FUNCTIONAL RELATIONSHIPS BETWEEN BLOOD VESSELS AND SINUSES OF THE LYMPH NODES UNDER NORMAL CONDITIONS AND IN EXPERIMENTAL DISTURBANCES OF THE BLOOD AND LYMPH CIRCULATION

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The question of possible exchanges between lymph and blood in the lymph nodes deserves attention, since it has been shown that particles of India ink injected into a node via the artery are found in lymphoid tissue and lymphatic sinuses [12]. Later, particles of thorotrast were noted to move from blood vessels of the lymph node into the surrounding lymphoid tissue [3]. It was thought that in its path through the lymph node the lymph loses a part of the water, the latter then being reabsorbed into the circulatory bed [4]. This point of view has been maintained by several investigators [2, 8, 11] who offered only indirect proofs that such a process occurs. Among such proofs are the following: the appearance in the blood of dyes injected into the lymphatics, until the dyes were found in the thoracic duct; the smaller diameter of the efferent vessels from the lymph node in comparison with the total diameter of the afferent vessels; the angioarchitecture of the lymph node reminiscent of that of the kidney; widespread blood vessels and everfilled sinuses of the lymph nodes under conditions of venous stasis. Recently, American investigators have attempted to obtain direct experimental proof of an essential, direct relationship between the lymph node sinuses and the veins [17, 9, 10]. To this end, gas bubbles have been injected into lymph nodes and then have been found in the veins draining the nodes. The conclusions drawn on the basis of such traumatic methods, appear unconvincing.

The aims of the present study were: 1) to obtain quantitative data on the transfer of fluids from the lymphatic bed to the circulatory bed (if such occurs) in the lymph node; 2) to elucidate the direction of the exchange between blood vessels and lymph node sinuses upon impeding the blood vessels and lymph node sinuses upon impeding the blood outflow; 3) determine whether it is possible for the fluid in the afferent vessels to move to the regional lymph node during ligation of the efferent vessels.

EXPERIMENTAL

The experiments were performed on 24 adult dogs. The popliteal lymph node served as the object of study. Tyrode solution at a pressure of 100 mm H_2O was injected into the afferent vessel of the popliteal lymph node. Into this, in a system of flexible plastic tubes, was inserted a graduated glass capillary with internal diameter of 1.5 mm. Inside the capillary the air bubbles were moved at a rate and direction which depended on the linear rate of passage of fluid through the afferent vessel to the lymph node. In the efferent vessel a second needle was placed against the lymph flow; this needle was connected by plastic tubing with a second glass graduated capillary of the same diameter as the first. From the rate of movement of the air bubbles into the second capillary one can deduce linear rate of fluid passage from the lymph node via the efferent vessel. By knowing the value of the linear flow rate of the fluid and the mean diameter of the capillaries, one can determine the volume of fluid passing into the lymph node and leaving it per unit time (one minute). The details of this system have been described previously [1].

TABLE 1. Comparison of Fluid Volumes Entering and Leaving the Popliteal Node (in ml/min)*

No. of expt	Fluid volume entering node	Fluid volume leaving node
1	45	5,85 (0,13 from A)
2	60	36 (0,60 „ „)
3	7,95	4,5 (0,56 „ „)
7	16,5	13,5 (0,82 „ „)
9	8,27	14,25 (1,72 „ „)
10	8,75	5,25 (0,60 „ „)
11	8,49	5,32 (0,62 „ „)
12	60	36 (0,60 „ „)
13	65,2	63,7 (0,98 „ „)
14	19,2	10,6 (0,56 „ „)
16	34,8	7,8 (0,22 „ „)
17	62,7	42,3 (0,69 „ „)
18	48,6	12,9 (0,27 „ „)
19	24,9	13,8 (0,55 „ „)
20[a]	19,7	13,1 (0,66 „ „)
20[b]	24,8	16,7 (0,65 „ „)
21	32,1	21 (0,65 „ „)
22	35,5	19,9 (0,57 „ „)
24	26,3	18,4 (0,70 „ „)
26	23,7	9,75 (0,41 „ „)
27	14,7	9,15 (0,62 „ „)
28	27	11,88 (0,44 „ „)

*Pressure in afferent vessel 100 mm H_2O.

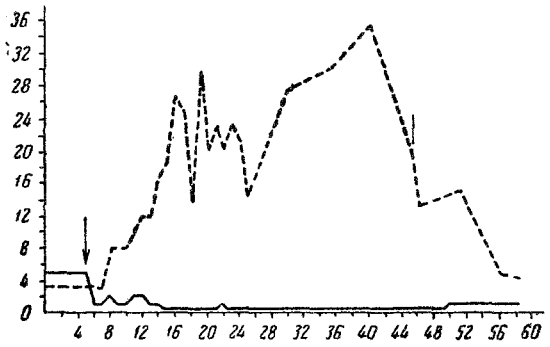

Fig. 1. Perfusion of the popliteal lymph node of the dog with Tyrode solution. Abscissa: time (in min), ordinate: rate of flow of perfusion fluid (in mm/min). Solid line: rate of entry of fluid into the node; dotted line: rate of outflow of fluid from the node. Arrow: ligation of femoral vein.

