Some Factors Affecting Renal Lymphatic Pressure

By Yale J. Katz, M.D.

Renal lymphatic pressure was found to be increased either by partial occlusion of the renal vein or total occlusion of the ureter. Simultaneous measurement of interstitial and lymphatic pressure revealed that with compression of the renal vein the rise in interstitial pressure preceded the rise in lymphatic pressure.

It is curious that little has been written and even less is said regarding the renal lymphatics. This could be because the renal lymphatics indeed play an insignificant role in the total function of the kidney, yet there have been some observations over the past century that suggest the reverse is the case. In 1863, Ludwig and Zawarykin noted that the obstruction of the ureter in the rabbit caused a marked distention of the hilar renal lymphatic vessels. Estimations by Schmidt and Hayman, in 1929, indicated that the renal lymphatic flow may be equal to if not greater than the urinary flow in dogs. Kaiserling and Soostmeyer, in 1939, observed that blockade of the renal lymphatics caused an increase in flow of urine. It would seem then that the renal lymphatics may have an important role in the fluid economy of the kidney.

Goodwin and Kaufman have recently suggested that the lymphatics may function as a sort of safety valve for the kidney, called upon to function when the kidney is faced with a pressure increase due to a ureteral obstruction. A similar view was expressed by Babics in 1951.

The chance observation that the capsular lymphatic vessels of 2 dogs with pyelonephritis were markedly enlarged presented an opportunity to observe the effect of ureteral and venous obstruction on lymphatic pressure, putting to a test the safety valve concept of the function of the renal lymphatics.

METHODS

Two male dogs with moderate renal functional impairment, later found to have pyelonephritis, underwent abdominal exploratory surgery using pentobarbital anesthesia. A dilated renal capsular lymphatic vessel was catheterised using a fine polyethylene catheter with the end of the catheter directed toward the lateral border of the kidney. The catheter was constructed from a 2 inch length of polyethylene tubing\* 50 gage, (0.58 X 0.965 mm., inside X outside diameter) cemented to a 4 foot length of 90 gage tubing (0.86 X 1.27 mm., inside X outside diameter). Pressure was measured with a strain gage, Statham model no. P23BB, and recorded by the Offner model M direct writing oscillograph. The system was filled with 0.85 per cent sodium chloride and heparin, 20 mg./100 ml.

Renal interstitial pressure was measured directly using a low volume displacement capacitance manometer, polyethylene tubing 90 gage and a 24 gage hypodermic needle 9 mm. long with closed end and side hole perforations. Four holes were drilled to 0.22 mm. diameter and staggered up the shaft of the needle. The system was filled with saline containing heparin and the pressure measurement was made by inserting the needle into the kidney after a baseline determination by holding the needle at the point of entry.

RESULTS

The prominent capsular lymphatic vessels of dog 1-L are seen in figure 1. A lymphatic vessel in preparation for cannulation is shown in figure 2. Figure 3 is a tracing showing the effect of obstruction of the renal vein on the pressure in the lymphatic vessel. Approximately 5 sec. after compression of the


†Sanborn electromanometer model 131 C.

‡Prepared by Mr. Bruce Kennedy and Frank Bradley, Kennedy Machine Shop, Pasadena, Calif.
renal vein the lymphatic pressure rose, starting at 7.5 mm. Hg and reaching a maximum of approximately 50 mm. in 20 sec. Seven seconds after release of the renal vein the pressure began to decline, falling to 10 mm. Hg in 2.5 min. The upper level of pressure is considered only approximate because above 40 mm. Hg the response is not linear at the selected sensitivity of the manometer.

Figure 4 shows the effect of occlusion of the ureter on lymphatic pressure. Here the rise in lymphatic pressure was a gradual one and paralleled the development of acute hydronephrosis. As the kidney enlarged and became more tense, the pressure rose slowly reaching the maximum in 31 min. At this time the kidney was extremely tense and quite suddenly the lymphatic pressure fell, doubtless due to closure of the vessels at the point of exit from the kidney because of the tenseness of the kidney.

Figure 5A and B show simultaneous recordings of interstitial and lymphatic pressures. In figure 5A, the catheter in the lymphatic vessel became kinked at the
FIG. 5. Effect of compression of renal vein on renal lymphatic and interstitial pressure. The first arrow indicates time of compression of the renal vein and the second arrow indicates time of release of the compression. A. The lymphatic catheter was inadvertently kinked prior to release of the renal vein.

point of maximum pressure causing a precipitous fall in the pressure. In figure 5B, the interstitial pressure does not show either the extent of pen deflection or the sustained elevation seen in 5A. This was due possibly to some leak at the point of insertion of the needle into the kidney. Nevertheless, both tracings demonstrate that the rise in interstitial pressure preceded the rise in lymphatic pressure when the renal vein was occluded.

In dog 2-L, the lymphatic pressure averaged 7 mm. Hg. With partial occlusion of the renal vein, the pressure rose to an average of 32 mm. Hg. After release of the clamp on the renal vein, the lymphatic pressure fell to an average of 20 mm. Hg. At this point, when the renal artery was occluded, the lymphatic pressure fell to 8 mm. Hg. With the renal artery occluded, compression of the renal vein failed to produce a rise in lymphatic pressure. When the renal artery clamp was released with the renal vein still occluded, the lymphatic pressure promptly rose to 53 mm. Hg.

Occlusion of the renal vein appeared to cause an increase in the rate of flow of the lymph, but exact measurements were not made in these experiments.

DISCUSSION

The elevation of lymphatic pressure in response to ureteral or venous obstruction in the dog suggests that the lymphatic system provides the means of pressure release for the kidney. Should such a situation prevail in the human subject, blockade of the lymphatics, coupled with overload or blockade of the ureter, would cause an elevation of pressure within the kidney. Goodwin and Kaufman suggest that the pathogenesis of several renal disorders involve blockage of this "safety valve" of the kidney. They suggest that the severe renal damage that occurs in hydronephrosis when infection supervenes and activation of nephritis and nephrosis all may be a consequence in part of blockade of the lymphatics by an unrecognized lymphangitis. These authors report a case of spontaneous rupture of the kidney in a woman with metastatic carcinoma involving the ureter and the abdominal lymphatics, and suggest that the catastrophe was a result of an increased pressure in the kidney deprived of its safety valve.

The observation that the rise in interstitial pressure occasioned by occlusion of the renal vein preceded the rise in lymphatic pressure, is consistent with the notion that the lymphatic fluid originates in part at least from the interstitial fluid, a notion first expressed by Ludwig nearly 100 years ago.

Since it was noted that renal lymphatic flow increased with partial compression of the renal vein, the possibility that this event takes place in instances of elevated renal vein pressure such as heart failure is suggested. Studies are in progress to determine whether in conditions simulating heart failure the renal lymphatics are involved in the deviation
to the systemic circulation fluid and sodium that would appear in the urine under condition of normal renal vein pressure.

SUMMARY

Two dogs with pyelonephritis were observed to have markedly enlarged renal capsular lymphatics. These were catheterized. Obstruction of the renal vein caused a rise in lymphatic pressure from 7.5 mm. to approximately 50 mm. Hg. Simultaneous measurement of interstitial and lymphatic pressure revealed that with compression of the renal vein the rise in interstitial pressure preceded the rise in lymphatic pressure. These studies suggest that one of the functions of the renal lymphatic system is to act as a safety valve for the kidney when exposed to elevations in the ureteral and venous pressure.

SUMMARIO IN INTERLINGUA

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Circ Res. 1958;6:452-455
doi: 10.1161/01.RES.6.4.452

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/6/4/452

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