PECULIARITIES OF REFLEX REGULATION OF HEMODYNAMIC CHANGES UNDER THE INFLUENCE OF TRANSVERSELY DIRECTED ACCELERATION FORCES

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The perpendicular direction of the acceleration vector in relation to the longitudinal axis of the body is extremely advantageous physiologically: the effect of the mechanical factor on the organism and especially on the cardiovascular system is diminished. In studies of this question [5-7], changes in hemodynamics with the longitudinal direction of the acceleration vector are explained as the result of an acute rise in hydrostatic pressure. Under given conditions the blood is distributed among different parts of the body under the influence of the force of inertia, provoking a disruption in the blood supply to different organs. With acceleration directed at right angles to the longitudinal axis of the body, the circulatory system does not experience such a marked effect of hydrostatic pressure. However, changes in the functions of respiration and circulation (arising as a consequence of the increased constriction of the thoracic and abdominal cavities and being expressed in an increased respiratory rate) a fall in mean arterial pressure, and a decrease in the minute volume of blood [2-4] are observed in this case. Under the conditions described, in which the effect of hydrostatic pressure on the cardiovascular system is brought to a minimum, a more favorable possibility is created for the evaluation of the mechanism of hemodynamic changes resulting from acceleration. With this goal a series of acute experiments was performed on dogs which underwent acceleration first with intact reflex regulation of the circulation and then after bilateral denervation of the carotid sinuses.

EXPERIMENTAL

The experiments were performed on 24 dogs of both sexes weighing 10-15 kg. Acute experiments were carried out on 35 aminals with intact carotid sinuses and on 38 after denervation. Under morphine (0.75 mg/kg), chloralose (50 mg/kg), and nembutal (10 mg/kg) anesthesia the dogs were incubated for closed system artificial respiration. Under oscilloscopic control a probe was introduced into the cavity of the left ventricle (via the left carotid artery) and into the aorta (via the femoral artery). For recording of pressure the probes were connected to a tensometric recording apparatus rigidly connected to the arm of a centrifuge. The recorders were placed at the level of the probed cavity. The longitudinal axis of the recorder was directed parallel to the radius of the centrifuge arm. The animal underwent overloading of 9 g for one minute on a centrifuge with arm radius of 3.7 m in the direction of the chest and spine. Then bilateral denervation of the carotid sinuses was performed and the dog was spun again. The left ventricular and aortic pressures were recorded, as well as the EKG in two leads on a "Mingograf" V-42 apparatus before, during and after the centrifugation.

For characterization of the changes in minute volume of blood and the peripheral resistance we used the following formulas [8]:

$$V_m = P_p \cdot R, \tag{1}$$

$$W = \frac{P_{av}}{V_m}, \tag{2}$$

TABLE 1. Changes in Function of the Cardiovascular System Under the Influence of a Perpendicularly Directed Force of 9 g for 1 min

Index		Before spinning	Force		After spinning			
			30 sec	60 sec	1st min	3rd min	5th min	7th min
	Rate (in strokes/min)	130	169	184	138	130	130	130
		160	179	191	162	160	160	160
Left ventricular pressure (in mm of Hg)	Systolic	159	134	161	175	164	162	158
		184	129	158	195	191	181	185
	Diastolic	13	15	10	13	13	13	13
		16	15	14	16	16	16	16
	Mean after ejection phase	147	124	150	164	152	152	150
		170	119	138	177	174	168	172
	Mean after systole	127	104	120	134	119	127	126
		140	96	108	148	131	138	139
Aortic pressure (in mm of Hg)	Systolic	136	105	139	153	142	139	140
		152	96	119	162	157	147	153
	Diastolic	90	79	111	104	102	98	98
		117	72	96	115	121	113	116
	Pulse pressure	46	26	28	49	40	41	42
		35	24	23	47	36	34	37
	Mean after systole	122	95	126	133	129	128	126
		137	85	106	143	141	140	138
	Mean after cycle	107	86	120	122	116	112	109
		130	79	101	132	135	129	132
Minute volume (in relative units)		5 980	4 394	5 152	6 762	5 200	5 430	5 460
		5 600	4 296	4 393	7 614	5 760	5 440	5 920
Peripheral resistance (in relative units)		0,0204	0,0215	0,0244	0,0196	0,0248	0,0237	0,0232
		0,0244	0,0197	0,0240	0,0188	0,0262	0,0257	0,0233

