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LYMPH FLOW RATE AND PROTEIN-ELECTROLYTE COMPOSITION OF THORACIC DUCT LYMPH IN EXPERIMENTAL ACHOLIA

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Acholia is accompanied by changes in many of the organs and systems of the body [2, 6]. The bile deficiency adversely affects the state of the digestive organs above all. It has also been shown that the composition and quantity of lymph flowing along the thoracic duct (TD) vary by a greater degree than the peripheral blood depending on the metabolic changes taking place in organs of the gastrointestinal tract [5]. In the investigation described below, the state of the blood and lymph circulations and also the protein and electrolyte composition of the lymph in TD were studied in experimental acholia.

EXPERIMENTAL METHOD

Experiments were carried out on 14 cats of both sexes weighing 2.5-3.5 kg. The animals were divided into two groups: 1) control animals ($n = 8$), 2) cats with experimental acholia ($n = 6$). A model of acholia was created by introducing a polyethylene tube in the proximal direction into the bile duct. The distal portion of the bile duct was ligated near the point where it enters the duodenum. The end of the tube was exteriorized on the anterior abdominal wall. Lymph was obtained from the experimental animals by cannulation of TD at the point where it enters the mouth of the jugular vein (under thiopental anesthesia). The rate of the lymph flow was determined by measuring the volume of lymph (in ml/kg body weight) escaping from TD through the cannula in 1 min. The central venous pressure (CVP) was determined by means of Waldman's apparatus in the posterior vena cava. Investigations on animals of the experimental group were carried out on the 20th day of production of experimental acholia. The animals were killed by injection of a lethal dose of the anesthetic.

EXPERIMENTAL RESULTS

Acholia was shown to be accompanied by marked changes in the lymph flow and also in the electrolyte and protein composition of the lymph (Table 1). The rate of lymph flow in the animals with acholia was only half of that in the control. Concentrations of protein and K^+ in the lymph were reduced in acholia. Disturbance of the lymph flow rate in acholia was accompanied by marked hemodynamic disturbances: depression of the contractile function of the myocardium (CFM) and hypotension [7], a decrease in cardiac output (CO) and CVP [8]. In animals with acholia, CVP was almost 3 times lower than in the control.

The state of CFM was studied after 20 days of acholia (Table 2). The heart rate of these animals decreased from the 1st to the 10th occlusion of the aorta. A progressive fall of arterial pressure also was observed in animals with acholia, from initial hypotension. The maximal pressure (P_{\max}), as an important pa-

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TABLE 1. Velocity of Lymph Flow and Electrolyte and Protein Composition of Lymph and CVP in Acholia ($M \pm m$)

Experimental conditions	Velocity of lymph flow, ml/kg/min	K ⁺	Na ⁺	Total protein, g/liter	CVP, mm water
		μmoles/liter			
Control	0,057±0,09	3,6±0,2	141±7	71,4±67	46±6,2
Acholia	0,027±0,003*	2,6±0,2***	138±4	52,5±5,5*	17±5,2**

Legend. *P < 0.05, **P < 0.02, ***P < 0.01 compared with control.

TABLE 2. Parameters of CFM in Acholia ($M \pm m$)

Experimental conditions	Parameter	Occlusion of aorta				
		1st	3rd	5th	8th	10th
Control	Heart rate, beats/min	199 \pm 4,9	187 \pm 2,1	188 \pm 3,5	179 \pm 19,8	184 \pm 13,7
	P _{max} , mm Hg	245 \pm 10,9	248 \pm 8,6	238 \pm 7,7	245 \pm 8,3	240 \pm 10
	P _{max} /T, mm Hg	3,37 \pm 0,2	2,6 \pm 0,4	2,6 \pm 0,2	2,6 \pm 0,3	2,4 \pm 0,1
Acholia	Heart rate, beats/min	132 \pm 12,7*	135 \pm 12,2**	132 \pm 9,7*	125 \pm 32,1**	113 \pm 37,4*
	P _{max} , mm Hg	198 \pm 8,5**	194 \pm 11,7**	184 \pm 9,3*	186 \pm 10,2*	172 \pm 10,4*
	P _{max} /T, mm Hg	2,28 \pm 0,3***	1,9 \pm 0,4**	1,7 \pm 0,08***	1,7 \pm 0,5**	1,5 \pm 0,3***

Legend. *P < 0.001, **P < 0.01, ***P < 0.05 compared with control.

