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Review Article

Age-Related Changes in the Cardiovascular System and Their Impact on Morphology, Hemodynamics, Neurohumoral Regulation, And Response to Physical Exertion

Bon L. I *, Maksimovich N. Ye., Znavets P.A

Grodno State Medical University.

***Correspondence Author:** Bon E.I, Grodno State Medical University.

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Abstract

Although aging of the cardiovascular system is not the primary cause of organismal aging as a whole, it significantly influences its rate and manifestations. These changes reduce the body's ability to adapt and increase the risk of developing diseases. Despite the fact that these changes are regular and progressive in nature, their severity and individual features vary depending on genetic, environmental, and behavioral factors. Studying age-related changes in the heart and blood vessels helps to more accurately diagnose, treat, and prevent circulatory system diseases, as well as to distinguish natural age-related transformations from pathological ones. Understanding the processes of myocardial and vascular aging allows not only to predict the likelihood of cardiovascular complications but also to select individualized treatment for elderly people. This article reviews the main age-related changes in the cardiovascular system: structural remodeling of the vascular wall and myocardium, features of central and peripheral hemodynamics, disorders of neurohumoral regulation, as well as the nature of the cardiovascular system's response to physical exertion in elderly and senile individuals.

Key words: aging; cardiovascular system; vascular remodeling; arterial stiffness; hemodynamics; cardiac output; myocardial contractility; neurohumoral regulation; autonomic nervous system; physical exertion; exercise response; vascular tone; muscle fibers; vagus nerve

Morphological changes in blood vessels

With aging, the structure of the vascular wall changes [11]. The main changes occurring in large arterial trunks manifest as sclerotic thickening of the inner membrane – the intima, atrophy of the muscular layer, and decreased elasticity [13]. These changes lead to an insufficient capacity of vessels not only to dilate but also to constrict, which, along with altered central regulation of vascular tone, disrupts the adaptive abilities of the circulatory system [10]. Primarily and to a greater extent, the large arterial vessels of the systemic circulation, especially the aorta, are affected [14]. Decreased elasticity of the pulmonary trunk is noted only in patients of older age groups [1-3]. Age-related changes in vascular elasticity manifest as an increase in pulse wave velocity, changes in the rheogram curve shape, and temporal parameters [11,19]. With age, the number of functioning capillaries per unit area decreases, and their basement membrane thickens, which leads to a decrease in the intensity of transcapillary exchange [6].

Hemodynamic features

He loss of elasticity by large arterial vessels and the increase in peripheral vascular resistance cause a certain rise in blood pressure with age (systolic pressure more so) [11,16]. On the other hand, the increase in the volume of large arterial trunks, especially the aorta, and the decrease in cardiac output act in the opposite direction, limiting a significant rise in blood pressure [1]. Venous pressure decreases with age [5]. This is due to weakened tone and decreased elasticity of the venous wall, leading to an expansion of the total cross-sectional area of the venous bed [4-12]. In elderly and senile age, the value of cardiac output decreases [1]. In elderly individuals, this decrease is mainly associated with a slowing of heart rate; in senile age, a significant decrease in stroke volume is noted [1,5]. In elderly and old people, against the background of reduced cardiac output, active redistribution of regional blood flow is observed [16-18]. At the same time, cerebral and coronary circulation, despite a progressive age-related decline, is maintained at a fairly high level, whereas renal and hepatic circulation significantly decreases [7]. Since basal metabolism decreases with age, the reduction in cardiac output in elderly and old people can be considered a natural reaction of the cardiovascular system to a decrease in tissue oxygen demand [17]. Furthermore, the arteriovenous oxygen difference increases with age, which is associated with a rightward shift of the oxyhemoglobin dissociation curve in old age and a slowing of blood flow velocity in various parts of the cardiovascular system [1]. Such a restructuring of the hemodynamic system partially compensates for the increase in energy expenditure during cardiac work under conditions of increased resistance to cardiac output due to elevated peripheral vascular resistance [11,16].

Decreased myocardial contractility

A decrease in myocardial contractility is noted with age, as indicated by data from numerous non-invasive and invasive research methods [1]. Among the causes of this phenomenon, morphological changes in the "senile" heart should be noted, manifested as progressive myocardial sclerosis, focal atrophy of muscle fibers, and proliferation of elements of low-elasticity connective tissue [2,8]. These changes lead to a decrease in the efficiency of the Frank-Starling mechanism [5]. The age-related decrease in energy processes in the myocardium, along with disturbances in mineral metabolism, creates a basis for limiting the heart's adaptation reserve [14]. In old age, the thresholds for the influence of the sympathetic nervous system on myocardial contractility increase, and a decrease in the inotropic effect of catecholamines is noted [10]. Age-related changes in the myocardium of a morphological, metabolic, and regulatory nature create conditions for energy-dynamic insufficiency of the heart under conditions of intense activity [2,5].

