TABLE I. Right Duct Lymph Flow and Protein Concentration in Anesthetized and Unanesthetized States (1st and 2nd day P.O.).

	Right duct flow (ml/15 min)			Right duct protein (g/100 ml)		
	Anesthetized	Unanesthetized		Anesthetized	Unanesthetized	
Dog No.	At operation	1st day P.O.	2nd day P.O.	At operation	1st day P.O.	2nd day P.O.
779	1.4	1.0	—	—	2.70	—
780	1.7	1.9	.6	2.13	2.66	2.64
790	2.5	2.2	2.3	3.48	2.45	3.16
792	1.1	2.8	2.0	2.70	2.44	3.00
809	1.1	3.1	1.7	3.60	2.63	2.20
Avg	1.6	2.2	1.7	2.98	2.58	2.75

Unfortunately, multiple lymph collections could not be made on successive days, hence many important studies on continuous lymph flows in chronic states could not be made. One principal problem was that in the unanesthetized animal, movement of the right shoulder caused the large plastic tube to erode from the external jugular vein. To overcome this problem, and obtain flows in the unanesthetized dog, the external jugular vein was altered so as to form a collecting channel for right lymphatic outflow.

Drinker found that the average lymph flow was 1.1 ml per hour. However, this was a flow from only one of several lymphatics(1). Courtice found right duct flow to be 2.3 ml per hour(8). Using the "chamber" technic, the mean right duct flow was 4.2 ml per hr (5). In this study, the mean right duct flow was 6.4 ml per hour on day of operation, 8.8 ml per hour on the following day, and 6.8 ml per hour on the third day. Protein concentration of right duct lymph was not significantly changed in the unanesthetized state.

Summary. A method of right duct lymph collection from a jugular venous collecting channel which may be used in the unanesthetized animal has been described. There was little evidence of a significant change in right duct lymph flow or right duct protein concentration in anesthetized and unanesthetized animals.

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Development of Reversible Hematuria and Oliguria Following Elevation of Renal Venous Pressure. (28140)

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Proteinuria has been reported following exercise and during radical changes in body posture(1). Winton(2) also noted its presence in some instances in the isolated perfused kidney following limited elevation of renal venous pressure. Several explanations of this

phenomenon have been suggested(1), but its cause remains obscure. A series of experiments was therefore carried out on the isolated and intact dog kidney to investigate the acute effects of renal venous pressure elevation. The experimental preparations utilized in this study are of particular value in that varied conditions of pressure are carefully controlled.

Methods. Isolated kidneys. Male and female dogs were intravenously anesthetized with sodium pentobarbital, 30 mg/kg. Isolated dog kidneys were perfused with heparinized whole blood from an isolated heart-lung system(3). The kidney was transferred to the perfusion circuit without interruption of blood flow(4). The system was primed with whole blood from a donor dog, a kidney was taken from another, and a third dog supplied the heart and lungs. A constant renal artery pressure was maintained with a Starling shunt device (range 100-150 mm Hg). Urine samples were collected from the catheterized ureter and urine flow rates were measured with a graduated cylinder and stopwatch. Renal venous pressure was elevated by progressively tightening a screwclamp on a length of large bore polyethylene tubing secured in the renal vein. All pressures were recorded using Statham pressure transducers connected to a Sanborn direct-writing recorder.

Intact kidneys. The left kidney was exposed through a flank incision. The renal vein was either (a) cannulated and venous blood continuously returned to the jugular or femoral vein by a pump or (b) isolated, but otherwise undisturbed. In the first instance, venous pressure was elevated with a screwclamp on the tubing and pressure monitored through the tubing proximal to the clamp. In the latter case, a No. 27 gauge needle was secured in the vein for pressure measurements and venous pressure was elevated by applying tension on the vein with a ligature placed distal to the needle. The ureter was catheterized with a small bore polyethylene tube and urine flow rates were measured as described above. Mean systemic arterial pressure was monitored from a catheterized femoral artery and registered on a Sanborn direct-writing recorder.

