compared with the control. A decrease in the absolute number of CFU also was observed during repeated tests on seven patients during progression of the disease.

It can be concluded from the results of this quantitative study of stromal precursors of bone marrow mechanocytes in different periods of idiopathic triple hypoplasia of hematopoiesis and acute leukemia in children that stromal precursors of mechanocytes do not remain intact in diseases involving the hematopoietic stem cells in the pathological process. Fibroblast precursors of the stroma are most severely damaged in severe hypoplasias of hematopoiesis, whether idiopathic or drug-induced (the beginning of remission of acute leukemia). The results showing the inhibitory action of chemotherapy of acute leukemia on fibroblast precursors of stromal mechanocytes must be regarded as important. Drug-induced inhibition of precursors of stromal mechanocytes differs from that observed in idiopathic hypoplasia of hematopoiesis in the much greater severity of the process despite an equal degree of reduction in the bone marrow cell population.

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DYNAMICS OF THE LYMPH CIRCULATION AND PROTEIN–CELL REACTION OF THE CENTRAL LYMPH DURING THE EARLY POSTRESUSCITATION PERIOD

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KEY WORDS: lymphatic circulation; postresuscitation period.

Experimental and clinical studies in the last decades have demonstrated the principles and led to the recognition of a new nosological entity – postresuscitation sickness [6]. However, many aspects of its pathogenesis still remain unexplained. In particular, our knowledge of the pathophysiology of the lymphatic system in the course of the postresuscitation period is quite inadequate. Yet the lymphatic system, an inseparable part of the cardiovascular system and a connecting link for all the body fluids, plays an important role in the maintenance of homeostasis and it largely determines the intensity of metabolic processes in the microcirculatory system as well as the specific and nonspecific resistance of the body to injury and stress [1, 2].

The object of the present investigation was to study the state of the lymphatic circulation and the biochemical and cytological composition of the lymph in the course of the early postresuscitation period.

EXPERIMENTAL METHOD

Experiments were carried out on 27 dogs of both sexes weighing 10-23 kg. Exteriorization of the thoracic duct at the point where it enters the left venous angle, and of the femoral

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TABLE 1. Change in Velocity of Lymph Flow during Clinical Death and Early Postresuscitation Period (M ± m)

Experimental conditions	Velocity of lymph flow, ml/min/kg body weight (× 10)
Normal state	0,193±0,014
At height of bleeding	0,674±0,112*
Clinical death	0,049±0,097 †
Restoration of spontaneous breathing	
30 sec	0,492±0,081*
5 min	0,688±0,079*
30 min	0,366±0,027*
1 h	0,314±0,052‡
2 h	0,245±0,019†

*P < 0.01.
†P < 0.02.
‡P < 0.05.

veins and arteries was carried out under thiopental anesthesia preceded by trimeperidine premedication. Clinical death was induced in the experimental animals by free bleeding from the femoral artery. The animals were revived 3 min after the beginning of clinical death by Negovskii's method [5] without the use of stimulants. To prevent the blood from clotting heparin (500 AU/kg) was injected intravenously. The arterial blood pressure and respiration of the animals were recorded throughout the experiment. The velocity of the lymph flow was judged from the quantity of lymph escaping through a polyethylene cannula, inserted into the thoracic duct, and expressed in ml/min/kg body weight. The leukocyte count and total protein concentration were determined before bleeding and 5 and 30 min and 1 and 2 h after the beginning of restoration of spontaneous breathing. In films of lymph stained by the Romanovsky–Giemsa method 1000 lymphoid cells were counted and the relative percentages of individual types of leukocytes were calculated. Protein was determined with the IRF-22 refractometer. On the basis of the results thus obtained the transport function of the lymphatic system was determined; its criterion was the quantity of protein transported by the lymph in 1 min, allowing for changes in the velocity of the lymph flow. Analysis of the cellular reaction of the thoracic duct lymph was based on the same principle.

EXPERIMENTAL RESULTS

The results of investigations of the lymphatic circulation are given in Table 1. They show that during the dynamics of the terminal states changes in the lymphatic flow were phasic in character: an initial increase at the height of bleeding and a decrease in the subsequent stages of clinical death. In the course of restoration of the vital functions of the experimental animals the volume of lymph escaping through the thoracic duct was always increased: 5 min after the appearance of spontaneous respiratory movements there was a more than threefold increase in the flow of lymph compared with the initial level. At all times of the investigation the absolute quantity of protein transported by the lymphatic system was significantly increased (Table 2).

In the postresuscitation period the number of cells of the lymphoid series per unit of lymph fell progressively, but because of an increase in the velocity of the lymph flow in the first 30 min of the postresuscitation period the absolute number of cells transported by the lymphatic system per unit time was significantly greater than normally. At other times of the investigation, despite a significant increase in the velocity of the lymph flow, transport of cells by the lymphatic system was reduced. As Table 2 shows, in the postresuscitation period the qualitative shifts were accompanied by quantitative changes in the cell composition of the lymph: an increase in the number of undifferentiated cells of the lymphoid series and a decrease in the relative percentages of small and medium-sized lymphocytes.

