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Blood Pressure and Contractile Function of Heart Ventricles at the Early Stages of Hypertonic Process

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In rabbits, arterial hypertension was characterized by progressive elevation of systolic and diastolic blood pressure. The contractile function of the left ventricle augmented, but its potential working capacity decreased. Opposite changes were observed in the right ventricle. It was hypothesized that the compensatory mechanisms in the right ventricle during arterial hypertension are triggered at the very onset of the pathology, while in the left ventricle they develop later.

Key Words: *arterial hypertension; myocardium; contractility; diastolic function*

Three stages are traditionally distinguished in the development of arterial hypertension, each stage produces peculiar shifts in systemic hemodynamics [5]. Arterial hypertension changes myocardial contractility, which frequently leads to the development of cardiac insufficiency [1,4,6-8]. In addition, arterial hypertension is often accompanied by impairment of diastolic function of the heart [2,9-11]. According to WHO classification, the first stage of essential hypertension is characterized the absence of objective alterations in visceral organs. However, the data are accumulating that some functional and morphological alterations develop in the heart at the very onset of arterial hypertension, which finally leads to the formation of "hypertonic heart" [5].

Our aim was to assess the contractile function of the myocardium in the right and left ventricles and to study the compensatory mechanisms that counterbalance myocardial overload during the initial phase of arterial hypertension.

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MATERIALS AND METHODS

Experiments were performed on 25 male Chinchilla rabbits weighing 2.7-3.8 kg. The animals were divided into 5 groups (control and 4 experimental groups). In experimental rabbits, arterial hypertension was modeled according to Goldblatt by constriction of the abdominal aorta to $^1/_3$ initial diameter proximally to the origin of the renal arteries. One, 2, 4, and 6 weeks after surgery, systolic (BP_{syst}) and diastolic (BP_{diast}) blood pressure were measured. The real and maximum (extremal) parameters were measured in the left and right ventricles under normal conditions and during 5-sec occlusion of aorta and pulmonary artery, respectively [3,5]. This procedure was used to measure the real and maximum pressure as well as the real and maximum rate of intraventricular pressure rise in both ventricles. The end-diastolic pressure was also measured in the left and right ventricles. The measurements were performed on a Mikard soft-hardware setup (a digitizer coupled with a computer). BP was measured with an electromagnetic transducer calibrated before and after the experiments using a water column. The digitized signals were fed into PC, in which a fragment of blood pressure curve was written with Micam original software. BP was measured in

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acute experiment in common carotid artery, whose proximal end was cannulated with a plastic tube filled with physiologic saline and connected to electromagnetic pressure transducer. Functional state of the heart was assessed by the parameters of contractile activity of the myocardium. One of the basic methods for evaluation of this state is direct measurement of blood pressure in ventricular cavities. The data were processes statistically using original software developed in Department of Pathological Physiology in Russian University of People Friendship. The calculated statistical parameters were the means, SE, SEM, Student's *t* test). Correlation analysis was carried out to reveal strong ($r>0.75$), moderate ($0.50<r<0.75$), and weak ($r<0.50$) dependence between the examined parameters.

RESULTS

Both BP_{syst} and BP_{diast} increased significantly to the end of week 1 of modeling the renovascular arterial hypertension (Fig. 1). By week 2 BP somewhat decreased in comparison with the previous week, but still significantly surpassed the control value. To the end of week 4, BP_{syst} and BP_{diast} stabilized at high levels, so the difference between these values measured on weeks 4 and 6 was insignificant.

The real pressure in the left ventricle markedly increased starting from hypertension week 1 and stabilized to the end of week 4 (Table 1), which was probably caused by increased peripheral resistance to blood flow.

At the beginning of hypertension process, the maximum pressure in the left ventricle did not increase and even significantly decreased (Table 1). Elevation of the maximum pressure in the left ventricle was observed only at the end of week 4. Therefore, during the development of arterial hypertension, the potential efficiency of the left ventricle myocardium decreased.

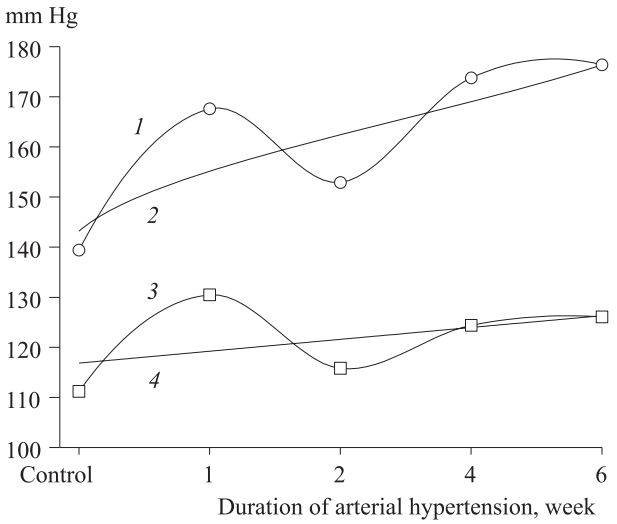

Fig. 1. Dynamics of systolic (*1*) and diastolic (*3*) pressure and the corresponding trends (*2* and *4*) during the development of arterial hypertension.

