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Е.С. Фомина*[1], В.С. Никифоров[2]

[1] — СПб ГБУЗ «Николаевская больница», Петергоф, Россия
[2] — ФГБОУ ВО «Северо-Западный государственный медицинский университет им. И.И. Мечникова» Минздрава России, Санкт-Петербург, Россия

АРТЕРИАЛЬНАЯ ЖЕСТКОСТЬ И СОСУДИСТОЕ СТАРЕНИЕ: ПОСЛЕДСТВИЯ АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИИ

E.S. Fomina*[1], V.S. Nikiforov[2]

[1] — Nicholas hospital, 198510, Peterhof, Russia
[2] — North-Western State Medical University named after I.I. Mechnikov, Saint Petersburg, Russia

Arterial Stiffness and Vascular Aging: Effects of Hypertension

Резюме

В настоящем обзоре освещены вопросы взаимосвязи возраста и артериальной гипертензии, наблюдаемой в процессе старения организма. Проанализированы основные структурно-функциональные изменения, лежащие в основе повышения сосудистой жесткости. Отмечено сходство сосудистых изменений при старении и при артериальной гипертензии. Рассмотрено негативное влияние повышенного центрального артериального давления на органы-мишени. Уделено внимание анализу артериальной жесткости, как маркеру сосудистого старения. Отдельно освещены показатели каротидно-феморальной скорости распространения пульсовой волны, сердечно-лодыжечного сосудистого индекса (CAVI), лодыжечно-плечевого индекса, пальце-плечевого индекса и индекса аугментации. Рассмотрена прогностическая и клиническая ценность параметров сосудистой ригидности. Также отмечена независимая диагностическая и прогностическая ценность показателя сердечно-лодыжечного сосудистого индекса (CAVI).

Ключевые слова: *артериальная гипертония, старение, скорость пульсовой волны, центральное аортальное давление, индекс аугментации, эластичность артерий, артериальная жесткость, сосудистая жесткость*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

This review highlights the relationship of age and arterial hypertension observed in the aging process. The main structural and functional changes underlying the increase in vascular stiffness are analyzed. The similarity of vascular changes in aging and arterial hypertension was noted. The negative effect of increased central blood pressure on target organs is considered. Attention is paid to the analysis of arterial stiffness as a marker of vascular aging. The parameters of the carotid-femoral pulse wave propagation velocity, the cardio-ankle vascular index (CAVI), the ankle-brachial index, the finger-brachial index, and the augmentation index were examined separately. The prognostic and clinical value of the parameters of vascular

*Контакты: Елена Святославовна Фомина, e-mail: el.fomina@bk.ru
*Contacts: Elena S. Fomina, e-mail: el.fomina@bk.ru
ORCID ID: https://orcid.org/0000-0003-1747-108X

stiffness is considered. In particular, the clinical guidelines for arterial hypertension report the need to use arterial stiffness indicators to improve the accuracy of cardiovascular risk stratification, especially in medium-risk patients. Measurement of vascular stiffness and central aortic pressure should be recommended as one of the methods for stratifying cardiovascular risk in patients with intermediate SCORE risk, as well as in those whose target organ damage was not detected by routine methods. The article also notes the independent diagnostic and prognostic value of the CAVI.

Key words: *arterial hypertension, aging, pulse wave, central aortic pressure, augmentation index, arterial elasticity, aortic rigidity, vascular stiffness*

Conflict of interests

The authors declare no conflict of interests

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AH — arterial hypertension, AI — augmentation index, BP — blood pressure, CAVI — cardio-ankle vascular index, LV — left ventricle, PWV — pulse wave velocity, SBP — systolic BP

Introduction

The elderly and senile population is rising [1]. Arterial hypertension (AH) is the main factor contributing to the risk of cardiovascular complications and mortality in this age group [2].

