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Central Hemodynamics, Myocardial Mass of The Left Ventricle of The Heart and Diastolic Function of The Ventricles of The Heart in Patients with Arterial Hypertension after a Six-Month Follow-Up

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ABSTRACT

Ninety-six patients with mild, moderate and severe arterial hy-pertension were studied. The control group consisted of 26 healthy subjects. Sixty-eight patients have had essential hypertension for 10+7.7 years, 28 patients -chronic glomerulonephritis for 8.915.3years. The study methods included echocardiography and Doppler echocardiography. Left ventricular hypertrophy was found in 75% of patients with essential hypertension and in 60.7% of patients with chronic glomerulonephritis. Eccentric right ventricular hypertrophy was found in the most of hypertensive patients. The majority of hypertensive patients had prolonged active relaxation and impaired Doppler spectrum of left ventricular and right ventricular filling, impaired passive myocardial properties and increased end-diastolic pressures. A long-term period of no pharmacological treatment (6 months, 2 years) leaded to the progression of left ventricular hypertrophy and diastolic dysfunction. Both events shared the common mechanism of progression. Sporadic antihypertensive treatment with Adelphan" was unable to prevent the above unfavorable changes. The main factors of myocardial hypertrophy progression were increased blood pressure and thethickening of myocardial walls. Short-term pharmacological treatment with nifedipine (30-40 mg per day) and amlodipine (10 mg per day) produced similar hypotensive effects with a weak tendency to an improvement in diastolic function and no effect on left ventricular myocardial mass. Long-term treatment with amlodipine improved left ventricular and right ventricular active relaxation, caused diastolic unloading of left atrium, significantly decreased left ventricular myocardial mass, predominantly improved the spectrum of diastolic filling and passive diastolic properties of the right ventricle despite no significant differences in the effects of both agents of blood pressure. The similar effects of ifedipine were less pronounced. A decrease in blood pressure, improvement in diastolic function, regression of left ventricular mass share the common mechanisms of progression.
***Key words**: Arterial hypertension, diastolic dysfunction, myocardial mass, long-term follow-up.*

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INTRODUCTION

The problem of arterial hypertension as an essential or hypertension disease, as well as symptomatic hypertension, is an important social problem in developed countries with economies in transition, since such an economic situation, on the one hand, creates a background for an increase in the incidence of this disease and the associated increase in morbidity and mortality, on the other hand, for treatment of hypertension with modern pharmacological agents, subject to long-term use of drug treatment [4,7,12,269].

Calcium antagonists (CA) are now included in the group of basic drugs for the treatment of patients with hypertension [3,6,113,132]. Multicenter, placebo-controlled studies of the use of calcium antagonists in the treatment of hypertension have shown high clinical efficacy (studies: HOT, TOMHS, Systr-Eur, STONE, Syst-China) in terms of preventing the development of cerebral stroke [64,67,97,103,170,184].

This study was based on the fact that the work was to select patients with AH with two nosological forms with the same duration of AH, randomized by sex and age, in order to analyze in the future and due to what factors the parameters of LVML and diastolic function of the heart change. At the initial stage, a group of 96 patients with hypertension was formed in which 68 patients were diagnosed with GB, and 28 patients had a hypertensive form of chronic glomerulonephritis.

During long-term therapy, nifidipine (drug "Corinfar" by AWD Germany) and amlodipine (drug "Norvask" from Pfizer USA) were used. mg. The dose of the drug was selected individually by titration. During long-

term follow-up of patients and during long-term therapy, A/D levels were monitored using the Kortokov method, central and intracardiac hemodynamics were assessed by echocardiography and Doppler echocardiography, which were performed using the Toshiba SSH 60A system (Japan) in accordance with the recommendations of the American Association of Echocardiography.

