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Relation of Nephron Recruitment to Detectable Filtration and Recovery of Function after Release of Ureteral Obstruction (41683)

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Abstract. In this study, the role of nephron recruitment to measurable filtration in the recovery of whole kidney GFR following release of ureteral obstruction of 18 hr duration was characterized in the young rat with a modification of Hanssen's technique. Following release of bilateral (BUO) or unilateral (UUO) ureteral obstruction, the expected increases in fractional sodium and water excretion were observed and remained constant throughout the interval of the study (approximately 3 hr). However, the GFR of the postrelease kidney of rats subjected to BUO and UUO increased significantly within this time frame. Surprisingly, the percent increase in GFR was greater in the the UUO group. In both groups, there was a positive correlation between the number of filtering nephrons and time after release. The increase in the number of filtering juxtamedullary and superficial nephrons detectable with this technique was symmetrical after release of UUO and BUO. However, at all intervals of study, there were significantly fewer filtering nephrons in the postrelease kidney of the UUO and the BUO group. These studies indicate that the mechanism of recovery of GFR in the postrelease kidney in the two settings of ureteral obstruction is quite different.

The concept that the response of individual nephrons to ureteral obstruction might be heterogeneous was originally proposed by Yarger *et al.* (1). In these studies, the single nephron glomerular filtration rate (SNGFR) of surface nephrons was measured with the technique of renal tubule micropuncture after release of bilateral ureteral obstruction (BUO) of 24 hr. They found that SNGFR was reduced proportionately less than whole kidney glomerular filtration rate (GFR), and suggested that this disparity was due to better preservation of the GFR of surface nephrons than of juxtamedullary (JM) nephrons. Jaenike (2) reported that following release of BUO, the rate of tubule flow in surface nephrons was highly variable. In these studies, 28% of surface nephrons were found to be either nonfiltering or had filtration rates that were too low to assess with the technique of renal tubule micropuncture. Based on these observations, Jaenike suggested that the different effects of obstruction on whole kidney and single nephron GFR were due to heterogeneity in the filtering capacity of all nephrons rather than to a selective loss of JM nephron function. This hypothesis received further support from several subsequent studies (3-5). Harris and Yarger (3) determined the number of filtering nephrons in the postrelease kidney and found that there was a significant reduction in both nephron groups although JM nephrons were more profoundly affected. Slightly different results were obtained by Wilson (4), who measured the distribution of SNGFR between superficial and JM nephrons using a modification of Hanssen's technique (6). He found that the ratio of superficial to JM nephron GFR was not different from controls. Subsequent to these results, we measured SNGFR of surface and JM nephrons by renal tubule micropuncture and found that filtration fell proportionately in both kinds of nephrons (5). To date, however, it remains unclear how nephron heterogeneity in these two types of nephrons is affected by time after release nor have such changes been correlated with those of whole kidney GFR.

The effects of unilateral ureteral obstruction (UUO) on nephron heterogeneity are less well defined. Jaenike (7) was the first to note the striking variability in the appearance of the tubules on the surface of the postrelease kidney. In these studies, he found that most of the tubule lumina appeared narrowed and many were actually collapsed. This morphologic appearance was associated with a marked decrease in tubule fluid flow rate that was due

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to enhanced fluid reabsorption and a heterogeneous decrease in SNGFR (3, 7–9). Thus, heterogeneity of nephron function was also apparent in this setting. Whether surface and JM nephrons are symmetrically affected remains unclear at this point. Using direct techniques, we found that the SNGFR of JM nephrons was more preserved than that of superficial nephrons (9). On the other hand, we have reported that the percentage of JM nephrons with detectable filtration 90 min after release was profoundly depressed although not out of proportion to that of the superficial nephrons (9). Harris and Yarger have reported similar findings (3). No studies have attempted to correlate these changes with time after release or even contrast the total GFR or SNGFR of the kidney after release of BUO and UUO.

The present study was designed to measure the percentage of nephrons with detectable filtration after release of BUO and UUO, and determine how recovery of GFR is related to the number of nephrons apparently recruited to filtration. Differences in the response of the UUO and BUO kidney will also be delineated as well as how these differences relate to time after ureteral release.

Methods. Approximately 18 hr before study, 46 male Sprague–Dawley rats weighing 45–60 g were anesthetized lightly with ether and both ureters were exposed through a midline abdominal incision. In one group (sham), the ureters were visualized but not touched. In another group (UUO), either the left or right ureter was ligated with 4-O silk at a point approximately one-third of the distance from the bladder to the renal pelvis. In a third group (BUO), both ureters were ligated. All rats were fed standard rat chow until the time of the initial surgical procedure. Thereafter, they were deprived of food but not water.