The relationship between age and AH observed during the aging process is of great practical importance [3]. This is accompanied by a number of changes in the vascular system, particularly endothelial dysfunction, increased vascular stiffness, vascular wall remodeling and inflammation, defined as the so-called «vascular phenotype» of AH [4]. Such changes in blood vessels in young individuals with arterial hypertension suggest «premature» or «early» vascular aging [5].

Structural and Functional Changes in Blood Vessels During Aging

Physiological changes in the vascular wall are progressive and develop throughout life [6, 7]. Subclinical thickening of the intima-media complex is associated with aging and is also a predictor of future cardiovascular events [8].

Physiological aging affects elastic type vessels more than muscle type vessels; changes develop regardless of the progression of atherosclerosis [9]. The main changes during arterial remodeling affect the intima and middle layer [10]. Along with morphological rearrangement due to atherosclerosis, age-related involution of structural proteins — elastin, fibulin, collagen — is observed in the arterial medial layer and, as a result, thinning, splitting, and fragmentation of elastic fibers develop [7]. Their degeneration is associated with increased collagen, decreased elastin and calcium deposition [11]. Calcification of vascular media is a sign of vascular aging. Endothelial cells of the intima change their size and shape; their function progressively worsens [12]. There is a thickening of the subendothelial layer, separation of endothelial cells from smooth muscle cells, and an increase in the amount of connective tissue [13]. In the end, functional changes progress in the form of endothelial dysfunction [12]. In particular, endothelium-dependent vasodilation decreases due to less production of biologically active substances with a vasodilating effect by endothelial cells, i.e., endothelin-1 and nitric oxide (NO) [14]. Nitric oxide is also known to have an anti-atherosclerotic effect [15]. Decreased vascular elasticity leads to the reduced release and bioavailability of nitric oxide, which is the trigger for the formation of atherosclerotic plaques and may result in a further increase in arterial stiffness [16].

Parallels Between Vascular Changes in Hypertension and Aging

Many mechanisms associated with vascular changes during aging are also activated in hypertension leading to impaired rheological properties of blood, endothelial dysfunction, vascular inflammation, remodeling and increased arterial stiffness [7, 17]. In individuals with risk factors due to genetic, environmental, or intrauterine determinants of fetal development, the processes of vascular changes are accelerated; they lead to early vascular aging, which predisposes to cardiovascular diseases [18]. Numerous risk factors such as smoking, hypercholesterolemia, hypertension, type 2 diabetes mellitus intensify arterial aging processes, partly due to increased oxidative stress, activation of pro-inflammatory mechanisms and changes in the regulation of the renin-angiotensin-aldosterone

system [19]. Association, mainly with diabetes, significantly increases the risk of micro- and macrovascular complications and cardiovascular morbidity [20]. As with aging, in case of arterial hypertension, there is decreased endothelium-dependent vasodilation, decreased bioavailability of NO, breakdown of NO synthase, increased oxidative stress, and development of endothelial dysfunction [21]. In large arteries, these molecular and cellular processes are manifested as increased arterial stiffness, which causes elevated central blood pressure, leading to isolated systolic hypertension, which is common in the elderly population [22]. However, the exact mechanisms causing these cellular and vascular events are not entirely clear, and it is quite difficult to differentiate the "age-related effect" from the "blood pressure effect". Vascular properties are thought to depend on the effect of several interdependent factors that change along with body aging throughout life [23].