These investigations demonstrated the important role of the lymphatic system in compensation of the disturbed indices of homeostasis and preservation of optimal conditions for metabolism in the interstitial spaces, as well as mobilization and redistribution of cells

TABLE 2. Protein and Cell Composition of Lymph and Transport Function of Lymphatic System during Early Postresuscitation Period (M ± m)

Index	Restoration of spontaneous breathing				
	normal state	5 min	30 min	1 h	2h
Total protein, g/liter	42,3±1,5	36,8±1,6‡	34,6±1,7†	37,6±1,7†	37,9±1,6†
Absolute quantity of protein transported by lymph/min/kg body weight	0,816	2,531	1,266	1,180	0,928
Number of cells in 1 μl lymph	12 685±1 086	8 221±1 686**	8 239±876†	5 100±1 237†	3 955±1 276**
Absolute number of cells transported by lymph/min/kg body weight*	244 820	565 605	301 547	160 140	96 898
Cell composition of lymph, %					
small and medium-sized lymphocytes	91,65±0,88	89,44±0,9‡	86,637±3,60	83,08±1,79†	83,36±0,5†
large lymphocytes	5,50±0,32	7,37±0,41†	7,90±0,59†	9,07±0,80†	10,63±1,64†
prolymphocytes	2,20±0,23	1,27±0,50**	4,19±2,81	4,58±1,64	4,65±1,42
blast cells	0,47±0,11	0,21±0,14	0,94±0,67	0,96±0,31	0,68±0,29
eosinophils	0,19±0,23	1,70±0,27†	0,59±0,38	0,46±0,26	0,68±0,46

*Mean protein content or mean number of cells given, allowing for changes in velocity of lymph flow.
†P < 0.01.
‡P < 0.02.
**P < 0.05.

of the lymphoid series in the body during the early hours of the postresuscitation period. A concrete expression of the participation of the lymphatic system in restoration of the circulating plasma volume and of the protein content in the blood stream is mobilization of extravascular proteins and fluid by the lymphatic system, which was reflected in the present experiments by an increase in the velocity of the lymph flow and in the transport of protein by the lymph. The cause of the increase in the lymph flow was probably an increase in the venular-capillary pressure under the influence of endogenous cathecholamines, and also a disturbance of vascular permeability. There is evidence in the literature of an increase in the concentration of circulating endogenous catecholamines and an increase in vascular permeability in the early hours of the postresuscitation period [3, 7]. An increase in pressure in the capillaries and venules is known to lead to transudation of fluid into the interstitial space and to interfere with its absorption into the venous system, which ultimately leads to an increase in lymph formation [10, 11]. Meanwhile the experiments showed that catecholamines activate the contractile function of the lymphatics and accelerate the lymph flow in the thoracic lymphatic duct [4, 9].

The dynamics of the cells in the present experiments agreed with views on the role of the lymphoid tissue as a reserve of ready-made nuclear material and of energy resources, as well as of lymphoid cells capable of activating myelopoiesis in response to injury and stress [1, 12]. The transient fall in the relative percentage of blast cells and prolymphocytes in the first 5 min of the postresuscitation period can be explained by the particular features of the lymph circulation within the lymph node [8]. In the early stages of the postresuscitation period a "direct" type of lymph circulation within the lymph node probably predominates, when the afferent lymph, bypassing the interstitial sinuses of the lymph node via peripheral sinuses, passes directly into the efferent lymphatics. This type of lymph cicu-lation, aimed at the rapid mobilization of extravascular fluid, prevents the passage of afferent lymph through the cortical substance of the lymph node, rich in young lymphoid cells, and so leads to a decrease in the relative proportion of these cells in the efferent lymph. Later the lymphatic circulation probably switches to the more favorable "indirect" types and ensures the supply of undifferentiated cells of the lymphoid series of prolymphocyte and blast type into the lymph. In the later periods of the investigation, the considerable (almost threefold) decrease in the number of cells of the lymphoid series in the lymph can probably be attributed to functional exhaustion of the lymphoid tissue.

The results of these investigations thus point to the important role of the lymphatic system in the mobilization and redistribution of extravascular proteins and fluid in the body and also of cells of the lymphoid series in the early postresuscitation period.

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EFFECT OF PARENTERAL NITROGEN FEEDING ON BLOOD AND LIVER AMINO ACID SPECTRUM IN THYROXINE POISONING

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KEY WORDS: thyroxine poisoning; parenteral nitrogen feeding.

It has been shown in experimental thyrotoxicosis, characterized by marked changes in tissue metabolism, that parenteral feeding lowers the amino nitrogen level in the blood, tissues, and urine, which is raised in this pathology, and restores the normal content of nucleic acids in the tissues, indicating stimulation of anabolic processes [2, 3].

In continuing our investigations of experimental thyrotoxicosis, the aim was to study the effect of parenteral nitrogen feeding on the amino acid spectrum of the blood and liver in this pathology.

EXPERIMENTAL METHOD

Experiments were carried out on 50 albino rats weighing 220-250 g. Thyroxine poisoning was induced in rats kept on the ordinary animal house diet, by injection of thyroxine in a dose of 10 μg/100 g body weight daily for 30 days [4]. After administration of thyroxine for 30 days, all the animals were kept for 3 days on a protein-free diet, consisting of starch, sugar, sunflower oil, yeast, mixed salt, and vitamins. Against the background of this diet, for 7 days the animals of group 1 received subcutaneous injections of physiological saline, those of group 2 received the amino acid mixture polyamine, and those of group 3 improved casein hydrolysate in a dose of 0.3 g conventional protein/100 g body weight. The results of the experiments were compared with data for healthy rats kept on the ordinary animal house diet (normal) and also with data for animals with thyrotoxicosis receiving physiological saline only (protein deprivation). Animals not poisoned with thyroxine, and receiving injections of physiological saline during protein deprivation, formed a separate group.

EXPERIMENTAL RESULTS

Short-term (for 10 days) protein deprivation in rats without thyroxine poisoning led to

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