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The Cardiac Lymphatics in Experimental Chronic Congestive Heart Failure* (33883)

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It has been shown that the right duct lymph system plays an important role in removing fluid from the lung in experimental chronic congestive heart failure (6). While there has been recent interest in the cardiac lymphatics in various pathologic states (3), the role of the cardiac lymphatics in chronic congestive heart failure has not been established, principally because it is difficult to cannulate the small lymphatics and to produce chronic congestive heart failure in the experimental animal. Both of the technical obstacles are surmountable and the present paper reports the effect of experimentally induced chronic congestive failure on canine cardiac lymph flow.

Methods. Six dogs weighing between 15 and 30 kg were anesthesized with sodium pentobarbital (29 mg/kg). Respirations were maintained with an intermittent positive pressure respirator. A left lateral thoracotomy was performed to provide adequate exposure for visualization of the heart and mediastinum. The pericardium was opened and 1 ml of T 1824 dye (Evans blue 0.5% aqueous solution) was injected into the subepicardial layers of the pulmonary conus. After a few minutes the dye filled the lymphatics in the region of the cardiac lymph node lying between the superior vena cava and innominate artery at the base of the heart. Using a Zeiss dissecting microscope a cardiac lymphatic was cannulated with a polyethylene tube (size 10, 20, or 50) and the lymph samples were collected in 15-min aliquots for 3 hr.

Chronic congestive heart failure was produced in a second group of six dogs weighing between 17 and 21 kg by the creation of an aorticocaval anastomosis below the renal arteries and the administration of 25 mg of deoxycorticosterone trimethylacetate twice a week and a 6 g salt diet (6). The animals were edematous and in marked congestive failure after 19–23 days. They were anesthesized with sodium pentobarbital (23 mg/kg) and placed on a positive pressure respirator. A left lateral thoracotomy was performed and the cardiac lymphatics were visualized and cannulated. Lymph was collected as described above in the control group. In both groups femoral artery blood was drawn after 2 hr of lymph flow. The RBC, WBC, SGOT, LDH, total protein, sodium, potassium, and chloride concentrations were determined. Similar studies were done on pooled lymph flow samples after periods of 1, 2, and 3 hr.

Results. The effluent cardiac lymphatics

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TABLE I. Lymph Flows (ml/hr).

Control (6)[c]	2.28 ± 0.43[a]	(1.20–3.80)[b]
Congestive failure (6)	3.16 ± 0.35	(2.20–4.46)

[a] SE.
[b] Range.
[c] No. of dogs.

and the draining lymph nodes of the animals in chronic congestive failure were grossly larger than the corresponding lymphatics and nodes in the controls. In addition, the blue dye was not as readily apparent in the experimental dogs. It was not unusual to find the intermittent appearance of blood in the generally clear lymph of both groups. The average lymph flow was 2.3 ml/hr in the control group and 3.2 ml/hr in the experimental group. These differences were not statistically significant (Table I). Blood samples of the experimental and control groups appeared to be of the same composition (Table II). The second and third hour SGOT and first hour LDH levels were significantly higher in the control series. The total protein content, however, was lower in the lymph of the experimental animals. The composition of the lymph, in other respects, appeared to be similar in both experimental and control groups (Table III).

TABLE II. Composition of Blood in Control and Experimental Dogs.

Test	Mean ± SE (range)	
	Control	Congestive failure
RBC (× 10^6)	5.587 ± 0.339 (4.650–6.960)	4.518 ± 0.095 (4.290–4.840)
WBC	11,500 ± 3081 (1100–21,000)	15,500 ± 1989 (7600–21,000)
SGOT	45 ± 10 (10–80)	52 ± 16 (17–130)
LDH	70 ± 17.8 (27–135)	288 ± 120 (42–870)
Total protein	5.08 ± 0.28 (3.8–5.8)	4.80 ± 0.59 (3.7–7.6)
Na	148 ± 1.7 (141–152)	149 ± 1.4 (144–153.4)
K	3.0 ± 0.1 (2.8–3.2)	3.1 ± 0.24 (2.1–3.8)
Cl	119 ± 1.6 (114–124)	115 ± 2.8 (104–120)

Discussion. It has been demonstrated that the mammalian heart has an extensive lym-

TABLE III. Composition of Cardiac Lymph.

