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Test of the Safety Valve Function of the Renal Lymphatic Vessels[1] (38094)

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The renal lymphatic system has been suggested to function as a safety valve mechanism protecting the kidneys from excessively high intrarenal pressures during diuresis or urinary tract obstruction (1). This hypothesis seems likely since renal damage has been reported to follow lymphatic obstruction (2). Further, it has been shown that changes in renal lymph pressure follow those of other intrarenal pressures when the latter are increased by a variety of means (3, 4). Interruption of free lymph drainage in such experiments is of minimal importance since only one of ten or more lymph vessels (5) is utilized for pressure measurement. In the present experiments, the safety valve hypothesis of renal lymphatic function is tested by monitoring intrarenal pressures before and during complete renal lymphatic obstruction.

Methods. Mongrel dogs (20–30 kg) were anesthetized with intravenous sodium pentobarbital (30 mg/kg), and the left kidney was exposed through a flank incision. The left ureter was exposed and catheterized with polyethylene tubing for collecting urine, and the femoral vein was catheterized to facilitate administration of additional anesthetic as needed. Lymphatic obstruction was accomplished in experimental dogs by ligating all observable hilar lymphatic vessels and ligating the fatty tissue at both poles of the kidney. In dogs used for control data care was taken to prevent interruption of normal lymph drainage. Capsular and hilar renal lymph pressure and renal subcapsular pressure (estimate of total tissue pressure) were measured as previously described (4). Intrarenal venous pressure was obtained via a catheter inserted through the renal vein wall and advanced into the venous system of the kidney. Criteria for proper catheter tip placement were those of Hinshaw (6). All pressures were continuously monitored using appropriate strain gages and a multichannel polygraph. After obtaining the initial observations 30 min after lymphatic obstruction, intrarenal venous pressure was elevated in increments of 10 mmHg by increasing tension on a suture passed around the renal vein. Additional observations were made 120 min after lymphatic ligation in a separate series of experiments. All animals undergoing 2 hr lymphatic ligation received sustaining infusion of 9% NaCl at 2 ml/min.

Results. As shown in Table I, renal lymphatic obstruction caused a significant increase in capsular lymph pressure (L_CP) after 30 min, but not after 120 min. Although increased, hilar lymph pressure (L_HP) was not significantly different ($p > 0.05$) from control 30 min following lymphatic obstruction. Intrarenal venous pressure (IRVP) and subcapsular pressure (SCP) were unchanged at both 30 and 120 min.

Table II shows increases in SCP, L_CP, and L_HP which accompany 10 mmHg increments of IRVP. Relative changes are shown for control (patent lymphatics) and

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TABLE I. Capsular and Hilar Renal Lymph Pressure, Subcapsular Pressure and Intrarenal Venous Pressure. Control Observations Are Compared with Those Obtained 30 and 120 Min Following Renal Lymphatic Obstruction ($n = 5$).

		Control	30 min	120 min
Capsular lymph	$\bar{x}$	9.6	13.6*	11.2
Pressure (mmHg)	SE	1.4	0.9	3.1
Hilar lymph	$\bar{x}$	10.2	17.8	—
Pressure (mmHg)	SE	2.3	2.6	—
Subcapsular	$\bar{x}$	21.2	21.6	15.9
Pressure (mmHg)	SE	3.1	4.2	1.3
IRVP	$\bar{x}$	27.8	20.4	26.0
(mmHg)	SE	7.2	2.7	3.7

* $p < 0.05$.

experimental (renal lymphatic occluded) animals. Absolute values for these parameters before IRVP alteration are those shown in the first two columns of Table I. Each increment in IRVP was accompanied by a rapid increase in each of the other parameters. In addition, no statistical difference could be detected between responses of control animals and those of animals having renal lymphatic obstruction ($p > 0.05$).

Discussion. Failure of acute renal lymphatic obstruction to cause significant changes in renal SCP and IRVP argues against an important safety valve function for these vessels. It was noted that SCP, IRVP, and LP are relatively stable for periods up to 2 hr following lymphatic obstruction. If renal lymphatic obstruction were to result in increased intrarenal pressures due to loss of the lymphatic "safety valve," then the pressure changes should be evident within the time period studied. In previous work (7) it was found that IRVP increases produced by partial obstruction of renal venous outflow caused rapid and profound increases in both SCP and renal lymph pressure. These responses were unchanged following renal lymphatic obstruction in the present study, again suggesting that renal lymphatic vessels are not primarily involved with regulation of intrarenal pressures. These findings are of particular interest since it might be argued that the renal lymphatic vessels function only during periods of accelerated lymph production. Previous investigators have found changes suggestive of renal edema following lymphatic obstruction (8, 9). The available data suggest that the results of such experiments may vary according to the animal species used and the time of effective lymphatic obstruction. The rabbit kidney (8)

TABLE II. Changes in Subcapsular Pressure (ΔSCP), Capsular ($\Delta L_C P$) and Hilar ($\Delta L_H P$) Renal Lymph Pressure which Accompany 10 mmHg Increments in Intrarenal Venous Pressure (ΔIRVP). Means and Standard Errors are Shown for Control Animals (C) with Patent Renal Lymphatic Vessels and Experimental Animals (E) with Occluded Renal Lymphatics.

	ΔSCP (mmHg)		$\Delta L_C P$ (mmHg)		$\Delta L_H P$ (mmHg)	
ΔIRVP mmHg	C $n = 10$	E $n = 10$	C $n = 5$	E $n = 6$	C $n = 5$	E $n = 5$
10	11.7 ± 1.6	9.4 ± 0.9	10.8 ± 1.8	7.6 ± 1.2	6.8 ± 2.9	8.2 ± 3.9
20	24.6 ± 3.3	18.8 ± 1.5	23.2 ± 2.9	15.6 ± 1.0	13.6 ± 6.0	12.8 ± 5.2
30	34.2 ± 3.8	28.7 ± 2.0	34.3 ± 3.4	26.8 ± 1.9	20.8 ± 8.5	21.4 ± 6.1
40	45.0 ± 4.4	37.4 ± 2.1	47.5 ± 4.3	37.8 ± 2.3	28.2 ± 11.2	29.6 ± 7.7

appears to be more susceptible to lymphedema than that of the dog (9). Further, the histological changes observed following prolonged renal lymph stasis in the dog could result from interstitial accumulation of toxic products of tissue metabolism (2) rather than from pressure related changes.

The moderate increases in capsular and hilar renal lymph pressures observed in dogs with obstructed renal lymphatic vessels (Table I), may not represent abnormally increased pressure in the renal tissue fluid compartment. Since lymph leaves the kidney by ten or more trunks (5), it cannot be expected that the pressure within the lymph capillaries would be reflected in an extrarenal lymph trunk while other routes of egress are patent. Lymph stasis, however, would cause the lymphatic pressure measurements to approach lymphatic capillary and interstitial fluid pressures. As noted in Table I, capsular and hilar lymph pressures obtained during lymph stasis are substantially less than SCP. Further, these lymph pressures are well within the canine renal interstitial pressure range published by Gottschalk (10).

Summary. Renal lymph pressure, intrarenal venous pressure, and subcapsular pressure were measured before and during complete renal lymphatic obstruction. These parameters were unchanged during lymph stasis. Further, the responses of these parameters to elevated renal venous pressure were unchanged following lymphatic obstruction. It is concluded that the renal lymphatic system generally does not function as a pressure "safety valve" but may function to relieve the renal interstitium of possibly toxic metabolic products.

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