MATERIAL AND METHODS

In accordance with the practical tasks set in the study and the study protocol, we observed two groups of patients with AH: in the first group, the duration of observation was six months, and the second - two years. In accordance with the study protocol, each group of AH patients included patients with both AH and CGN; at the initial stage of the study, we did not find a significant difference in most of the studied parameters between the groups. Upon further observation of the patients, it was found that some patients, despite the recommendations to carry out antihypertensive therapy, did not take any antihypertensive therapy (these patients were later combined by us into groups of patients without treatment), while the other part of patients with hypertension took enalapril only in cases of clinical symptoms. such as headache, a feeling of heaviness in the occipital region (later on, these patients were combined by us into a group of patients with hypertension of irregular therapy). In accordance with the study protocol, data analysis was carried out in three areas: central hemodynamics, left ventricular myocardial mass, diastolic function of the heart. In the group of patients with hypertension who did not take antihypertensive therapy, the number of patients was also sufficient to analyze the pattern of changes in parameters depending on the etiology of hypertension, i.e. separately in patients with GB and in patients with CGN.

RESULTS

Tables 1-8 present the results of the six-month follow-up.

Table No. 1 Changes in the parameters of central hemodynamics in groups of patients with hypertension during a six-month follow-up, M±σ

	6 months without treatment		6 months of irregular therapy		P		
	before	after	before	after			
Index:	1	2	3	4	1-3	1-2	3-4
mean arterial pressure, mm. Hg	175±30,1	192±32,0	181±26,8	196±22,2	nd	**	*
DBP, mm.Hg	112±18,0	120±19,7	118±12,8	125+12,3	nd	*	*
Heart rate, beats/min	74,0±12,8	73,1±11,8	72,2±8,9	69,1±7,4	nd	nd	nd
DP	99,3±25,9	106±30,5	101±19,6	103±16,0	nd	nd	Nd
UI, ml / m 2	42,1±9,8	43,7±13,0	43,8±9,6	45,1±11,1	Nd	nd	nd
SI, l / min * m 2	3,10±0,89	3,19±1,11	3,16±0,79	3,15±0,97	nd	nd	Nd
OPPS, dyn*s*cm-5	1950±400	2070±509	1930±488	2090±529	Nd	nd	nd
LVEF, %	62,3±6,2	62,3±10,5	63,8±8,3	65,2±7,1	nd	nd	Nd
Vcf, s-1	1,24±0,19	1,23±0,34	1,21±0,21	1,19±0,24	nd	nd	nd

Note:*-p<0.05; **-p<0.01; nd-differences are unreliable

Table number 2. Changes in the parameters of central hemodynamics in groups of patients with GB and CGN during a six-month follow-up M±σ

	GB CGN		GB CGN				
	GB CGN	GB CGN	GB CGN	GB CGN			
Index:	1	2	3	4	1-3	1-2	3-4
mean arterial pressure, mm.Hg	174±28,9	189±30,0	178±32,5	196±35,0	nd	*	*
DBP, mm.Hg	110±13,0	116±17,7	114±22,9	124±21,8	nd	nd	*
Heart rate, beats/min	73,0±13,2	73,5±11,6	75,3±12,8	72,6±12,5	nd	nd	nd
DP	96,3±21,9	104±25,3	103±30,6	109±36,6	nd	nd	nd
UI, ml / m 2	38,0±5,8	39,3±5,9	47,0±11,5	49,0±17,1	*	nd	nd
SI, l / min * m 2	2,74±0,48	2,88±0,61	3,54±1,09	3,57±1,45	*	nd	nd
OPPS, dyn*s*cm-5	2080±414	2120±511	1790±330	2010±519	*	nd	*
LVEF, %	61,5±4,9	61,9±7,6	63,2±7,5	62,7±13,5	nd	nd	nd
Vcf, c^{-1}	1,22±0,14	1,21±0,20	1,27±0,24	1,25±0,47	nd	nd	nd