On the day of the study, the rats were anesthetized with inactin (Promonta, Hamburg, FRG) given intraperitoneally (60–100 mg/kg body wt). Tracheostomy and intubation were then performed and polyethylene catheters were placed in one or both jugular veins and the right femoral artery. Both kidneys were exposed through a midline abdominal incision and dissected free of the peritoneal attachments. A ligature (4-O silk) was then placed around each renal pedicle. A catheter was placed in the bladder for collection of urine from the right kidney in the control group and the contralateral untouched kidney in the group that had undergone UUO. The obstruction was relieved by incising the ureter above the ligature. In the UUO group the ureter of the obstructed kidney was then cannulated with a polyethylene catheter (PE No. 50) so that timed urine collections could be obtained. In the BUO group, both ureters were cannulated. Immediately after relief of obstruction an inulin prime was given followed by an infusion of normal saline which contained inulin in sufficient amounts to maintain plasma levels at 50–100 mg/100 ml. In both control and UUO rats, the mean infusion rate was calculated at 65 μl/min/100 g body wt. A slightly greater amount of saline was given to the group subjected to BUO in order to replace urinary fluid losses (85 μl/min/100 g body wt). After an equilibration period of 60 min, one to three timed urine collections were obtained. Arterial samples and pressure measurements were obtained at the midpoint of each urine collection. Body temperature was monitored with a rectal probe and maintained between 36 and 38°C.

A modification of Hanssen's technique (3, 6, 9) was used to determine the percentage of filtering nephrons immediately before release of obstruction and 90 and 180 min following release of BUO and UUO. At the designated time (0, 90, or 180 min postrelease), a 10% sodium ferrocyanide solution was infused intravenously at the rate of 3.0 ml/min. Approximately 30 sec after the infusion was begun, the left and right renal pedicles were ligated. The kidneys were quickly removed and frozen in a dry ice and acetone mixture. The frozen kidneys were fragmented and maintained at −20°C for 18 hr in a solution of 95 ml ethanol, 5 ml concentrated HCl, and 50 g ferric chloride. The fragments were then incubated at 39°C for 80 min in 20% HCl and then at room temperature in a solution of 1% acetic acid and 100% ferric chloride solution for 24 hr. Superficial and juxtamedullary nephrons were then microdissected from at least five fragments of each kidney. These fragments were of sufficient size to allow random dissection of at least 10 superficial and 10 juxtamedullary nephrons. Nonfiltering nephrons were defined as those units in which

TABLE I. BODY WEIGHTS, BLOOD PRESSURE, AND PLASMA PARAMETERS IN THE THREE GROUPS OF RATS

	Body weights (g)	Blood pressure (mm Hg)	Hematocrit (%)	BUN (mg/100 ml)	Na (mEq/liter)	K (mEq/liter)
SHAM (N = 8)	65.5 ± 1.7	99.4 ± 3.4	36.3 ± 1.0	17.9 ± 1.4	152 ± 2	4.4 ± 0.2
UUO (N = 20)	63.1 ± 2.3	101.0 ± 2.0	36.7 ± 1.0	20.8 ± 2.0	145 ± 2	4.6 ± 0.1
BUO (N = 18)	59.6 ± 2.4	91.1 ± 2.8†	32.2 ± 1.0†	71.9 ± 5.7*	144 ± 1	7.7 ± 0.4*

Note. Values are mean ± SE. †Denotes that values are significantly different from SHAM and UUO rats at a level of $P < 0.01$, *<0.001.

there were no detectable Prussian blue granules in the tubule lumen.[1]

The concentration of inulin in plasma and urine was determined by an anthrone method (10). Plasma and urinary sodium and potassium concentrations were estimated with standard flame photometry (Instrumentation Laboratories, Inc., Lexington, Mass.). The osmolality was measured with a vapor pressure osmometer (Wescor, Inc., Logan, Utah). Blood urea nitrogen (BUN) was determined by an enzymatic method (11).

Mean differences in whole kidney function and the percentage of superficial and juxtamedullary nephrons that were filtering were tested for significance using Student's t test for unpaired data when comparing two groups of animals and for paired data when comparing measurements obtained in the same animals (12).

Results. Table I shows the mean values for body weight, blood pressure, and plasma parameters measured in the three groups of animals. After release of BUO, the BUN and plasma potassium levels were significantly greater than the mean values obtained in the sham-operated and UUO group. Further, rats subjected to BUO had a lower hematocrit.