RESULTS

The volume of fluid passing into the lymph node per minute under a pressure of 100 mm H_2O, as a rule, exceeds the volume of fluid leaving the node in the same time. This decrease in amount of fluid during its flow through the lymph node in individual instances was extremely significant (see Table 1).

The phenomenon noted may be explained only by a transfer of part of the fluid out of the lymphatic sinuses into the blood capillaries. The possibility of fluid leakage from the node via unnoted efferent vessels was excluded by the result of an injection of trypan blue into the hind foot pad: the dye rapidly colored the lymphatic bed of the extremity. In this way all the efferent vessels of the popliteal node became entirely visible. One of these was retained for insertion of the needle and the rest were ligated.

The results obtained are in accord with the data described in the literature [5, 6]. With use of the inulin method of "purification," borrowed from renal physiology, it was successfully shown that within the lymph node there exist the conditions for active exchange between blood and lymph. Experimenting on the superficial cervical nodes of the dog, the author also noted a difference between the volume of fluid passing into the node and that exiting from it. During perfusion of the lymph nodes the author used the system suggested here [5, 6].

The results of the above-described experiments lead us to the opinion that under conditions of venous hyperemia within the lymph node an opposite phenomenon may be observed, i. e., fluid from the blood capillaries may pass into the lymph sinuses. To prove this hypothesis we performed experiments of three types: in the first series of experiments, against a background of lymph node perfusion by Tyrode solution through the afferent lymphatic vessel, a brief constriction of the femoral vein was produced; in the second series, against a background of node perfusion through the lymphatic vessels, the vein was ligated, after which perfusion was continued as long as possible; in the third series of experiments the femoral vein in one extremity was ligated, and after one, three, seven, and nine days a comparative perfusion of the lymph nodes of both legs was performed.

In experiments in series II a rather constant and extremely characteristic picture was observed. The volume of fluid passing from the afferent vessel per unit time was markedly decreased, falling to zero in individual experiments. In two experiments a retrograde flow of fluid was even observed, flowing from the node into the afferent vessels. At the same time the amount of fluid flowing from the node into the efferent vessel either decreased negligibly, did not change at all, or, most often, increased (see Fig. 1). A peculiar situation arose: the volume of fluid

leaving the node exceeded the volume injected into the node via the afferent vessel. We explained this by the fact that, under conditions of venous hyperemia within the lymph node, a part of the fluid from the venous capillaries shifts into the lymphatic sinuses. Under these conditions, naturally, the transport function (carrying capacity) of the lymph node for fluid entering from the afferent vessels is decreased. The prevalence of outflow values over inflow for the node after ligation of the femoral vein continues for a varying period—in different cases from several minutes up to two hours. At a much later time the flow into the node exceeds the outflow. However, the results of comparative perfusion of hyperemic and intact lymph nodes, carried out one, three, seven, and nine days after unilateral ligation of the femoral vein, indicate that the conditions which hinder reabsorption of lymph by the blood vessels within the lymph node exist for a rather long period after the production of venous stasis in the extremity. For example, there are the data on the comparative perfusion of the intact lymph node and a node which remains for one day within a zone of venous hypertonia. If the volume of fluid entering the node via the afferent vessel is taken as 100 in both cases, then the outflow from the lymph node in the intact extremity is 47% while that from the hyperemic extremity is 64%. Thus, the level of lymph reabsorption by a hyperemic lymph node is less than by an intact one.

In considering the close functional relationship between the blood vessels and lymphatic sinuses, it may be hypothesized that the constriction of the large collecting lymphatic vessels would not lead to complete cessation of lymph flow in the peripheral ("prelymphatic") lymph vessels, especially if the lymph pressure rose in the latter. To verify this hypothesis all the efferent vessels of the popliteal lymph nodes in both extremities were ligated. Despite this procedure, the movement of fluid injected into the afferent vessel at a pressure of 100 mm H_2O did not cease. In this case the observations were continued for 20 min after ligation of the efferent vessels.

During this entire time Tyrode solution was infused into the afferent vessel (and subsequently, into the popliteal node also) at a rate of five to six mm/min. In the second case observations were continued for 30 min and a gradual movement of the fluid to the regional lymph node was noted.

The experiments here described show that even under conditions of blockage of the collecting lymphatics drainage of tissue by the lymphatic system is still possible; however the path taken by the peripheral lymph in this case is short—only to one of the regional lymph nodes, where its reabsorption into the blood vascular system is effected. On the whole, data presented in this communication indicate that the regional lymph node is the organ of redistribution of fluid between the venous and lymphatic beds, depending on the anatomic-functional status of the lymphatic and venous pathways of tissue drainage.

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