Note:*-p<0.05; **-p<0.01; nd-differences are unreliable

From the presented data, it can be seen that the irregular intake of an antihypertensive drug does not prevent the growth of A\D, and, consequently, the severity of the course of the disease increases, and a separate prognosis worsens, in the early stages of the formation of the course of hypertension, hyperkinetic changes in hemodynamics predominate (this can also be indirectly evidenced by the fact an increase in such a parameter as the LV ejection fraction, which reflects the overall contractility of the left ventricle of the heart.) The pattern of the relationship between the increase in SI and the change in EF can be traced both in the group of untreated AH patients as a whole and in the group of patients and in the group of patients with GB: so in the AH group, UI - EF r=0.575(P<0.001), in the HD group r=0.792(P<0.001) a similar pattern can be observed in the group of six-month follow-up with irregular UI therapy – LV EF r=0.763 (P<0.01). All this leads to a slight increase in the value of C (cardiac index), expressed to a large extent in patients with hypertension who did not take any antihypertensive therapy. In patients with GB, this phenomenon may also be associated with the activation of the sympathoadrenal system, leading to a slight increase in heart rate. That. It is through an increase in cardiac output in the early stages of AH formation that the heart tries to adapt to the conditions of its functioning under conditions of an increase after exercise.

From the presented data it can be seen that already six months of constantly elevated blood pressure is enough (in the absence of any effective drug intervention) for such an adaptive reaction of the LV as its hypertrophy to manifest itself (the mass index of the LV myocardium increases over six months in the group of patients with hypertension who do not take antihypertensive therapy, by 7.1%; p<0.05), which is implemented not only due to an increase in the end-diastolic volume of the left ventricle - EDV (in the group of patients with hypertension without treatment, the increase was 5.6%), but most importantly due to an increase in the thickness of the interventricular septum - TMZhPd and the posterior wall of the left ventricle - TZSLZhd. This fact is also confirmed by the data of the correlation analysis, so in general, for the group of untreated patients with AH, the correlation coefficient is iMMLVpenn-EDVVt: r=0.72 (p<0.001), =0.545 (p<0.01). Similar data were obtained by us for the group of patients with GB, at the same time, according to the correlation analysis, the leading role in the group of patients with CGN is still played by the increase in EDVlt (correlation coefficient iMLV LV penn-EDV t: r=0.815, p<0.001). It should be noted here that in patients with AH, the leading hemodynamic factor in the increase in LVML is the increasing afterload (increase in blood pressure), and this pattern can be traced both in the group of AH patients without treatment and in the group of AH patients with irregular intake of an antihypertensive drug.

Table 3. Changes in the mass of the myocardium of the left ventricle and indices of the functional state of the left ventricle of the heart in groups of patients with hypertension during a six-month follow-up, M±σ

	6 months without treatment		6 months of irregular therapy		P		
	before	after	before	after			
Index:	1	2	3	4	1-3	1-2	3-4
TMZhPd, cm	1,22±0,21	1,25±0,20	1,16±0,16	1,15±0,11	nd	nd	nd
TZSLZhd, cm	1,11±0,14	1,14±0,17	1,07±0,11	1,10±0,08	nd	Nd	nd
TSpzh, cm	0,74±0,12	0,80±0,12	0,73±0,07	0,74±0,11	nd	*	nd
KDRLV, cm	5,08±0,48	5,19±0,56	5,22±0,36	5,23±0,32	nd	nd	nd
KDOLZht, ml	123±28,5	130±34,6	131±20,0	131±18,5	nd	nd	nd
MMLJpenn, gr	279±91,3	302±113	271±57,7	276±54,0	nd	*	nd
LVMIpenn, g/m2	152±49,3	163±58,9	140±30,4	143±29,7	nd	*	nd
EDV/MMLV, ml/g	0,52±0,10	0,51±0,10	0,56±0,11	0,55±0,05	nd	nd	nd
VMN, dyne/cm2	343±72,7	365±66,4	384±58,2	396±41,1	nd	nd	nd
iKDNSLzh, * 103 dyne / cm2	69,2±57,8	63,0±29,7	58,8±12,4	55,9±14,1	nd	nd	nd
IFS, mmHg/g*min	49,4±15,1	49,4±12,6	50,0±13,5	50,8±12,4	nd	nd	nd