TABLE I. Effect of Increased Renal Venous Pressure on the Kidney.*

Exp	Hematuria	% RBC in urine	Urine flow (% of control)	Renal LVP* (mm Hg)
	Heart-lung-kidney preparation			
1	+	1.5	10	63
2	+	.9	69	61
3	+	.1	87	25
4	+	1.0	17	58
5	+	1.8	86	55
	Intact dog kidney preparation			
1	+	.5	7	68
2	+	.5	42	43
3	+	.6	44	62
4	+	1.0	43	38
5	+	.1	39	60
6	+	.2	37	63

* Minimal increase in large vein pressure required to elicit hematuria.

Urine samples were collected in centrifuge tubes during a 5 or 10 minute period and 1 cc samples of each collection period were centrifuged in Wintrobe tubes for urine hematocrit determination.

The renal venous pressure elevations were sustained for 5 or 10 minutes at each pressure level.

Results. The experimental data are summarized in Table I. Results show that hematuria was present in all experiments following venous pressure elevation. The volume of red blood cells present in the urine is small and variable (0.1-1.8%). Urine flow rate decreased in all experiments to a variable degree (7 to 87% of control values). The large vein pressure necessary to produce hematuria appeared to be similar in both isolated (mean 53 mm Hg) and intact (mean 53 mm Hg) kidneys. Hematuria disappeared following return to control pressure. Fig. 1 shows the decrease in urine flow rate for a wide range of renal venous pressures expressed on a percentage basis. Hematuria was not observed in either group until the urine flow rate was significantly reduced (mean of both groups: 44% of control).

Discussion. Results show that gross hematuria, including red blood cells and hemoglobin, appears when a relatively high venous pressure is produced. If the pressure is further elevated, the degree of hematuria increases and is sustained until control venous pressure is restored, when it disappears. He-

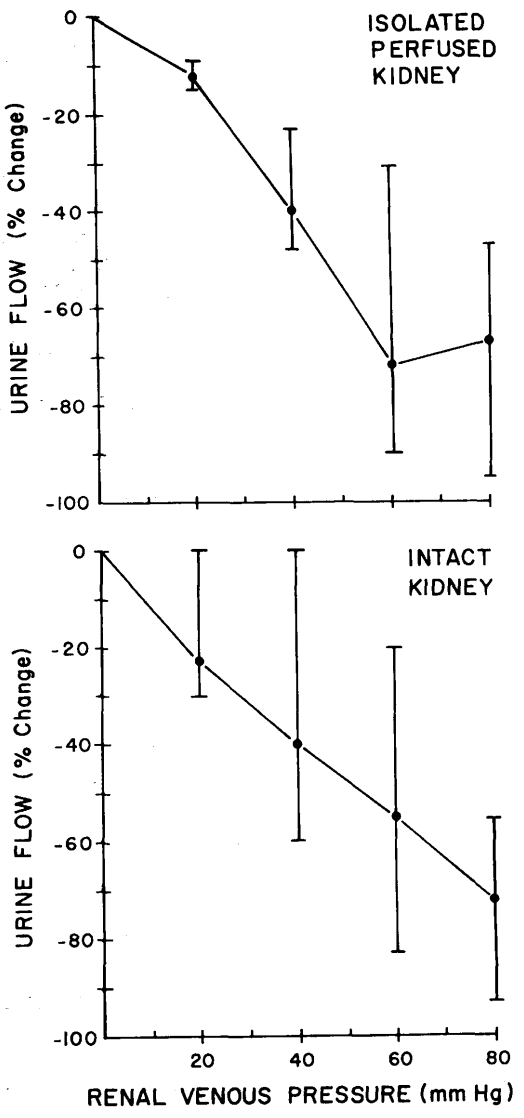

FIG. 1. Effect of increasing renal venous pressure on urine flow rate. Vertical lines indicate total range at various pressures.

moglobinuria, present in the isolated kidney, was not present in the intact organ under conditions of increased renal venous pressure. Although absent at low venous pressures in the isolated organ, the subsequent appearance of hemoglobinuria at elevated pressures could be accounted for by increased levels of plasma hemoglobin (due to the perfusion pumps) or decreased tonicity of urine (due to absence of ADH).