Arterial Stiffness in Terms of Cardiovascular Disease Continuum

Mechanical changes associated with structural and functional disorders are characterized by decreased compliance, elasticity/extensibility and increased stiffness of the vascular wall [24]. Narrowing of the arterial lumen due to the destruction of elastic fibers in the middle layer and collagenous remodeling leads to increased pulse pressure in the aorta and increased pulse wave velocity (PWV) [24, 25]. Increased aortic stiffness causes changes in the functioning of the cardiovascular system as a whole. A less extensible aorta cannot effectively dampen blood volume ejected into the systole by the left ventricle, leading to increased central systolic blood pressure (SBP), thereby increasing the afterload on the left ventricle (LV) and contributing to its hypertrophy [26]. Since the efficiency of cardiac output is determined by the global contractile function of the LV, the elasticity of main arteries, and general peripheral resistance, it is the central and not peripheral BP that determines the level of the afterload on LV walls during systole [27]. High aortic SBP for a given stroke volume requires a large amount of energy in LV systole, which ultimately reduces the efficiency of cardiac output [28]. Reduced coronary blood flow due to increased central pulse BP in vessels with increased stiffness can pathogenetically determine LV diastolic dysfunction in patients with AH and may ultimately lead to heart failure [29]. This hypothesis is confirmed by the fact that the risk factors for heart failure include AH, atherosclerosis, and age, which are also associated with increased arterial stiffness and central BP. Early detection of heart failure without reduced ejection fraction is an urgent problem; functional diagnostic methods are actively used for its management [30, 31]. Therefore, the analysis of the elastic properties of blood vessels from the perspective of a comprehensive assessment of the effect of cardiovascular risk factors on the prognosis may be promising.

Assessment of Vascular Stiffness

Assessment of arterial stiffness was proposed as a marker of vascular aging. Since invasive assessment of arterial wall elasticity during vascular catheterization is quite laborious and not economically viable for screening, non-invasive methods, particularly volumetric sphygmography, dopplerography, and magnetic resonance imaging (MRI), are used widely. One of the primary methods for assessing arterial stiffness is the determination of pulse wave velocity (PWV) using sphygmography [32]. A value of carotid-femoral PWV of more than 10 m/s may indicate an increased risk of adverse cardiovascular events [33]. Modern devices with various tranducers are used to determine PWV. The Complior System (Colson, Les Lilas, France) includes a mechanical tranducer; methods based on applanation tonometry (for example, a «conventional» SphygmoCor device, AtCor Medical, West Ryde, NSW, Australia) include a Millar piezoelectric «tonometer». Devices such as the VP1000, Omron Healthcare (Kyoto, Japan), VaSera — N1000 (Fukuda Denshi, Japan) are based on the oscillometric method (Fig. 1).

VaSera — N1000 (1500) volumetric sphygmography devices enables to measure PWV on the ankle-brachial segment; the cardio-ankle vascular index (CAVI), ankle-brachial index (ABI), finger-brachial index (FBI) and augmentation index (AI) are automatically calculated based on the measurement [34] (Fig. 2, 3).

It should be noted that as a derivative of cardiopulmonary PWV, the CAVI index can be considered a parameter of "true arterial stiffness" that is less dependent on intravascular BP [35]. Table 1 shows threshold CAVI values in different age groups of the Russian population [34].

Besides the assessment of arterial stiffness based on PWV, analysis of aortic pulse pressure and augmentation index (AI) may be important for describing the state of the cardiovascular system since they characterize the elasticity, which has an effect on the formation of reflected vascular waves [31, 36].

Local vascular stiffness using imaging methods can be determined by analyzing the diameter of superficial and deep arteries in systole and diastole. Examples of the assessment of local aortic stiffness include MRI and ultrasound echo tracking [25, 32].

