TABLE 3. Index of Atherosclerotic Plaque Involvement of Thoracic Aorta in Animals with Experimental Atherosclerosis Administered Polysaccharide

Group	Index of aortic involvement, %
1 (control)	41.3±5.4
2	38.2±7.1
3	24.3±4.7*
4	20.7±2.9*

charide is a substance which evidently loses its potency under the effect of gastric juice.

Hence, oral polysaccharide has a marked antiatherosclerotic effect, manifested in the delayed development of experimental atherosclerosis and in the reduction of atherosclerotic lesions of the aorta. The latter effect is indicative of angioprotector properties. The polysaccharide is less effective in manifest hypercholesterolemia. In a certain dose this substance is not inferior in specific activity to atheroid and has a similar effect on the lipid spectrum. Porcine duodenum polysaccharide can thus be considered promising material for the development of an antiatherosclerotic drug.

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Functional and Morphological Interventricular Relationships during Chronic Overload of the Primarily Intact Left Heart in a Model of Experimental Vasorenal Hypertension

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Modern cardiology is characterized, above all, by the accumulation of rich experience in conservative and surgical management of various types of damage to the left and right heart. Nevertheless, studies of the operating mechanisms of each of the ventricles are most frequently performed separately, which contradicts the principle of an integral approach to the investigation of the heart as a unified system of organs, and makes it difficult to reveal both general and individual patterns of activity of each of the ventricles.

The aim of our study was to explore the functional and morphological relationships between the ventricles during hemodynamic overload of the primarily intact left heart on an experimental model of vasorenal hypertension.

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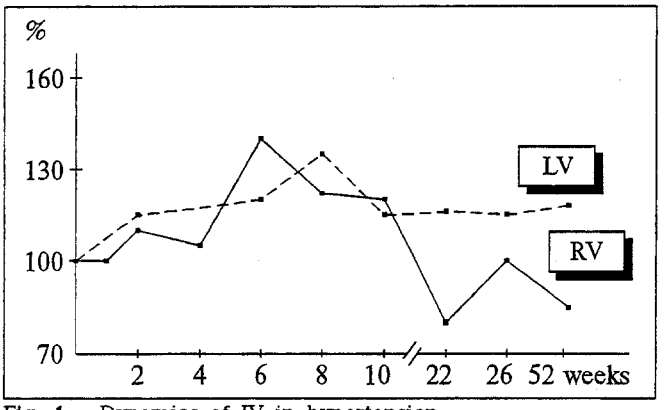

Fig. 1. Dynamics of IV_r in hypertension.

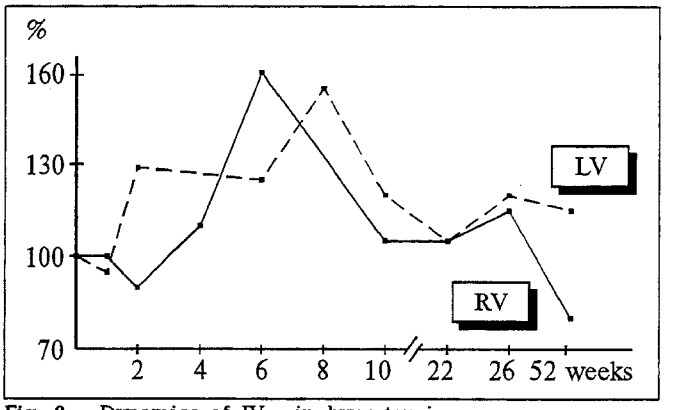

Fig. 2. Dynamics of IV_{max} in hypertension.

MATERIALS AND METHODS

Experiments were carried out on 178 adult male Chinchilla rabbits weighing 2.5-3.2 kg. Vasorenal arterial hypertension was simulated by occlusion of the abdominal aorta by one third of its initial diameter directly above the site of origin of the renal arteries [1].

Functional and morphological investigations of the ventricles were performed in groups comprising 14-16 rabbits during weeks 1, 2, 4, 6, 8, 10, 22, 26, and 52 postoperation. Under conditions of an acute experiment the systolic (AP_{max}) and diastolic (AP_{min}) arterial pressure (for monitoring the development of arterial hypertension) were electromanometrically recorded on a Mingograf-82 (Siemens-Elema, Sweden) device by cannulating the left common carotid artery in novocain-anesthetized (0.5% solution) animals.

After cannulation of both ventricles, the real (IV_r) and maximal (IV_{max}) intraventricular pressures were isometrically recorded on a Mingograf-82 polygraph for a 5-sec occlusion of the ascending aorta or of the pulmonary artery for the left (LV) or right (RV) ventricle, respectively.

TABLE 1. Dynamics of Systolic (AP_{max}) and Diastolic (AP_{min}) Arterial Pressure in Rabbits in Different Stages of Development of Experimental Vasorenal Hypertension.

Time of observation	AP_{max}, mm Hg	AP_{min}, mm Hg
Control	136±3	109±3
1 week	164±5	138±5
2 weeks	178±6	144±5
4 weeks	184±5	153±3
6 weeks	182±6	146±6
8 weeks	183±9	149±6
10 weeks	180±3	145±3
22 weeks	188±4	157±7
26 weeks	188±6	147±10
52 weeks	158±6	137±4

Note. All results reliably differ from the control.

Ultrathin sections (250-350 μ) of the papillary muscles were examined under Tesla-BS-540 (Czechoslovakia) and Siemens-10 (Germany) transmission electron microscopes (magnification 10,000-80,000). Morphometric analysis was performed after Paukov et al. [2] in 15 electronograms (EG) obtained for each rabbit at a magnification of 22,000. Mitochondrial areas were measured with the aid of a digitizer on a Datamini PC using software developed in our Department.