Note:*-p<0.05; nd-differences are unreliable

Table 4. Changes in the mass of the myocardium of the left ventricle and indices of the functional state of the left ventricle of the heart in groups of patients with hypertension and CGN during a six-month follow-up, M±σ

	GB		CGN				
	before	after	before	after			
indicator:	1	2	1	2	1-3	1-2	3-4
TMZhPd, cm	1,24±0,12	1,23±0,15	1,19±0,28	1,27±0,26	nd	nd	*
TZSLZhd, cm	1,12±0,11	1,15±0,13	1,11±0,17	1,12±0,21	nd	nd	nd
TSpzh, cm	0,77±0,12	0,79±0,13	0,72±0,12	0,80±0,12	nd	nd	*
KDRLV, cm	4,96±0,33	5,03±0,38	5,22±0,59	5,37±0,69	nd	nd	nd
KDOLZht, ml	116±17,6	121±20,7	132±36,7	142±44,4	nd	nd	nd
MMLJpenn, gr	268±44,6	280±59,9	293±128	328±155	nd	nd	*
LVMIpenn, g/m2	142±22,9	148±31,0	165±68,1	182±78,3	nd	nd	nd
EDV/MMLV, ml/g	0,49±0,08	0,49±0,09	0,55±0,11	0,54±0,12	nd	nd	nd
VMN, dyne/cm2	328±50,1	336±33,1	362±91,7	399±80,0	nd	nd	nd
iKDNSLzh, * 103 dyne / cm2	73,7±24,8	73,1±33,2	63,8±18,3	50,8±19,6	nd	nd	nd
IFS, mmHg/g*min	48,9±15,6	50,4±10,8	49,9±15,0	48,1±14,8	nd	nd	nd

Note:*-p<0.05; nd-differences are unreliable

Table No. 5 Changes in the diastolic function of the left ventricle of the heart in patients with hypertension according to a six-month follow-up, M±σ.

	6 months without treatment		6 months of irregular therapy		P		
	before	after	before	after			
Index:	1	2	3	4	1-3	1-2	3-4
nFIR, ms	91,6±26,9	98,4±25,6	86,1±19,7	87,1±17,5	nd	nd	nd
PE, m/s	0,55±0,18	0,54±0,17	0,57±0,11	0,56±0,10	nd	nd	nd
RA, m/s	0,56±0,14	0,57±0,13	0,62±0,08	0,57±0,07	nd	nd	nd
RE/RA	1,07±0,52	1,04±0,51	0,94±0,22	0,99±0,22	nd	nd	nd
1/3 FN, %	50,6±13,4	49,6±13,1	49,3±12,4	51,8±13,3	nd	nd	nd
ION, %	78,3±14,5	81,3±14,4	77,4±16,3	76,5±16,9	nd	nd	nd
Kolzhlp, ml/mmHg	43,1±24,2	33,8±24,5	41,2±17,9	35,0±12,0	nd	nd	nd
KDD, mmHg	10,4±6,4	9,3±3,9	9,5±2,1	8,8±2,1	nd	nd	nd
VLPt, ml	57,1±18,0	65,5±25,2	59,4±14,8	58,7±11,8	nd	nd	nd

Note: nd-differences are not significant

With regard to the parameters of diastolic function of the left ventricle of the heart a in the group of patients with hypertension without taking antihypertensive therapy during a six-month follow-up, all changes are only in the nature of a pronounced trend. Changes in the diastolic function of the left ventricle relate to the processes of active relaxation, which slows down, which manifests itself in the form of an elongation of the period of isovolumic relaxation (the FIR period). In the absence of therapy, the leading factor leading to elongation of active relaxation is an increasing afterload (nFIRlv-TMZhPD correlation coefficient r=0.375; p<0.05, and this pattern is most pronounced in patients with CGN: r=0.571; p<0.05 There is also a certain relationship between changes in the parameters of the diastolic function of the heart, namely, between changes in active relaxation and changes in the nature of diastolic filling of the left ventricle, so in general for the group of hypertension without treatment /3FNlzh r=0.496; p<0.01; similar changes can be traced as in the group of patients with GB: nFIRlzh-RE/RAlf r=0.505; p<0.05, nFIRlvg- KDDlv r=0.725; p<0.001 and nFIRlvl-1 /3FNlzh r=0.631; p<0.01, and in the group of patients with CGN

Table 6: Changes in the parameters of diastolic function of the left ventricle in groups of patients with GB and CGN during a six-month follow-up, M±σ.