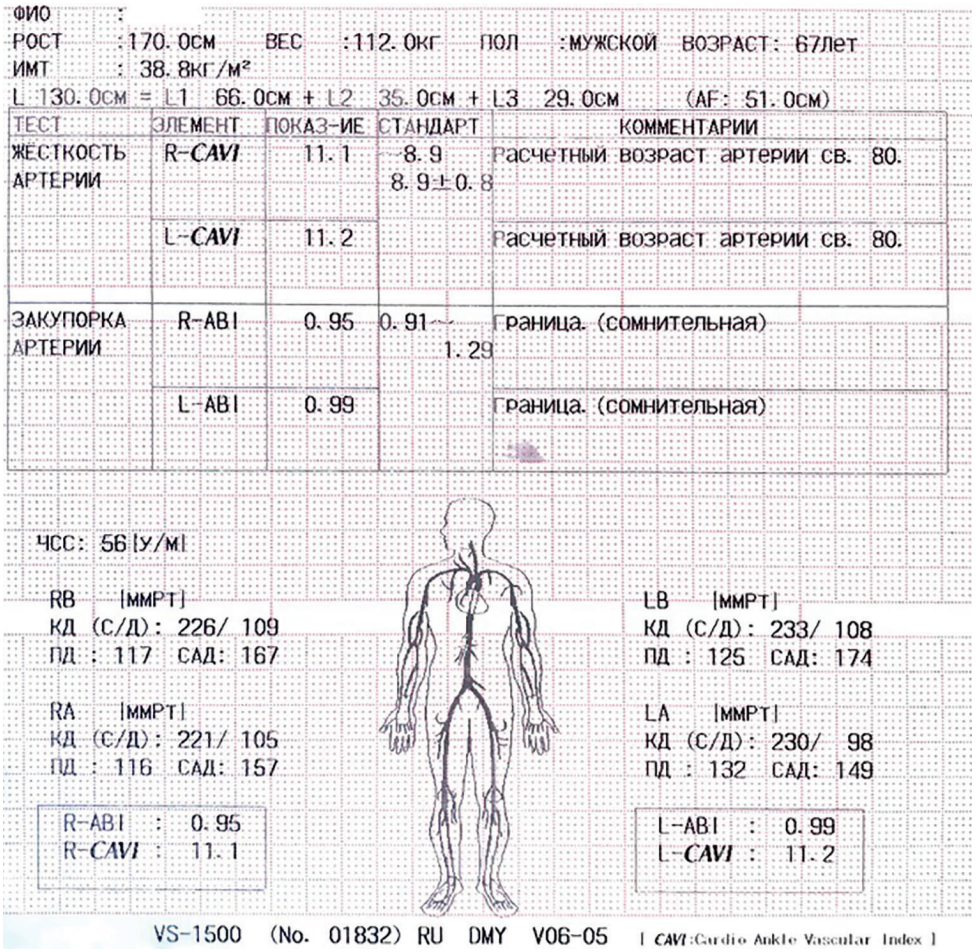

Figure 1. *CAVI evaluation Protocol. The figure shows (from top to bottom) ECG curves, phonocardiograms, plethysmograms from the right and left upper and lower extremities*