Test[a]	Mean ± SE (range)	
	Control	Congestive failure
RBC count (× 10^6)		
(a)	0.286 ± 0.125 (0.075–0.650)	0.250 ± 0.132 (0.020–0.900)
(b)	0.231 ± 0.091 (0.016–0.510)	0.154 ± 0.98 (0.016–0.640)
(c)	0.288 ± 0.112 (0.020–0.759)	0.198 ± 0.148 (0.014–0.930)
WBC		
(a)	3160 ± 782 (1400–5400)	4760 ± 4000 (200–25,000)
(b)	3180 ± 673 (800–4900)	1950 ± 1330 (100–8500)
(c)	2900 ± 337 (1800–3700)	6700 ± 6177 (320–35,000)
SGOT		
(a)	1283 ± 521 (368–3130)	373 ± 123 (68–900)
(b)	715[b] ± 190 (428–1470)	235 ± 51 (60–420)
(c)	552[c] ± 79 (410–850)	223 ± 68 (94–546)
LDH		
(a)	775[b] ± 200 (230–1445)	325 ± 78 (27–500)
(b)	506 ± 110 (314–940)	264 ± 53 (140–440)
(c)	377 ± 74 (134–590)	287 ± 74 (110–520)
Total protein	3.8[b] ± 0.17 (3.3–4.3)	2.9 ± 0.38 (1.5–4.4)
Na	151 ± 2.6 (144–160)	154 ± 2.8 (147–166)
K	2.8 ± 0.13 (2.5–3.1)	3.1 ± 0.29 (2.1–4.0)
Cl	126 ± 1.7 (122–133)	115 ± 6.4 (93–130)

[a] Hour specimen taken: (a) first; (b) second; and (c) third.
[b] $p < .05$.
[c] $p < .01$.

phatic system with well defined subendocardial, myocardial, and subepicardial lymphatics (4). The lymph passes from the subepicardial plexus into draining valved trunks. The latter usually unite into one or two trunks which drain the entire heart and leave it along the anterior surface of the pulmonary artery. Here it enters a lymph node designated the cardiac lymph node, lying between the superior vena cava and innominate artery. At times, however, the cardiac lymphatics may enter a pretrachial node on the ascending limb of the arch of the aorta prior to entering the cardiac lymph node (2). The lymphatics ultimately ascend to the right lymphatic duct and occasionally to the thoracic duct.

The mean lymph flow recorded in our control animals was higher than that noted by Drinker *et al.* (1) (0.8 ml/hr), and slightly less than the mean flow recorded by Miller (2) (3.2 ml/hr). Rusznyak *et al.* (5) question that Drinker's group collected all the cardiac lymph and suspected that the true lymph flow was twice as great as indicated.

The mean cardiac lymph flow in chronic congestive failure was slightly higher, but not significantly different statistically from the control dogs although there was an apparent gross increase in the size of the lymphatics in the failure group. This is unlike the pulmonary lymph flow, which markedly increases in chronic congestive failure (6). It is likely that the "spongy" environment of the lung offers minimal tissue resistance, whereas the more rigid environment of the heart offers a relatively higher impedance to lymph accumulation. The cardiac lymph flows observed in chronic congestive failure suggest that there was no significant change in cardiac capillary permeability, hydrostatic, oncotic, or tissue pressure relationships.

The clinical inference from the data would suggest that chronic congestive failure while slightly increasing lymphatic size does not *per se* lead to a significant increase in cardiac lymph flow. It would be of interest to learn what occurs with congestive heart failure associated with effluent lymph obstruction (*e.g.,* fibrosis, myocarditis, or myocardiapathies), or increased cardiac venous pressure (*e.g.,* severe right atrial hypertension).

Summary. The cardiac lymphatics were cannulated in 12 dogs. Chronic congestive failure was previously induced by creation of an aorticocaval fistula and administration of deoxycorticosterone trimethylacetate and salt in six dogs. The cardiac lymph flows averaged 3.16 ml/hr and 2.28 ml/hr in the failure and control animals, respectively, and the effluent cardiac lymphatics were larger in the experimental animals. The composition of the lymph (RBC, WBC, Na, K, Cl) was similar, except for slightly increased SGOT, LDH, and total protein concentration in the control group. The composition of blood (RBC, WBC, SGOT, LDH, total protein, Na, K, Cl) was not significantly different in the control and experimental groups. The data suggest that in chronic congestive failure the cardiac lymphatics expand slightly, but cardiac lymph flow is not significantly increased. This is in contrast to the pulmonary lymphatic system in chronic congestive failure in which great expansion and increase in lymph flow occurs.

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