	GB		CGN				
	before	after	before	after	P		
Index:	1	2	3	4	1-3	1-2	3-4
nFIR, ms	99,3±15,3	108±24,6	74,1±23,3	79,4±23,3	nd	nd	nd
PE, m/s	0,49±0,11	0,47±0,11	0,63±0,22	0,64±0,19	*	nd	nd
RA, m/s	0,58±0,12	0,58±0,11	0,52±0,15	0,55±0,16	nd	nd	nd
RE/RA	0,86±0,25	0,82±0,21	1,33±0,65	1,30±0,63	*	nd	nd
1/3 FN, %	48,6±11,8	45,9±12,8	53,0±15,3	54,1±12,4	nd	nd	nd
ION, %	77,9±14,8	82,6±15,0	78,8±14,7	79,7±14,1	nd	nd	nd
Kolzhlp, ml/mmHg	36,8±20,4	31,4±20,3	50,7±26,9	36,7±29,3	nd	nd	nd
KDD, mmHg	11,5±4,1	10,8±4,0	9,11±8,4	7,41±3,0	nd	nd	nd
VLPt, ml	63,3±16,9	63,8±17,7	49,4±16,8	67,9±32,7	*	nd	*

Note:*-p<0.05; nd-differences are unreliable

Table 7. Changes in the diastolic function of the right ventricle of the heart in patients with hypertension according to six-month follow-up, M±σ.

	6 months without treatment		6 months of irregular therapy				
	before	after	before	after	P		
Index:	1	2	3	4	1-3	1-2	3-4
nFIR, ms	38,6±21,3	44,6±31,4	34,1±18,6	20,7±6,9	nd	nd	*
PE, m/s	0,38±0,08	0,38±0,06	0,40±0,07	0,44±0,09	nd	nd	**
RA, m/s	0,40±0,09	0,37±0,09	0,36±0,05	0,35±0,07	nd	nd	nd
RE/RA	1,0±0,25	1,07±0,32	1,12±0,25	1,29±0,37	nd	nd	nd
1/3 FN, %	49,1±12,0	47,5±11,7	54,3±11,6	57,2±13,6	nd	nd	nd
ION, %	79,2±12,9	82,5±13,7	74,4±11,9	72,9±13,8	nd	nd	Nd
Kolzhlp, ml/mmHg	27,4±16,7	21,8±14,4	25,8±11,8	22,8±6,4	nd	Nd	nd
KDD, mmHg	9,8±2,7	8,2±2,3	8,0±2,0	6,7±1,1	*	nd	nd

Note:*-p<0.05; **-p<0.01; nd-differences are unreliable

Table number 8. Changes in the parameters of diastolic function of the right ventricle in groups of patients with GB and CGN during a six-month follow-up, M±σ.

	GB		CGN				
	before	after	before	after	P		
Index:	1	2	3	4	1-3	1-2	3-4
nFIR, ms	37,6±16,2	37,0±12,7	40,0±26,9	58,7±41,1	nd	nd	*
PE, m/s	0,37±0,06	0,37±0,06	0,40±0,09	0,40±0,07	nd	nd	nd
RA, m/s	0,39±0,11	0,38±0,10	0,40±0,08	0,37±0,08	nd	nd	nd
RE/RA	0,98±0,26	1,03±0,24	1,01±0,25	1,13±0,40	nd	nd	nd
1/3 FN, %	51,1±13,5	48,2±12,8	46,7±9,9	46,7±10,5	nd	nd	nd
ION, %	75,8±14,7	80,9±14,6	83,4±9,2	84,5±12,8	nd	nd	nd
Kolzhlp, ml/mmHg	24,0±14,3	21,8±13,8	31,6±19,0	21,7±15,6	nd	nd	nd
KDD, mmHg	9,8±2,8	8,5±2,4	9,8±2,7	7,8±2,1	nd	nd	*

