Renal Lymph Sodium and Potassium Concentrations Following Renal Vasodilation[1] (37352)

R. D. BELL, W. L. PARRY, AND W. G. GRUNDY

Department of Urology, University of Oklahoma Medical Center, Oklahoma City, Oklahoma 73104

It is generally agreed that renal lymph concentrations of sodium and urea are greater than those of blood plasma (1, 2). Further, these solutes are normally found in high concentration in the interstitial fluid of the renal medulla (3). It has, therefore, been suggested that the renal medulla is an important site of renal lymph formation (2). In contrast, recent studies have shown that medullary lymph formation could be detected only during pronounced overall renal vasodilation (4). The present study was designed to investigate the possibility of altered delivery of medullary sodium into renal lymph during renal vasodilation produced by renal arterial infusion of acetylcholine.

Methods. Greyhound dogs or mongrel dogs (20–30 kg) having the general body conformation of greyhounds were used. Intravenous sodium pentobarbital (30 mg/kg) was used as anesthesia, and the left kidney was exposed through a flank incision. The ureter was catheterized for urine collection, and the femoral artery and vein were catheterized for collecting arterial blood and administering additional anesthetic, respectively. Renal artery infusion was accomplished using a Harvard syringe pump attached by polyethylene tubing to a 20 g hypodermic needle inserted through the wall of the renal artery. Infusions used were 0.9% saline during control periods and acetylcholine chloride (ACh) (0.01 mg/kg/min) during experimental periods. Renal blood flow (RBF) was monitored using an electromagnetic flowmeter (Micron RC 1000). Renal hilar lymphatic vessels were catheterized as previously described (1), and timed serial samples of lymph were collected in 25 μl calibrated capillary tubes. Sodium and potassium concentrations in renal lymph, arterial blood plasma and urine were determined using an IL model 143 flame photometer.

Results. Renal arterial infusion of ACh caused dramatic increases in RBF, renal lymph flow and urine flow (Fig. 1a,b,c). As shown in Table I, renal lymph to arterial plasma concentration ratios (L/P) for sodium and potassium were unchanged by the renal response to ACh. Urine sodium concentrations were also relatively stable throughout the experiment, but urine potassium concentrations decreased consistently during ACh infusion.

Discussion. Previous investigators have suggested that renal lymph composition is determined by the rates of cortical and medullary lymph formation (2). It has also been shown that renal hilar lymph *p*-amino hippurate (PAH) concentration increases during ACh infusion, as would be expected following a significant increase in medullary lymph formation (4). Thus if the L/P ratio for sodium is, in part, dependent upon medullary lymph formation, then this ratio should increase, at least transiently, during ACh infusion. The experiments of the present study show no significant change in the sodium L/P during ACh infusion. Thus it seems that medullary lymph formation is not reflected in the L/P ratio for sodium, and that renal lymph does not participate in the medullary sodium washout observed by other workers (5). If renal lymph sodium concentration is partially derived from the medullary interstitial fluid sodium concentration, the sodium L/P ratio should decrease as medullary

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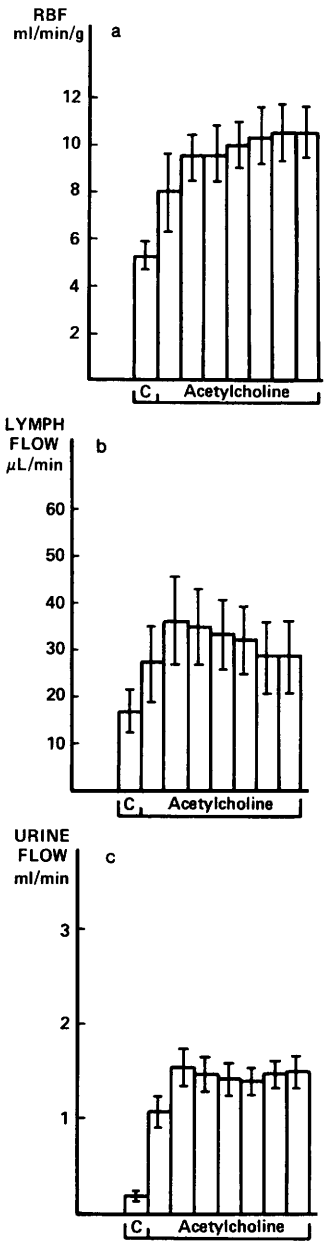

FIG. 1. Renal blood flow (a), hilar renal lymph flow (b) and urine flow (c) during control renal lymph collection (C) and 7 consecutive renal lymph collections of 25 µl each ($n = 5$). Renal arterial infusion of acetylcholine was begun after completing the control lymph collection.

sodium concentration decreases. As shown in Table I, no decrement in sodium L/P ratio can be detected during ACh infusion. Thus it is concluded that L/P ratios are independent of medullary tissue sodium concentration and medullary lymph formation. L/P ratios derived in the present study were, on the average, greater than unity. Nevertheless, when lymph and plasma electrolyte concentrations are compared after correcting for their respective protein concentrations (6) no statistical difference can be detected ($p > 0.05$). It is thus concluded that L/P ratios of the magnitude reported in the present communication do not necessarily imply a medullary component in renal lymph.

The present study supports the previous conclusion that ACh induced changes in lymph flow (4) and pressure (7) appear to be largely determined by cortical fluid dynamic effects of the drug (7). A small proportion of the lymph flow increase may be derived from the renal medulla (4), but the high medullary interstitial fluid sodium concentration is probably lost by diffusion before the lymph reaches the larger branches of the renal lymphatic system (8). Similarity of sodium and potassium L/P ratios also suggests that L/P solute ratios are independent of medullary interstitial fluid concentrations. Data currently available suggest that the mechanisms responsible for concentrations of specific solutes in lymph water different from those of plasma water are largely, if not entirely, within the renal cortex.

Summary. Renal lymph concentrations of sodium and potassium were compared with those of arterial blood plasma before and during ACh induced decrease in medullary sodium concentration. It was found that L/P ratios of both sodium and potassium were unchanged following renal arterial infusion of ACh. These data yielded the conclusion that L/P ratios for sodium are largely due to renal cortical mechanisms and are independent of medullary interstitial fluid sodium concentration.

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TABLE I. Urine Concentrations and Lymph to Plasma Ratios for Sodium and Potassium Before and During Renal Arterial Infusion of Acetylcholine.[a]

	Sodium				Potassium			
	Lymph/plasma conc. ratio		Urine (mEquiv/l)		Lymph/plasma conc. ratio		Urine (mEquiv/l)	
Sample	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Control	1.05	0.01	174.6	44.5	1.06	0.02	118.8	18.8
1	1.06	0.01	193.0	32.8	1.08	0.02	102.8	20.4
2	1.08	0.02	223.8	35.1	1.11	0.04	68.4	18.8
3	1.05	0.02	206.1	24.6	1.10	0.05	39.0	7.0
4	1.05	0.01	186.0	19.2	1.08	0.02	39.2	5.8
5	1.05	0.01	172.4	15.7	1.06	0.05	38.8	5.3
6	1.04	0.01	170.7	14.2	1.05	0.01	38.8	3.9
7	1.05	0.01	169.2	15.2	1.06	0.03	37.6	4.6

[a] Means and standard errors ($n = 5$) are shown for control and 7 consecutive 25 μl experimental collections.

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