In Table II, mean values for whole kidney functions are presented for rats that were studied for at least 3 hr following release of either UUO or BUO. Included in this table are results obtained from sham-operated rats studied for 3 hr after the institution of the maintenance intravenous infusion. As predicted, following release of BUO there was a marked increase in absolute and fractional water excretion that was associated with a similar increase in sodium excretion and a decline in urine osmolality. Similar results were observed following release of UUO, although the magnitude of the changes were not as striking. Thus, while absolute water and sodium excretion did not change significantly following release of UUO, fractional excretion rose nearly fivefold. As in the BUO group, there was a significant decline in the osmolality of urine obtained from postobstructed kidneys when compared either to urine from the contralateral control kidney (CCK) or to that obtained from the sham-operated group. Further, these results indicate that absolute and fractional water and sodium excretion and urine osmolality for the three groups of animals did not change significantly during the 3 hr of the study.

[1] Although it is conceivable that filtration is occurring at an undetectable rate in these nephrons, it must be quite low. Using micropuncture techniques, we were able to measure SNGFR in nephrons of the postrelease kidney which were below 1 nl/min (3 nl/min/g kidney wt) or less than 10% of control values (refs. 5, 9). If one assumes that the proximal tubule is a cylinder with a radius of 10 μm then the distance of the Prussian blue dye front from the glomerulus in a nephron with an SNGFR of 1 nl/min could be estimated using the following equation,

$$\text{Dye front} = \frac{\text{SNGFR}/30\ \text{s}}{\pi(10\ \mu\text{m})^2} = \frac{0.0005\ \text{mm}^2}{0.000314\ \text{mm}^3} = 1.6\ \text{mm}$$

Using the present technique Prussian blue granules are easily detectable as they enter the tubule lumen and therefore considerably less than 1 mm from the glomerulus. Thus, this population of nephrons includes an unknown percentage of nephrons which can be defined as "apparently" nonfiltering. In this group the rate of filtration is probably considerably less than 10% of normal.

TABLE II. WHOLE KIDNEY FUNCTIONS IN THE THREE GROUPS OF RATS

	Time after release[a] (min)		V (μl/min)		FE_{H_2O} (%)		FE_{Na} (%)		U_{Osm} mOsm/Kg H_2O	
	1st	2nd	1st	2nd	1st	2nd	1st	2nd	1st	2nd
SHAM (N = 5)	97.4 ± 7.9	165 ± 8	3.11 ± 1.39	2.77 ± 0.61	0.59 ± 0.32	0.66 ± 0.26	1.08 ± 0.88	1.20 ± 0.61	1642 ± 122	1647 ± 194
UUO (N = 14)										
CCK	93.6 ± 3.2	168 ± 8	2.28 ± 0.34	2.62 ± 0.34	0.49 ± 0.06	0.68 ± 0.08	0.23 ± 0.01	0.52 ± 0.16*	1947 ± 101	1945 ± 114
POK			2.83 ± 0.73	3.07 ± 0.68	4.14 ± 0.08	2.69 ± 0.53	3.42 ± 0.81	2.26 ± 0.53	367 ± 11	384 ± 14
BUO (N = 9)	75.3 ± 4.2	168 ± 8	10.3 ± 2.5	9.50 ± 1.69	18.0 ± 2.9	16.2 ± 2.9	12.8 ± 2.9	10.9 ± 2.7	360 ± 8	369 ± 9
SHAM vs POK			NS	NS	<0.025	<0.050	NS	NS	<0.001	<0.001
SHAM vs CCK			NS	NS	NS	NS	NS	NS	NS	NS
POK vs CCK			NS	NS	<0.001	<0.005	<0.005	<0.005	<0.001	<0.001
BUO vs POK			<0.005	<0.001	<0.001	<0.001	<0.005	<0.001	NS	NS

[a] Refers to time (mean ± SE) in minutes between release of obstruction and the midpoint of the first and final clearance period. In shams, values are the time between the institution of the sustaining solution and the midpoint of the clearance period. CCK, contralateral control kidney; POK, postobstructed kidney of the UUO group; V, urine flow rate; FE_{H_2O}, fractional excretion of water; FE_{Na}, fractional excretion of sodium; U_{Osm}, urine osmolality.

* Significantly different from the 1st period value at $P < 0.05$.

Mean values for GFR obtained in these three groups are plotted as a function of time after release in Fig. 1. As expected, the GFR of the sham-operated group did not change significantly during the interval of the study and averaged 440 ± 43 μl/min in the first period and 426 ± 36 μl/min in the final period. On the other hand, the GFR of the postrelease kidney of both UUO and BUO groups increased significantly ($P < 0.005$) within the time frame of the study. Approximately 90 min following release of obstruction, the mean GFR measured in the UUO and BUO group did not differ significantly (78.7 ± 15 vs 56.5 ± 8.3 μl/min, respectively). GFR values obtained nearly 3 hr after release of BUO averaged 69.1 ± 8.4 μl/min. In the UUO group the mean value averaged 110.6 ± 15.0 μl/min after this time interval. The percentage difference between these values averaged 54.1 ± 12.4% in contrast to only 19.5 ± 5.9% ($P < 0.05$) in those animals subjected to BUO. Thus, whole kidney GFR, at about 3 hr after release of UUO, was significantly greater ($P < 0.05$) than that of the BUO group after this time interval. The GFR of the contralateral control kidney of the UUO group actually fell slightly although significantly from a mean of 485 ± 36 to 415 ± 26 μl/min at 180 min ($P < 0.05$).