There are at least 3 possible sites of entrance for red blood cells into the urine. One possible region is the peritubular capillaries. Swann described these as being "leaky" and suggested that a free communication exists between these vessels and the interstitial space (5). Evidence for the existence of large pores in the peritubular capillaries is given by electron microscopy studies(6). However, a mechanism for getting the red cells into the urine from the interstitial space has not been proposed. Another possible entrance site for red blood cells is through the glomerular capillaries by virtue of an increased glomerular pressure, although it seems unlikely that venous pressure transmissions would be effective in increasing transmural pressure at the glomerular membrane(7). A factor opposing the glomeruli as the site of entrance is that renal venous pressure elevations are transmitted rapidly to the proximal tubules(7,8), which would decrease the glomerular transmural pressure. The third and most plausible site is "pores" in the walls of blood vessels bordering the renal pelvis which could be temporarily opened, allowing for passage of red cells into the urine. The reverse phenomenon, pyelovenous backflow, has been produced by maintaining moderate intrapelvic pressure for a sustained period, or for a shorter period at higher pressures(9,10). A direct, temporary communication can be established between the kidney pelvis and the large superficial pelvic veins by elevated intrapelvic pressure(9). The possibility of "veno-pelvic" flow merits further investigation.

As reported by Winton(2), oliguria was obtained following renal venous pressure elevation. At least two factors may contribute to the development of the oliguria: partial ureteral obstruction, allowing more time for reabsorption of water from the tubules(11) and increased back presure with decreased glomerular filtration rate(2,12-14).

Summary. The acute effects of elevated renal venous pressure in development of reversible gross hematuria and oliguria were studied. Both isolated and intact dog kidney preparations were utilized. Results demonstrate that gross hematuria was produced as renal venous pressure was elevated, but not until a relatively high pressure was produced.

Degree of hematuria increased as the pressure was further elevated, but disappeared rapidly following restoration to control pressure. The effect of renal venous pressure elevation on urine flow rate was one of progressive oliguria after a critical pressure was reached, and eventually anuria at the higher pressures. This effect was also reversible.

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Nucleic Acid Content of Mammary Glands of Rats Lactating 41 and 61 Days.* (28141)

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Lactation has been extended beyond normal length in mice(1) and rats(2,3,4) by replacing original litters with foster litters. In rats, either all litters gained at about the same rate(2,3) or litter weight gains declined one-third after the first 20 days and thereafter remained constant(4). Histological study of the mammary gland indicated that the lobule-alveolar system had not involuted (4).

The present investigation was undertaken to determine quantitatively the effects of extended lactation, by litter replacement, on mammary deoxyribonucleic acid (DNA) and ribonucleic acid (RNA) content and RNA/DNA ratio.

Methods. Lactation was extended in 11 rats to 41 days and in 12 rats to 61 days by providing eight 1- to 4-day-old foster pups every 21 days. Litter weight gain from the 7th to the 15th day after parturition or after litter replacement provided information on lactational performance. Body weights of mothers were recorded on 3rd day of lactation and at end of the extended lactation. Nucleic acid content was determined, as previously described(5), on 6 abdominal-inguinal mammary glands at the end of the extended lactation period.

Results. Total mg of DNA per 100 g of initial body weight (day 3 of lactation) were 10.56 and 10.94, and total mg of DNA were 23.25 and 24.33 for rats lactating 41 and 61 days, respectively. These estimates of mammary development did not differ significantly ($P>0.05$) from that of rats lactating 21 days (6). DNA per 100 g of final body weight was 9.58 and 9.48 for rats lactating 41 and 61 days, respectively. These values were significantly less ($P<0.05$) than those of rats lactating 21 days, but they merely reflect the increase in body weight which occurred during the extended periods of lactation.

Mammary glands of rats lactating 41 and 61 days contained 49.76 and 44.83 mg of

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