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ARTERIAL WALL STIFFNESS IN CARDIOVASCULAR DISEASE

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Abstract:

Korotkow's blood pressure level is a relatively inaccurate predictor of cardiovascular events, requiring the introduction of new diagnostic methods. In recent years, assessment of the effect on arterial stiffness has become a very important characteristic of the efficacy of antihypertensive drugs.The assessment of stiffness (central aortic pressure, augmentation index and pulse wave velocity) contributes to a better definition of cardiovascular risk and is a criterion for target organ damage and the effectiveness of antihypertensive therapy. In particular, a pulse wave velocity greater than 12 m/s is a significant risk factor. Significant reductions in stiffness have been observed to a greater extent with combination therapy, predominantly for the combination of calcium antagonists and angiotensin converting enzyme inhibitors/angiotensin receptor blockers.

Keywords: Arterial stiffness, pulse wave velocity, arterial hypertension, antihypertensive drugs, combination therapy

RELEVANCE.

Cardiovascular disease in a broad sense often refers to a disease of the peripheral vessels of the heart and brain, which occurs due to atherosclerosis, i.e. large and medium arteries in the body have atherosclerosis, which is caused by stenosis and blood clogging. Currently, there is an increase in the prevalence of chronic heart failure (CHF), which is partly due to a significant decrease in mortality from this disease and an increase in patients' life expectancy against the background of modern advances in treatment [1, 2].

The current classifications and treatment of heart failure (HF) largely depend on left ventricular (LV) ejection fraction (EF) (3). At the same time, almost half of patients with symptoms of CHF have a normal EF - the so-called preserved LV ejection fraction (PHF) (4). In this group of patients, diastolic dysfunction is predominantly found, characterised by increased LV wall stiffness and impaired LV relaxation, resulting in altered filling of the LV [5]. CHF with preserved EF often occurs in diseases such as arterial hypertension (AH), infiltrative cardiomyopathy (in amyloidosis, haemochromatosis), restrictive cardiomyopathy, diabetes mellitus, obesity. Mortality in CHNSFV exceeds 20% within 1 year [6, 7], and the prognosis is not different from CHF with reduced EF. In 2007, the Heart Failure and Echocardiography Association of the European Society of Cardiology published an opinion on the diagnosis of CHNSFV, formerly called diastolic CH (8). Echocardiography (echocardiography) is used for 2 of the 3 criteria required for the diagnosis of CHNSFV. In CHF, there is an increase in arterial wall stiffness (AWS), which is considered to be the central mechanism underlying the increase in systolic and hence pulse pressure (PP) and the development of cardiovascular disease (AH, coronary heart disease (CHD).

An increase in arterial pressure (AP) can lead to a dramatic impairment of LV systolic and diastolic function with the occurrence of pulmonary oedema in patients with CHF [9]. In addition, in patients with increased AC stiffness, an increase in LV wall stiffness and impaired LV relaxation can be observed [10]. It is also possible the influence of early reflected wave, which leads to increased systolic load on LV and decreased duration of ejection phase in systole with decreased stroke volume [11]. The assessment of LV EF is one of the key methods for the diagnosis of CHF. Currently, a variety of methods are used, mainly EchoCG and Doppler studies, the results of which allow assessing the prognosis in patients with CHF, especially in the presence of LV systolic dysfunction [12, 13]. However, diagnostic tests are either of low accuracy or are labour-intensive and time-consuming, resulting in higher treatment costs. In addition, this kind of examination is not frequently used due to the limitations of technical equipment and skilled personnel (14). Pulse wave velocity in chronic heart failure One of the criteria for assessing LV