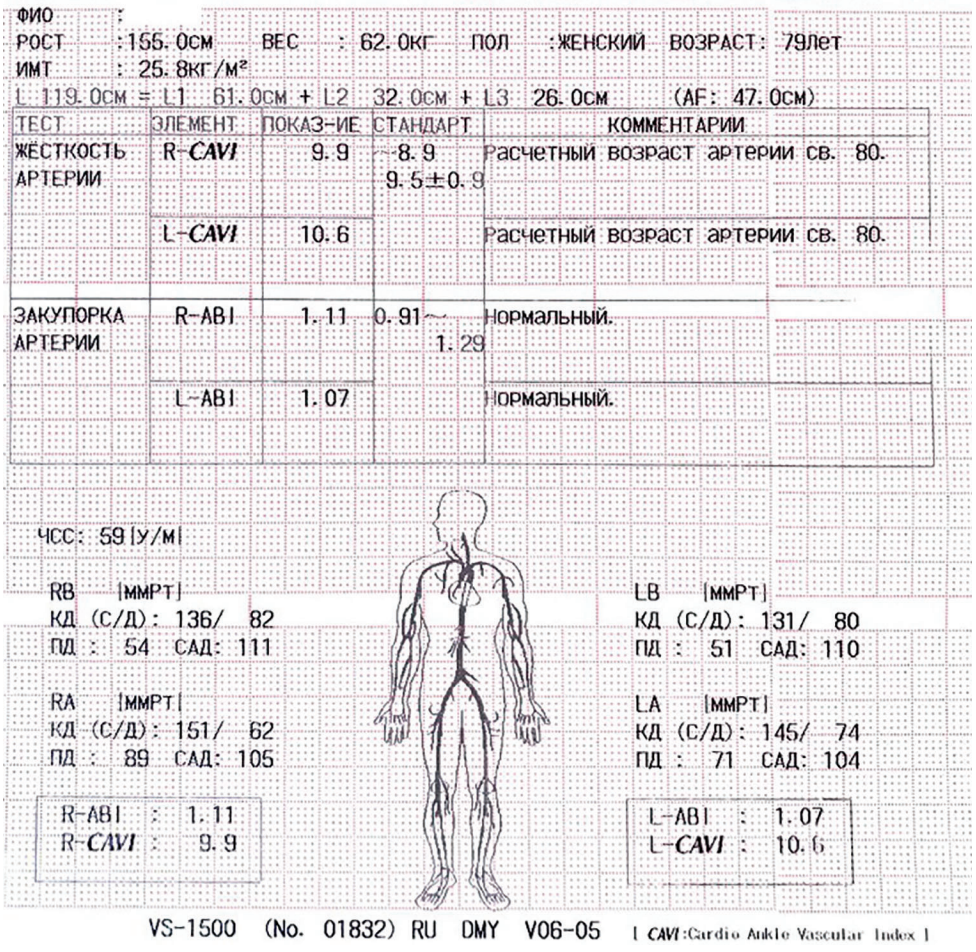

Figure 2. *CAVI evaluation Protocol, continued. The table shows the indicators of the cardio-ankle-vascular index (CAVI) on the right and left-top and the ankle-shoulder index (ABI) on the right and left-bottom, measured in a 79-year-old woman. The indicators presented in the table correspond to the age norm*