Note: In the first line—indices with intact carotid sinuses; in the second—after their denervation.

where V_m = the minute volume; P_p = the value of the pulse pressure; R = the rate of cardiac contraction; W = the peripheral resistance; and P_{av} = the value of the average aortic pressure. The values obtained with formulas do not permit quantitative evaluation of the minute volume and peripheral resistance. However, the use of these indices gives a relatively true representation of the direction of change in minute volume and peripheral resistance.

RESULTS

The mean values of the data obtained in the experiments are presented in Table 1. In all experiments the changes were synonymous. Initially, perpendicularly directed acceleration force on anesthetized dogs with intact carotid sinuses causes decrease in arterial pressure, speeding of cardiac activity, decrease in minute volume, and rise in peripheral resistance. The left ventricular diastolic pressure does not change.

It is without question that the mechanism of such hemodynamic changes includes the entire chain of interrelated reflex acts. Transversely directed acceleration force produces constriction of the thoracic and abdominal organs, disturbances of the respiratory function (inspiration is made difficult), and disturbances of the hemodynamics of the pulmonary blood circulation (a redistribution of the blood in the lungs occurs) [1]. This leads to a decrease in the blood inflow into the left heart, and as a result, to a decrease in the values of left ventricular minute volume and to a fall in arterial pressure.

With an increase in the peripheral resistance and an increase in the cardiac activity, which are observed from the very beginning to the very end of the overload effect, to the very end of the level of arterial pressure is restored

with a certain rise above the initial value. The values of the mean systolic pressure in the left ventricle and the aortic pulse pressure are elevated, but remain less than the starting level. The values of left ventricular minute volume are altered in an identical manner.

Thus, during the action of overload on anesthetized dogs two phases of change in the cardiovascular system are observed: a phase of disturbed circulatory function and a phase of compensation. The latter begins against a background of continuing action but proceeds, evidently, against insufficient blood flow into the left heart. In the first minute after spinning, an increase in the arterial pressure and minute volume are observed. The hyperfunction of the heart which occurs is evidently a compensatory reaction in response to the disruption in blood flow to various organs and tissues of the organism. Between 3 and 7 min after the end of centrifugation the functional state of the cardiovascular system is almost restored. It is characteristic that the level of arterial pressure, approaching the initial figure at the seventh minute, is sustained against a background of increased vascular tone and some decrease in minute volume. The initiation of compensatory mechanisms at the moment the overload effect begins occurs by the afferent impulses from the reflexogenic zones of the cardiovascular system, mainly the sinoaortic zone, which leads to a reflex increase in cardiac activity and elevation of vascular tone.

Denervation of the carotid sinuses is accompanied by a pressor effect. The heart rate increases and the vascular tone rises. The systolic pressure rises less than the diastolic, as a result of which the pulse pressure decreases and the minute volume falls a little. Against this background, at the start of the overload effect a more marked fall in arterial pressure is noted, which at the end of the acceleration has a tendency to increase, but not attain the initial level. This is related to the fact that the vascular tone falls during the course of centrifugation. In the first minute after spinning, a small increase in the arterial pressure relative to the initial level is noted. This difference is observed less in the corresponding period in animals with intact carotid sinuses. In this connection it is important to note that after denervation of the carotid sinuses a delay in the compensatory phase is observed. This phenomenon is but weakly apparent after spinning and is related, probably, to the later inclusion of the aortic reflexogenic zone. An effect on other receptive fields in the vascular bed is not excluded. In the course of 3 to 7 min after spinning a more marked swing of minute volume and peripheral resistance values is noted relative to the initial level. This lability in the normalization of hemodynamic changes is explained by the absence of reflex autoregulation of circulation by such important a center as the carotid sinus.

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