contractile function is pulse wave velocity (PWV) at the radial artery (dP/dt), which is independent of LV LV EF and postload levels [15]. In early systole, the LV generates blood pressure. The maximum rate of pressure rise roughly corresponds to the rate of pressure rise in the ascending aorta in the early phase of ejection, provided there is no aortic stenosis and without considering the pressure gradient associated with inertial forces during blood acceleration [16]. Проведенные ранее исследования в качестве гемодинамического индекса использовали индекс интенсивности пульсовой волны, определявшийся как dP/dt × (dU/dt) в любой точке кровеносной системы, где dP/dt и dU/dt являются производными АД и скорости [17]. The magnitude of the first peak of this waveform index can be derived from the formula (dP/dt)2/ρc, where ρ is blood density and c is SPV. The first peak has been found to be closely related to LV dP/dt index ($r = 0.74$; $p < 0.001$) in patients with CHD [17]. However, the index c is a marker of aortic wall stiffness, independent of LV contractile function, and ρ can be considered a constant. Thus, the key element of the equation is the dP/dt index. As the pulse wave travels along the arteries, it is reasonable to assume that its characteristics may change under the constant influence of local visco-elastic features as well as reflected waves, which may lead to systematic error and underestimate the delay time between the two measurement points [18]. However, J. M. Tartiere et al. (2007) demonstrated a strong association between SPV measured at the radial and carotid arteries ($r = 0.83$; $p < 0.001$) [19]. Increased AC stiffness in patients with CHF results from several factors [20]. Firstly, functional factors, such as impaired haemodynamics, decreased endothelial relaxing factor (nitric oxide) and increased neurohumoral factors may contribute to AC stiffness [21]. Secondly, the progression of arteriosclerosis associated with the atherosclerotic process leads to anatomical changes in the vascular wall and increases arterial stiffness, especially in elderly patients (22). A study by T. Meguro et al. (2009) found a statistically significant 18.1% increase in annual rehospitalisation and an 8.9% mortality in patients with high TMA measured at the brachial artery. Brachial artery SPV was found to be an independent prognostic factor, even after adjustment for age, sex, BP and brain natriuretic peptide levels [23]. The findings on rehospitalization rates and cardiovascular mortality (CVD) are consistent with the results of an earlier large-scale study in Japan, which reported one-year all-cause mortality in CHF patients with LV EF < 25%, end-diastolic LV diameter 60 mm, brain natriuretic peptide level 500 pg/mL, functional class III or with CHD at 15.0 11.4; 16.8; 16.3 and 10.8 %, respectively [24]. In addition, it was found that on rehospitalisation for exacerbation of CHF, the group with high brachial artery SPV had higher BP than the group with low SPV, despite similar compensatory haemodynamic parameters. Increased AP is associated with the development of LV dysfunction and clinical manifestations of CHF in the elderly and those with AH [28]. It is interesting to note that after the formation of systolic CHF, the results of studies on the prognostic value of AC stiffness show contradictory results. In patients with asymptomatic LV dysfunction [29] and systolic CHF [30], a positive correlation between increased AP and adverse outcome is found. At the same time, in patients with decompensated systolic CHF, as well as in patients with stable severe CHF, the relationship between PD and outcome is reversed; a low level of PD is an independent prognostic factor of mortality [31]. An explanation for the contradictory results may be the relationship between the heart with progressively impaired pumping function and the arterial system (ventricular-vascular interaction). A study by T. Weber et al. (2007) found no significant difference in aortic SPV in patients with non-ischaemic cardiomyopathy and in controls, furthermore, no significant correlation between SPV and PV was found in both the study population as a whole and in the group with preserved systolic function, This is in agreement with previous studies on carotid-femoral SPV (CFSPV) [32], while peripheral arterial SPV was found to be elevated in patients with systolic CHF [33]. In patients with impaired LV systolic function, there was a strong positive correlation between SPV at the aorta and LV contractile function indices. The results of several studies demonstrate a significant relationship between AC stiffness and small haemodynamic changes. One study found a significant increase in EFSPV with a sharp increase in heart rate (HR) in patients with an artificial pacemaker [34]. In healthy young men, a short LV ejection period determines an increase in CPV at rest and under conditions of increased sympathetic tone [35]. In a study conducted by J. M. Tartière et al. (2006), preserved PV was associated with increased CPPSV, a longer ejection period and low HR (36). M. Kawaguchi et al. in their study reported increased AC and LV wall stiffness in patients with CHFSPV compared with age-matched controls [37]. At the same time, contradictory results have been obtained in the assessment of proximal arterial wall stiffness. Using characteristic (wave) impedance as a direct marker of AC stiffness, G. F. Mitchell et al. and C. J. Pepine et al. found an increased stiffness of proximal arterial walls, while E. P. Kromer et al. showed the opposite results [33, 38, 39]. At the same time G. F. Mitchell et al. found an increase in CPPSV in patients with mild systolic or diastolic dysfunction treated with an angiotensin II receptor antagonist (candesartan) without an increase in CPPSV [33]. After prolonged vasodilator withdrawal, there was a tendency for stiffness of muscular arteries, such as the radial artery, to increase in proportion to the severity of systolic dysfunction compared to controls, however, no such changes were seen in the absence of prolonged vasodilator withdrawal .

CONCLUSION.

Thus, current studies emphasize the important role of elastic properties of the wall of the main and peripheral vessels, as well as the ventricular myocardium in the pathogenesis of CHF. There is a close relationship between the work done by the ventricles of the heart and the response of the wall of the main and peripheral vessels to the passage of blood through them. ATP analysis allows to estimate with high accuracy both elastic properties of arterial vascular wall of various calibers and LV performance. The assessment of PIV can be an effective diagnostic method in

certain groups of patients with CHF, e.g., in CHNSF, in whom diagnostic methods such as echoCG with EF assessment are insufficiently informative and do not reflect the severity of the disease.

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