Depression of myocardial bioelectrical activity

The main pattern clearly observed during aging is a decrease in the level of repolarization processes in the myocardium [6]. This is expressed in a decrease in the amplitude of the T wave on the electrocardiogram in all leads [8]. It should be noted that in leads I, II, aVL, V3–V6 in physiologically aging individuals, it is always positive, and the ST segment is on the isoline [11-18]. These data underlie the diagnosis of coronary heart disease (CHD) in geriatric patients, as they often exhibit minor ECG changes during acute coronary insufficiency [6,9]. The depolarization process also changes with age, the QRS complex widens, but normally does not exceed 0.1 s [8]. The electrical axis of the heart deviates to the left, indicating predominant changes in the left ventricular myocardium [3]. The electrical systole of the heart lengthens with age [6]. Conditions for the spread of excitation in the atria worsen, atrioventricular conduction and the spread of excitation through the ventricular myocardium slow down [1-6]. A regular sinus rhythm is characteristic of the physiological type of aging [15]. In elderly and old people, fluctuations in the duration of individual cardiac cycles, independent of the respiratory phase, may sometimes occur [19-21].

Disorders of neurohumoral regulation

With aging, conditioned reflex influences on the cardiovascular system weaken [9-13]. In elderly people, a complex motor dynamic stereotype with a pronounced cardiovascular component is established with greater difficulty than in young people [4]. Unconditioned reflexes of circulatory regulation also change, and inertia of vascular reactions is revealed [14]. In response to thermal stimuli, approximately half of individuals over 60 years of age exhibit an inadequate reaction [20]. Shifts in vascular tone and blood pressure are characterized by a prolonged recovery period [10,14]. Changes in the reflex regulation of the cardiovascular system are often associated with shifts in the functional state of the hemodynamic center [8-14]. Experimental studies have shown that with aging, the influences on the cardiovascular system originating from various brain structures change [15]. In turn, feedback also changes – reflexes from the baroreceptors of the carotid sinus and aortic arch weaken [18]. As a result, the mechanisms of blood pressure regulation deteriorate [16,20]. In old age, sympathetic nervous influences on the cardiovascular system weaken [10]. The mechanism of this phenomenon is associated with the destruction of sympathetic nerve endings, a decrease in noradrenaline synthesis, and inhibition of its reuptake [11,14]. Along with the weakening of sympathetic influences on the cardiovascular system, its sensitivity to catecholamines increases with age, which to some extent compensates for the decrease in sympathetic nervous influences [12,18]. At the same time, in the elderly, the positive inotropic effect of adrenostimulants, mediated through the β- and α-adrenergic regulatory links, significantly decreases [19-21]. Simultaneously, under the influence of catecholamines, rhythm disturbances occur more often with age, and myocardial energetics suffer [10,14].

During the aging process, the influences of the vagus nerve on the heart weaken, but its sensitivity to the cholinergic mediator increases [8]. The weakening of nervous influences on the cardiovascular system is largely associated with destructive changes in the neural apparatus and shifts in acetylcholine synthesis, as well as changes in the number and functional state of cholinergic receptors [7-12]. The reduction in nervous influences on the cardiovascular system is uneven and heterochronic in nature [15]. Thus, with age, a relative predominance of sympathetic influences on the heart rhythm is noted [10-13].

Age-related changes in hormone secretion, metabolism, and the state of cellular receptors largely determine shifts in metabolic processes and the function of the cardiovascular system in old age [14,16]. A decrease in the effective concentration of anabolic hormones (insulin, sex hormones) contributes to the development of functional insufficiency [16]. With age, the body's sensitivity to vasopressin and other hormonal substances, in particular angiotensin and histamine, increases [14].

Thus, with age, the role of neural mechanisms in the regulation of the cardiovascular system weakens, while the importance of humoral mechanisms increases [10,19]. At the same time, the overall reliability of neurohumoral influences decreases with aging, increasing the likelihood of regulatory mechanism failure and the development of pathology [10,21].

Features of the response to physical exertion

Functional stress tests, particularly physical exertion, are of great importance for revealing the functional capabilities of the cardiovascular system [3,15]. A regular decrease in physical performance is noted with age [3,4]. This is manifested by a decrease in the maximum possible load and the level of maximum oxygen consumption [1,4]. The reserves for increasing cardiac output during exercise diminish [3,5]. At the same time, hemodynamic support lags behind the increase in energy demands during physical exertion, leading to earlier activation of anaerobic energy sources and a decrease in the anaerobic threshold [3,19]. The inadequacy of hemodynamic support for exercise in the elderly is associated with a number of factors that cause a less economical response of the hemodynamic system [1,5]. Thus, with age, a more pronounced hypertensive reaction to physical exertion is noted, and peripheral vascular resistance decreases to a lesser extent [11,13]. In old age, the reserve for increasing stroke volume is limited, which is associated with a decrease in myocardial contractility and changes in the neurohumoral regulation of cardiac activity [1,5,10]. In elderly and old people, a delayed development of changes in hemodynamic and respiratory indicators induced by muscular work is noted [15]. This is manifested by a prolongation of the warm-up period, which is associated with a decrease in the lability and functional mobility of neural mechanisms regulating autonomic functions and a violation of optimal viscero-locomotor relationships [18]. A delayed recovery of functional indicators of the cardiovascular system after muscular work is characteristic of older age groups [15].

Thus, the response of the cardiovascular system to physical exertion in the elderly clearly indicates a limitation in the range of the body's reserve capabilities and a decrease in the efficiency of its activity [3,4,16].

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