulator of the kinetic energy of the heart [10,1,19-22]. It is at this segment of the vascular system that the heart develops compensatory hyperfunction to overcome the increased resistance to the ejected volume of blood [16]. The total elastic resistance of the arterial system also increases significantly in elderly and senile individuals [12]. But along with a decrease in elasticity and distensibility of large arteries, their ectasia and elongation occur, resulting in an increased volume of the arteries, especially the aorta, which to some extent compensates for the loss of shock-absorbing properties by large vessels [10].

Perhaps, under such circumstances, there would be no increase in blood pressure with age if the sclerotic process in the vessels did not involve the baroreceptor regions [16]. However, a decrease in the distensibility of the aortic and carotid artery walls in the baroreceptor zones and age-related changes in the functional properties of the baroreceptors themselves increase their excitation threshold, requiring higher blood pressure values to activate them in the blood pressure regulation system [12]. Due to age-related and atherosclerotic processes, sensitivity to hypoxia also increases in the higher regulatory centers (reticular formation, medulla oblongata); therefore, worsening of blood supply associated with age-related decrease in cardiac output increases the frequency of inadequate reactions, which usually manifest as a more or less persistent increase in blood pressure [4]. It must be borne in mind that under conditions of decreasing cardiac output with age and increasing hypoxia of organs and tissues, a slight increase in blood pressure plays a compensatory role, helping to improve tissue trophism [8].

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