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DEFINITION OF LEFT VENTURAL DIASTOLIC FUNCTION AND CHARACTERISTICS OF CHANGES IN HEART DISEASES

Summary. AH and IHD are the main causes of LVDD and diastolic HF (Mareev V.Yu., 2003). Violation of LV LV in patients with AH has a multifactorial nature and does not occur in isolation, but simultaneously with a violation of the morphofunctional state of other organs and systems, or with their pathogenetic participation, due to prolonged exposure to high blood pressure.

Key words: arterial hypertension. arterial pressure, ischemic heart disease, left ventricular ejection fraction.

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ОПРЕДЕЛЕНИЕ ЛЕВОЙ ВЕНТУРАЛЬНОЙ ДИАСТОЛИЧЕСКОЙ ФУНКЦИИ И ХАРАКТЕРИСТИКА ИЗМЕНЕНИЙ ПРИ ЗАБОЛЕВАНИЯХ СЕРДЦА

Резюме. АГ и ИБС - основные причины развития ДДЛЖ и диастолической СН (Мареев В.Ю., 2003). Нарушение ДФЛЖ у больных АГ имеет мультифакторную природу и происходит не изолированно, а одновременно с нарушением морфофункционального состояния других органов и систем, или при их патогенетическом участии, вследствие длительного воздействия высокого АД.

Ключевые слова: артеоиальная гипертензия. артериальная давления, ишемическая болезнь сердца, фракцией выброса левого желудочка.

Relevance. The significance and role of LVDD in the pathogenesis, clinical course, and prognosis of HF began to be determined only during the last three decades [4]. It has now been proven that diastolic disorders are the cause of HF in approximately 30-40% of cases [3]. Moreover, there is evidence that disturbances in the diastolic properties of the myocardium usually precede a decrease in LV pumping function and can even lead to the onset of HF symptoms in isolation [2].

AH and IHD are the main causes of LVDD and diastolic HF (Mareev V.Yu., 2003). Violation of LV LV in patients with AH has a multifactorial nature and does not occur in isolation, but simultaneously with a violation of the morphofunctional state of other organs and systems, or with their pathogenetic participation, due to prolonged exposure to high blood pressure [1].

According to the theory of "wear and tear" (Kushakovsky M.S., Balyabin A.A., 1991), hypertension is also the main cause of damage to the heart valves of the DC, which in old age, even without the formation of a defect, leads to the development of LVDD.

Purpose of the study. Based on the foregoing, the goal of the study was set: to find new mechanisms of pathogenesis, develop criteria for clinical and functional classification and give a comparative assessment of various types of treatment for left ventricular diastolic dysfunction in patients with hypertension and coronary artery disease.

Materials and research methods. The study included patients under the age of 70 years with a stable course of coronary heart disease (CHD) during the previous month, with a left ventricular ejection fraction (LVEF) of at least 45%. A mandatory criterion was the presence of a stable sinus rhythm in the patient.

Results of the study and their discussion. The features of the AH syndrome that contribute to the development of LVDD are the duration of its existence in the presence of a high level and daily load, both SBP and DBP, increased BP variability during the day, no decrease, increase and high BP load at night, as well as a high rate of morning rise HELL.

In the presence of LVDD in patients with AH, microalbuminuria occurs 6 times more often than in normal LV relaxation, while the identification of correlations between the fact of detection of microalbuminuria and LVDF indicators does not mean the direct involvement of microalbuminuria in the pathogenesis of impaired relaxation, but indicates the synchronous development of two processes - morphofunctional restructuring of the left ventricle and nephropathy, equally dependent on the severity of the AH syndrome, in particular, on the absence of a nocturnal decrease in blood pressure.

In most patients with AH, DD developed against the background of LV remodeling by the type of concentric hypertrophy with a tendency in the presence of volume overload to the formation of eccentric hypertrophy, and high correlations reliably confirmed the dependence of the development of LVDD on an increase in LV myocardial mass.

The development of LVDD in AH is facilitated by impaired endothelial function, which is expressed in a decrease in endothelium-dependent vasodilation and an increase in the serum concentration of endothelin-1 by 1.36 times, as well as vascular remodeling with an increase in the thickness of the intima-media complex and stiffness of the walls of the arteries.

Long-term hypertension with a predominant increase in the level, variability and daily load of SBP contributes to the development of DC AV, which is accompanied by an increase in the systolic rate of aortic blood flow and

pressure gradient between the left ventricle and aorta and is an independent risk factor for both hypertrophic remodeling and LV DD, progressing with increasing the number of valves affected by calcification, which does not allow even moderate DC AK to be considered a benign process.

Output. New criteria have been developed for determining the stage of LVDD based on a Doppler echocardiographic study of high-amplitude reflected motion signals and parameters of the transmitral blood flow, as well as an assessment of clinical and functional factors: features of the daily blood pressure profile, severity of MAU, the nature and degree of remodeling of the left ventricle and peripheral vessels, the degree of endothelial dysfunction, changes in HRV and the presence and prevalence of concomitant DCAC.

The effectiveness of the use of the myocardial cytoprotector metazidine in normalizing global and regional LVDD caused by ischemia and myocardial hibernation has been proven.

The combined therapy with ACE inhibitor enalapril and AAT-losartan has been proven to be more effective in reversing LV remodeling and normalizing LVEF, compared with monotherapy with each of these drugs separately.

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