Assessment of Differences between Responses of the Left and Right Heart Ventricles to Prolonged Systemic Arterial Pressure Elevation in Rabbits with Experimental Renoprival Arterial Hypertension

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Until now, characteristics of only the left ventricle have been mainly used clinically in assessing the state of a diseased heart and in developing new methods for its treatment [7-9], which is hardly justifiable. Our investigations in which contractile function of the heart was studied over time in animals with experimental renoprival arterial hypertension, have shown [2,6] that during the various stages of the disease the contractile functions of the left and right ventricle are altered unequally, and that alterations in the right ventricle may precede those in the left. These findings agree with clinical observations by a number of scientists [1-5,10,11] that the right ventricle becomes involved in the disease process concurrently with, or even before, the left ventricle in a variety of cardiovascular disorders, including hypertension. So far, however, no clinical or experimental analyses have been undertaken to assess prognostically the differences in contractile function between the left (LV) and right (RV) ventricle. In this study, we determined differences between the responses of the LV and RV to sustained elevation of arterial pressure in rabbits with renoprival arterial hypertension.

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MATERIALS AND METHODS

For the study, 100 male Chinchilla rabbits weighing 2.5-3.5 kg were used. They were assigned to 10 groups, 10 rabbits in each. One group served as control. All rabbits in the other nine groups were operated upon to narrow the abdominal aorta by one-third of its diameter at the origin of the renal arteries, which led subsequently to the development of persistent arterial hypertension [2, 6]. One, 2, 4, 6, 8, 10, 22, 26, and 52 weeks after the operation, the diastolic (AP_{min}) and systolic (AP_{max}) arterial pressures and peak systolic pressure (real intraventricular pressure, or IP_r) were measured in the cavities of the left and right ventricles (LV ID_r and RV ID_r) of the operated and control rabbits. In addition, the maximal intraventricular pressure, which characterizes the maximal achievable function by the left or right (LV IP_m and RV IP_m) was determined; by relating the values obtained to the weight of the corresponding ventricles, the real and maximal intensities of their functioning were defined (IF_r and IF_m).

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TABLE 1. Predicting the Time Course of Some Cardiovascular Parameters from Their Values in Stage 1 or 2 of the Disease Process in Rabbits with Experimental Renoprival Arterial Hypertension

Parameter	Group	Stage of disease process, weeks								
		1st			2nd			3rd		
		1	2	4	6	8	10	22	26	52
LV IP_r, mm Hg	Measured values	106	139	137	135	169	133	116	133	128
	Values predicted from stage 1	–	–	–	165	190	219	523	698	4574
	Values predicted from stage 2	–	–	–	–	–	–	137	135	123
RV IP_r, mm Hg	Measured values	24	23	26	38	27	25	25	28	22
	Values predicted from stage 1	–	–	–	27	29	31	45	51	117
	Values predicted from stage 2	–	–	–	–	–	–	6.8	4.5	0.29
LV IP_m, mm Hg	Measured values	214	232	233	237	264	235	224	220	228
	Values predicted from stage 1	–	–	–	248	261	276	376	407	819
	Values predicted from stage 2	–	–	–	–	–	–	230	227	202
RV IP_m, mm Hg	Measured values	43	47	45	62	54	51	40	44	41
	Values predicted from stage 1	–	–	–	47	48	49	55	56	74
	Values predicted from stage 2	–	–	–	–	–	–	28	23	6.5
LV IF_m, mm Hg	Measured values	293	258	233	209	211	208	226	196	200
	Values predicted from stage 1	–	–	–	165	131	104	26	17	0.85
	Values predicted from stage 2	–	–	–	–	–	–	206	205	199
LV IF_m, mm Hg	Measured values	126	118	105	131	98	107	96	92	81
	Values predicted from stage 1	–	–	–	93	82	73	35	29	5.8
	Values predicted from stage 2	–	–	–	–	–	–	55	47	12

The results were processed on an IBM PC/AT computer using statistical software which we had developed and which enabled us not only to determine the significance of differences by Student's *t* test, but also to perform correlation analysis and to predict from the results of regression analysis the time course of the above-mentioned indicators of ventricular function.

RESULTS

To be able to evaluate unambiguously the differences between the responses of the LV and RV to a sustained elevation of systemic arterial pressure, we resorted to mathematical prediction. This method had previously been used by one of us [2] to assess the extent to which the course of arterial hypertension during its 3rd (final) stage depended on the values of the parameters characterizing cardiovascular function in its 1st and 2nd stages. It was found that predicting the development of hypertension from parameter values measured during the 2nd stage yielded results close to those obtained experimentally in the final stage, whereas predicting from parameter values measured during the 1st stage produced results that diverged from experimental data so widely as to be incompatible with life. Hence it was logically concluded that during stage 2 of experimental hypertension the cardiovascular system functions at a new level with a good potential for adaptation, but that stage 1 of the hypertensive process (transitory hypertension), during which compensatory mechanisms are still in the process of formation, is characterized by the establishment not only of adaptational but also of pathogenetic mechanisms.

In the study described here, mathematical predictions were made for certain functional indicators of both the LV and RV (Table 1).

If follows from this table that predicting parameters (indicators) of contractile function for the LV in stage 3 from those measured in stage 1 gives quite unrealistic results. In contrast, the results of predictions made for the LV in stage 3 from stage 2 data are commensurate with those obtained experimentally.

The situation is different with the RV. The differences between experimentally measured parameter values and the parameter values predicted for the RV in stage 2 and, in the case of RV IP_r, even in stage 3 from the respective values in stage 1 are much smaller than the differences seen for the LV. This may be attributed to the fact that the adaptational capabilities of the RV are far greater than those of the LV, so that during the stage at which compensatory mechanisms are being established its function is compromised to a much lesser extent.

The reverse situation obtains when parameters of the RV in stage 3 are predicted from their values in stage 2: the divergence of predicted values from experimental data is now wide.

In our view, the above discrepancies between the results of predictions for the RV and LV may be explained as follows. As our previous studies have shown [2, 6], during stage 2 of the hyper-

tensive process compensatory mechanisms are established and the cardiovascular system is functioning at a new level with a new range of adaptational possibilities for the heart. Actually, however, the adaptation proves to be limited because the LV functions under conditions of a stably increasing load. In other words, the system becomes more rigid and inevitably involves the RV too, which thereby loses degrees of its freedom, i.e., its inherent capacity for a very plastic adaptation to changing conditions; as a result, it undergoes decompensation more rapidly than the LV.

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Effect of Direct (Galvanic) Current on the Ultrastructure of the Normal Isolated Perfused Heart and during Postischemic Reperfusion

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Galvanization by itself and as a means of therapeutic electrophoresis is used to treat cardiac patients with stenocardia of the 1st, 2nd, and 3rd functional classes and myocardial infarction also in the convalescence and postconvalescence stages [1]. There have been a few reports of heparin electrophoresis being used to prevent thromboembolic complications, and magnesium electrophoresis has been used to arrest stenocardia in acute myocardial infarction [3]. The clinical effect of direct current (DC) therapy in the treatment of different cardiac ischemic disorders is well known, but the mechanisms of the cardiac component involved in the action of this physical factor have not been clarified. There is hardly any information about the effect of DC on the subcellular organization of the normal and ischemic myocardium.

The aim of the present investigation was to study the effect of DC on the ultrastructure of the normal isolated perfused heart and in the course of its postischemic reperfusion.

MATERIALS AND METHODS

Experiments were carried out on 34 male Wistar rats weighing 280-320 g using a model of an iso-

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