The real (FAS_r) and maximal (FAS_{max}) functional activity of the structures showing the level of real and maximally attainable load to be overcome by the unit weight of each ventricle was calculated. For this purpose the dry weight of the myocardium was determined for both ventricles, this allowing us to rle out the possible effect of tissue edema on the actual contractile weight of the myocardium. The thickness of the myocardial fibers was also measured in the EG. The coefficient of energetic efficacy of the mitochondria (CEEM) was calculated [3].

All the numerical data were statistically processed after Student. The difference of the means was regarded as reliable for $p<0.05$.

RESULTS

A reliable increase of AP_{max} and AP_{min} was observed (Table 1). Beginning with the second week of simulation, a reliable rise of IV_r and IV_{max} was observed in the LV; also of interest was a reliable increase of IV_r and IV_{max} in the RV in the 6th and 8th weeks, when the maximal rise of the indexes of contractility was noted in the LV.

Beginning with the 4th week, the FAS in the LV gradually decreased due to the development of left ventricular myocardial hypertrophy, which was shown by the findings on the wet and dry weight. On the whole, the FAS_r and FAS_{max} in the RV also exhibited a trend toward a decrease as hypertension developed.

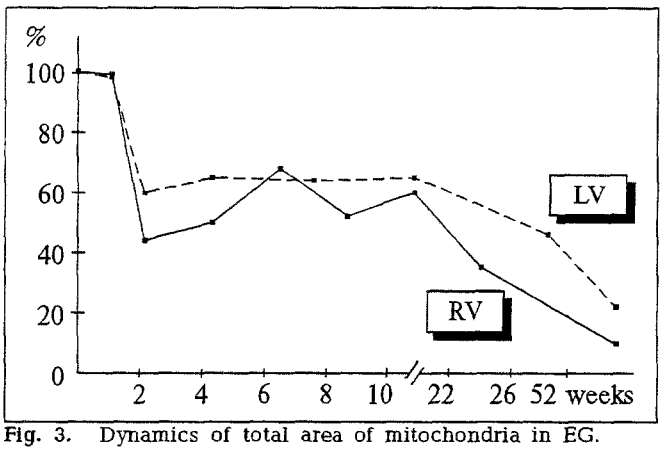

Fig. 3. Dynamics of total area of mitochondria in EG.

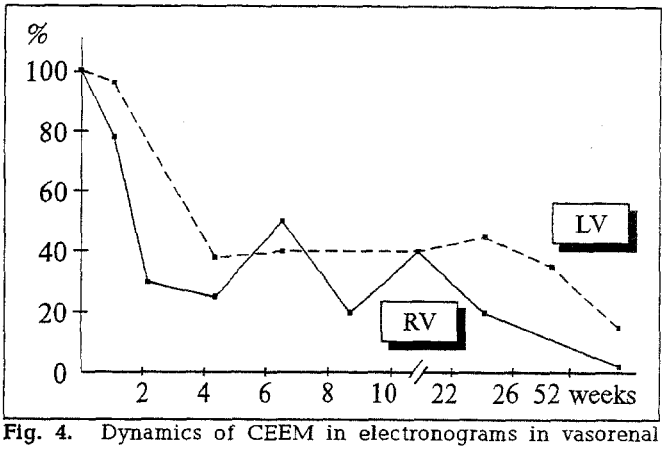

Fig. 4. Dynamics of CEEM in electronograms in vasorenal hypertension.

Analysis of the dynamics of the relative values of IV and FAS showed the following regularities: IV_r in both ventricles (Fig. 1) exhibited similar dynamics, but the functional activity of the RV peaked ahead of that for the LV; in addition, in the final stages of hypertension development IV_r in the RV was more reduced in comparison with IV_r in the LV. In the final stages of the experiment (beginning with the 22nd week) IV_{max} was higher in the LV than in the RV (Fig. 2). The changes of the relative parameters FAS_r and FAS_{max} had, on the whole, the same time course as the changes of the indexes of ventricular contractility.

The morphometric analysis of mitochondria in the LV and RV (Fig. 3) showed that the degree of decrease of the mitochondrial area and of number of cristae in the mitochondria was higher than that in the LV, this, in turn, being responsible for the much lower relative value of CEEM. Of note is the discordance between the dynamics of CEEM in the left and right ventricles during the development of arterial hypertension (Fig. 4).

The results of correlation analysis indicated that in the first stage of hypertension, the positive relationships between the parameters of contractility of the ventricles became markedly stronger. The correlation coefficients for the pairs (IV_r in the LV) - (IV_{max} in the RV) and (IV_r in the RV) - (IV_{max} in the LV) markedly increased, attesting to synchronization of RV activity and its decisive influence on the LV potential. In the second stage synchronization of the activity of the LV and RV abruptly decreased, this, in our view, being conducive to the broadening of the adaptive potencies. In the third stage of hypertension the degree of desynchronization now went beyond the bounds of adaptation requirements and formed the basis for an imbalance in the cardiac activity; this, along with a high level of AP, increased the myocardial load and contributed to an accelerated development of decompensation mechanisms.

To sum up, during the development of experimental vasorenal hypertension, the RV is coincidentally involved in the pathological process along with the LV, but works more actively and is decompensated faster than the left ventricle. However, throughout the process of adaptation, the RV preserves more degrees of freedom than does the LV. The degree of disturbances of the functional relationships between the ventricles changes as hypertension develops: at first, these disturbances provide for effective adaptation of the heart, while in the final stages of the process they are transformed into such a pathological form that they cause an imbalance in the entire heart activity and speed up the formation of a wear-and-tear complex in the myocardial structures of the "hypertrophized heart."

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