During the first stage of arterial hypertension, the real pressure in the right ventricle significantly decreased and began to recover only at the end of hypertension week 6, *i.e.* at the stage of stable compensation (Table 2). In the right ventricle, the maximum pressure significantly increased as early as by the end of week 1; during week 2 this increase persisted just as a trend; and to the end of week 4 this parameter significantly increased again and attained maximum values (Table 2). It can be hypothesized that during arterial hypertension the compensatory mechanisms are triggered in

TABLE 1. Parameters of Contractile Activity of the Left Ventricle (*M*±*m*)

Parameter	Control	Hypertension, week			
		1	2	4	6
VPR LV, mm Hg	150.12±1.76	186.80±2.47*	157.60±2.98*	185.10±2.76*	182.80±2.18*
VPmax LV, mm Hg	250.80±3.56	222.60±6.63*	207.32±4.69*	285.3±2.2*	274.52±2.38*
EDP LV, mm Hg	7.60±0.87	9.48±0.42	8.04±0.77	11.60±1.08*	8.48±0.31
The rate of pressure rise in LV, mm Hg/sec	2760±100	4020±110*	3210±70*	3470±380	3580±120*
The rate of pressure drop in LV, mm Hg/sec	-1920±30	-2450±70*	-2090±80	-2640±260*	-2930±140*
Maximum rate of pressure rise in LV, mm Hg/sec	4140±200	4740±220*	4620±320	7270±270*	5720±170*
Maximum rate of pressure drop in LV, mm Hg/sec	-3330±180	-3315±120	-3535±160	-4130±130*	-4060±180*

Note. Here and in Tables 2: VPR, real ventricular pressure; VPmax, maximum ventricular pressure; EDP, end-diastolic pressure; LV, left ventricle; RV, right ventricle. $p<0.05$ *compared to the control.

TABLE 2. Parameters of Contractile Activity of the Right Ventricle (*M*±*m*)

Parameter	Control	Hypertension, week			
		1	2	4	6
VPR RV, mm Hg	35.20±0.73	25.10±0.48*	24.80±0.56*	30.28±1.49*	38.6±1.3*
VPmax RV, mm Hg	50.80±1.09	56.30±2.18*	52.2±2.3	71.00±1.08*	61.32±1.18*
EDP RV, mm Hg	3.92±0.37	3.32±0.32	4.10±0.39	6.28±0.15*	5.8±0.3*
The rate of pressure rise in RV, mm Hg/sec	920±20	490±20*	510±20*	750±20*	810±30*
The rate of pressure drop in RV, mm Hg/sec	-900±20	-500±20*	-570±20*	-630±20*	-790±20*
Maximum rate of pressure rise in RV, mm Hg/sec	2030±40	1500±80*	1320±50*	1570±30*	1600±40*
Maximum rate of pressure drop in RV, mm Hg/sec	-1240±30	-770±20*	-860±40*	-780±20*	-920±20*

the right ventricle at the onset of hypertension process and precede similar mechanisms in the left ventricle.

The absence of significant correlation between BP_{syst} and real pressure during the first two weeks of hypertension process shows that in this period the increase in working capacity of the right ventricular myocardium is not related to elevated BP, but results from other mechanisms.

During the development of the hypertension process, the end-diastolic pressure in the left and right ventricles only tended to increase (Tables 2, 3), this rise became significant only to the end of week 4. This suggests that during the first stage of arterial hypertension, diastolic function of the heart ventricles was not impaired.

Thus, the first stage of arterial hypertension is characterized by instability in BP rise, which agrees with previous data on heterogeneous structural and functional alterations developed in the heart during the onset of compensatory processe [5]. During this period, the contractility of the left ventricular myocardium increases, while its working capacity decreases. At the same time, contractility of the right ventricle slightly decreases, and its potential efficiency augments. Thus, the compensatory mechanisms in the right ventricle are triggered earlier, than in the left one, and these mechanisms are not related to increased peripheral resistance to the blood flow. The first stage of arterial hypertension is not characterized by impairment of diastolic function of the heart ventricles.

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