Note:*-p<0.05; **-p<0.01; nd-differences are unreliable

DISCUSSIONS

As can be seen from tab. 7, and 8 in relation to the diastolic function of the right ventricle of the heart, the changes described in relation to the left ventricle are of a similar nature and manifest themselves in a similar form. Interestingly, in relation to the right ventricle, irregular intake of an antihypertensive drug for six months, even in conditions of an increase after exercise on the left heart, to a certain extent, can accelerate the processes of active relaxation and improve the spectrum of its diastolic filling, nevertheless,

in relation to passive diastolic properties, irregular antihypertensive therapy is not able to prevent the development of negative changes.
If we analyze the dynamics of right ventricular diastolic function indices in the group of untreated HD and CGN patients, it turns out that it is very similar, and in terms of active relaxation and compliance, changes in CGN patients even exceed similar changes in HD patients.
At the end of this section of the work, we compiled regression models of multiple linear regression for the increase in LVMM in the GROUPS of AH patients without therapy and AH patients irregularly taking enalapril, which took into account geometric factors, the growth factor after exercise, and changes in a number of parameters of LV diastolic function. The following results were obtained. From the presented data, it can be seen that in both the first and second models, only geometric factors are practically involved, such as an increase in the thickness of the interventricular septum or the posterior LV wall, as well as an increase in the end-diastolic volume of the LV cavity of the heart. It should be noted that in the absence of therapy, an additional factor in the progression of LVH is an increase after exercise and a change in end-diastolic pressure in the LV cavity. In contrast, in the case of irregular therapy, these factors, as well as other hemodynamic factors, did not participate in this model of the process.
Thus, the absence of any intervention in the natural course of the disease of a patient with hypertension, even for six months, leads to an increase in the severity of the latter in terms of blood pressure, which is accompanied by the progression of hypertrophy and diastolic dysfunction of the left and right ventricles of the heart. Irregular intake of an antihypertensive drug at the same time of observation of patients with hypertension is not able to prevent the progression of left ventricular hypertrophy and cause the reverse development of existing disorders of diastolic function of both the left and right ventricles of the heart, and only a few are able to slow down the rate of their development, and these changes primarily affect the passive diastolic properties of the myocardium, and, consequently, AH in this group of patients continues to progress. Therefore, in the treatment of a patient with hypertension, it is necessary to use drugs with the hemodynamic effects desired under the given conditions, which must necessarily include the possibility of the drug affecting the size of the cavity in the direction of its reduction, as well as the thickness of the myocardial walls, along with a decrease in the factor of increased after exercise (adequate correction of blood pressure).

CONCLUSIONS

1. The absence of an antihypertensive drug in patients with arterial hypertension with a follow-up period of six months and two years, regardless of the etiology of hypertension, leads to the progression of hypertrophy of the walls of the left ventricle and dilatation of its cavity. The absence of antihypertensive therapy also leads to the development of eccentric hypertrophy of the right ventricle of the heart.
2. Irregular antihypertensive therapy in patients with arterial hypertension, according to long-term observation, does not provide advantages over the option of no antihypertensive therapy in terms of preventing the growth of blood pressure, as well as the development of hypertrophy of the walls of the myocardium of the left and right ventricles of the heart. Irregular antihypertensive therapy can only slightly slow down the rate of progression of myocardial hypertrophy.
3. In conditions of refusal to take antihypertensive therapy in patients with hypertension, according to long-term observation, diastolic heart function disorders develop, which manifest themselves as elongation of active relaxation, disturbances in the Doppler spectrum of diastolic filling of the ventricles of the heart and deterioration of passive diastolic properties of the left and right parts of the heart, which is the most typical for patients with hypertension with the second stage of the disease.
4. Short-term antihypertensive therapy with amlodipine and nifedipine does not cause significant changes in the geometry of the heart, but can cause an acceleration of active relaxation, an improvement in the spectrum of diastolic filling of the ventricles of the heart and passive diastolic properties of the myocardium, as well as a decrease in diastolic overload of the left atrium and a decrease in end-diastolic pressure in cavities of the ventricles of the heart.
5. Data from six-month course therapy with amlodipine show its advantages over nifedipine in terms of reduction in LV myocardial mass during therapy (by 9% compared to 5.2% in the nifedipine group, n.d.), implemented in the case of the use of amlodipine due to a decrease in the thickness of the LV walls , as well as by reducing the size of its cavity. Both drugs also lead to the development of regression of hypertrophy of the wall of the right ventricle of the heart.
6. According to six-month therapy, amlodipine and nifedipine predominantly have a positive effect on the right side of the heart, which is manifested by a shortening of relaxation, an improvement in the diastolic filling spectrum and an improvement in the passive diastolic properties of the atrial and ventricular chambers, and the effects of amlodipine exceed those of nifedipine.