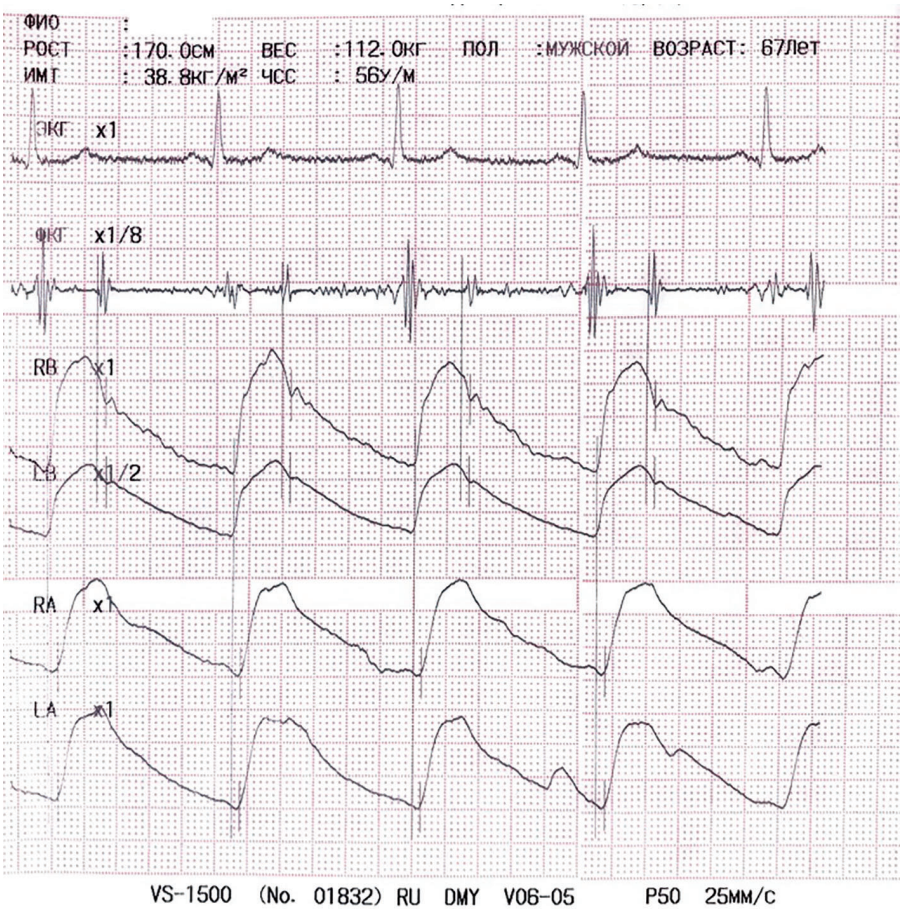

Figure 3. *CAVI evaluation Protocol, continued. The table shows the indicators of the cardiovascular-ankle-vascular index (CAVI) on the right and left-top and the ankle-shoulder index (ABI) on the right and left-bottom, measured in a 67-year-old man. Indicators (CAVI, ABI) presented in the table above the age norm (the upper limit of the norm for CAVI is 10, for ABI-0.9)*

Table 1. *CAVI indicators depending on age according to research data in the Russian population (M±SD) [34]*

Age	<20 years	21-30 years	31-40 years	41-50 years	51-60 years	61-70 years	>70 years
Indicator CAVI	6,7±0,76	7,2±0,61	7,4±0,63	7,55±0,7	8,0±0,67	8,5±0,64	9,8±1,51

Arterial Stiffness Application in Prediction

Assessment of arterial elasticity is important from a clinical point of view, since it correlates with the pathogenesis of a wide range of cardiovascular and cerebrovascular diseases, in particular, such as AH, cerebrovascular accident, vascular cognitive impairment [34, 37–39]. Improving knowledge and early detection of vascular aging can help improve the prevention of cardiovascular and cerebrovascular diseases. Cardiovascular diseases are known be asymptomatic for a long time since multiple organ lesions are based on a subclinical decrease in elasticity and an increase in arterial stiffness of main arteries [39]. Therefore, patients with subclinical lesions are at higher risk of developing a symptomatic disease compared with patients with traditional risk factors [39]. European (2018) and Russian (2020) clinical guidelines on AH allow using arterial stiffness parameters to increase the accuracy of cardiovascular risk stratification, especially in patients with moderate risk [40, 41]. The measurement of vascular stiffness and central aortic pressure should be recommended as one of the methods of cardiovascular risk stratification in patients with moderate risk on the SCORE scale (Systematic COronary Risk Evaluation), and if lesions of target organs are not detected by routine methods [27, 28]. There are data suggesting that central BP is a more reliable prognostic factor for death from cardiovascular diseases and all causes than BP in the brachial artery [29].

Several studies revealed the independent diagnostic and prognostic value of CAVI [41, 42]. According to some experts, CAVI may be useful for screening, ongoing monitoring and evaluation of the effect of treatment [34].

Conclusion

Remodeling of blood vessels during the aging of the human body is based on pathogenetic mechanisms that play an important role in the development of arterial hypertension. Assessment of stiffness in the main arteries is of great interest for the development of non-invasive diagnostic methods for pathophysiology, pharmacology,

and general practice, and is important for assessing cardiovascular risk and determining the prognosis for existing cardiovascular or cerebrovascular disease.

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Фомина Е.С. (ORCID: https://orcid.org/0000-0003-1747-108X): разработка концепции статьи, сбор и анализ литературных данных, написание рукописи, утверждение итогового варианта текста рукописи

Никифоров В.С. (ORCID: https://orcid.org/0000-0001-7862-0937): проверка критически важного интеллектуального содержания, редактирование текста, утверждение итогового варианта текста рукописи

Author Contribution:

All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication

E.S. Fomina (ORCID: https://orcid.org/0000-0003-1747-108X): development of the concept of the article, collection and analysis of literary data, writing of the manuscript, approval of the final version of the text of the manuscript

V.S. Nikiforov (ORCID: https://orcid.org/0000-0001-7862-0937): verification of critical intellectual content, editing of the text, approval of the final version of the text of the manuscript

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