TABLE 3. Values of SI and CI in Acholia ($M \pm m$)

Experimental conditions	CI, liters/min/m ²	SI, ml/m ²
Control	0,802 \pm 0,089	4,13 \pm 0,55
Acholia	0,69 \pm 0,02*	5,09 \pm 0,5*

Legend. *P < 0.05 compared with control.

parameter of myocardial contractility, also was reduced in these animals. The mean rate of development of maximal pressure (P_{max}/T) was 33% lower than in the control after only the first occlusion. Considerable changes also were observed in the systolic and cardiac indices (SI and CI respectively; Table 3). In animals with acholia SI was increased by 23% compared with the control. The heart rate was 33% lower. Since the level of propulsive activity of the heart can be maintained both by the stroke volume and by the heart rate, CI of these animals was lowered (Table 3).

When the possible mechanisms of disturbances of the lymphatic circulation in acholia are discussed, the first point to note is weakening of CFM and hypotension, with the result that the effective filtration pressure is reduced at the microcirculatory level and the process of lymph formation is impaired. The fall of venous pressure also leads to an increase in resorption of tissue fluid back into the blood stream, with consequent inhibition of lymph formation. Most lymph flowing from TD is known to be produced through intestinal function: It is directly proportional to intestinal peristalsis. Under conditions of acholia, a parietic state of the intestine develops as a rule, sometimes with complete inhibition of its peristalsis. Finally, lymph formation and transport are active processes and depend on the resorption capacity of the lymphatic capillaries and contractile activity of the lymphatic vessels [3, 11].

An investigation of the microcirculatory system of the mesentery in cats with acholia revealed marked inhibition of contractility of the lymphatic vessels. The frequency of their contractions was more than 3 times lower than in the control (4-5 and 15-16 respectively). The lymphatics were in a semiparetic state and were abruptly deformed. The autonomic and hormonal dyscoordination accompanying acholia and also the electrolyte imbalance, including in the lymph, are evidently the main causes of disturbance of resorption of lymph by the intestine and of the contractility of the lymphatic vessels.

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EFFECT OF INDUCED LIPID PEROXIDATION ON DARK ADAPTATION OF PHOTORECEPTORS OF THE ISOLATED FROG RETINA

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Very little is yet known about the mechanisms of visual adaptation of vertebrate photoreceptors. An important role in this process is ascribed to intracellular mediators [6, 8]. Three principal parameters characterizing adaptation in photoreceptors are distinguished: initial concentration of intracellular mediator, release of mediator into the intracellular space and the length of its life, and changes in conductance of Na leakage channels in the plasma membrane of the rods taking place during adaptation to light and darkness (membrane, or M, adaptation [8]). Consequently, besides mechanisms somehow changing the concentration of intracellular mediator, a definite role also is played by the plasma membrane and, in particular, by changes in conductance of its leakage channels. Meanwhile an unusually high concentration of polyunsaturated fatty acids, capable of being oxidized under the influence of light [3], has been observed in the vertebrate photoreceptor membrane, and lipid peroxidation (LPO) products can increase the permeability of both model [5] and biological membranes [4].

The aim of this investigation was to study the effect of LPO on dark adaptation of photoreceptors of the isolated frog retina.

EXPERIMENTAL METHOD

Experiments were carried out on frogs (*Rana ridibunda*) adapted to darkness for 16-18 h. The retina was isolated from the pigmented epithelium in weak red light in a solution of the following composition (in mM):

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