REFERENCES

1. J.F. Ren, S.B. Pancholy, A.S. Iskandrian et al.(1994). // Doppler echocardiographic evaluation of the spectrum of left ventricular diastolic dysfunction in essential hypertension. Am. Heart J. Vol. 127, No 4. Pt 1. P. 906-913
2. Drayer J.I.M., Weber M.A., de Young J.L. (1983). Blood pressure as a determinant of cardiac left ventricular muscle mass . Arch. Intern. Med.-Vol. 143. -P. 90-92.Esp. Cardiol.
3. D. Levy, K.M. Anderson, D.D. Savage et al. (1988). Echocardiographically detected left ventricular hypertrophy: of prevalence and risk factors Ann. Intern. Med. Vol. 108. -P. 7-13.
4. / D.D. Savage, J.I.M. Drayer, W.L. Henry et al. (1979). Echocardiographic assessment of cardiac anatomy and function in hypertensive patients. Circulation. Vol. 59. -P. 623-636. P. Kleine, E. Me
5. A. Sagie, E.J. Benjamin, M.lar alderisi et al. (1993). Echocardiographic assessment of left ventricular structure and diastolic filling in elderly subjects with borderline isolated systolic hypertension (the Framingham Heart Study) Am. J. Cardiol. Vol. 72, No 9. -P. 662-665.
6. E. Loufer, G. Jennings, E. Dewar et al. (1986). Echocardiographic measurements of left ventricular hypertrophy in untreated essential hypertension. Clin. Experim. Pharmacol. Phisiol. -Vol. 13, No 4. -P. 295-299.
7. R.F. Davies, H. Habibi, W.P. Klinke et al. (1995). Effect of amlodipine, atenolol and their combination on myocardial ischemia during treadmil exercise and ambulatory monitoring. Am J. Coll. Cardiol. -1995. -Vol. 25. -P. 619- 625
8. / M. Packer, C.M. O'Connor, J.K. Ghali et al. (1996). Effect of amlodipine on morbidity and mortality in severe chronic heart failure. New Engl. J. Med. Vol. 335. -P. 1107-1114.Cential
9. E. Nardi, G. D`Ignoto, V. Volpe et al. (1993). Effect of calcium antagonist amlodipine on left ventricular mass and diastolic filling in essential hypertension [abstract]. Am. J. Hypertens. Vol. 6. (Pt2). -P. 106A.
10. J.S. Gottdiener, D.J. Reda, B.M. Massie et al. (1997). Effect of single-drug therapy on reduction of left ventricular mass in mild to moderate hypertension. Circulation. Vol. 95. -P. 2007-2014.
11. J.R. Gonz'alez- Juanatey, J.M. Garc'ia-Acuna, C. Calvo-G`omez et al. (1997). Effect of verapamil and nitrendipine on the left ventricular mass and function (systolic and diastolic) in arterial hypertension/ Rev. Esp. Cardiol. 47(6):375-83

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