In Table III and Fig. 2 values are presented for the number of apparently filtering nephrons following release of UUO and BUO. In Table III, these values are contrasted to measurements obtained from sham-operated rats and from the CCK of the UUO group. In shams, the mean number of superficial nephrons with detectable evidence of filtration at 0 time averaged 96.1 ± 4.0% and did not change significantly as a function of time. Prior to release of BUO, 18.8 ± 1.7% of surface nephrons had blue granules present in the tubule lumen. This mean value did not differ significantly from that obtained for JM nephrons (17.2 ± 3.5%). Ninety minutes after release of obstruction, more than 50% of the nephrons counted were apparently filtering. In the superficial nephron population, the mean was 53.2 ± 3.4%, not different from the mean obtained for JM nephrons (55.9 ± 5.4%). Approximately 3 hr after release, over 70% of superficial and JM nephrons had been recruited. These values were still significantly

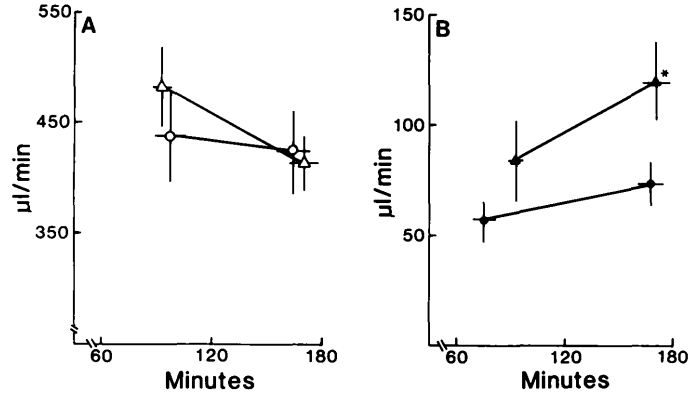

FIG. 1. Whole kidney glomerular filtration rate (Mean ± SE) as a function of time (mean ± SE) after release of bilateral (●) and unilateral ureteral obstruction (▲) is presented in panel B and contrasted to results obtained for the contralateral control kidney of the UUO group (△) and sham-operated rats (○), which are presented in panel A. *Indicates that this value is significantly different from the mean of the BUO group at this time interval at $P < 0.05$.

lower ($P < 0.05$) than the mean number of filtering nephrons for the sham-operated group.

Nephron recruitment to detectable filtration as a function of time after release of UUO was also homogeneous with respect to the two nephron populations (see Fig. 2). However, the percentage of cortical and JM nephrons filtering in the UUO group was always less than that of the BUO group. By 180 min following release of obstruction only 51.7 ± 6.8% of JM and 56.0 ± 5.2% of superficial nephrons were filtering. Thus, after this time interval 16–24% fewer nephrons were filtering in the UUO group than in the BUO group.

Discussion. The effects of UUO of 18 hr duration on renal function presented in Table II are comparable to values that we have reported previously in the weanling rat (5, 9) and to what has been reported in mature rats (1, 3, 8), the dog (13, 14), and humans (15–17). That is, after release of ureteral obstruction there is a marked decrease in GFR, and urine flow and sodium excretion are inappropriately high. In the BUO group, fractional water and sodium excretion was more than 10-fold greater than that of the control group. After release of UUO, the sodium content of the urine and the rate of urine flow were not different from the CCK and thus, in fractional terms, water and sodium excretion by the postrelease kidney were considerably greater than by the CCK. These differences in water and sodium handling by the postobstructed kidney did not change statistically during the interval of the study (see Table II).

Unlike the renal tubule abnormalities, the GFR of the postobstructed kidney improved significantly between 90 and 180 min after release of UUO and BUO (see Fig. 1). The mean difference between initial and final GFR measurements averaged 54%. This difference was greater than that measured in the BUO group, which was approximately 20%. Surprisingly, the effect of UUO on the percentage of filtering nephrons was more profound than that of BUO. Although superficial and JM nephrons were affected in a comparable fashion, there were significantly fewer filtering nephrons at each time interval than after release of BUO. However, as in the BUO group, nephron recruitment to detectable filtration was in evidence during the interval of the study. Thus, the present study provides additional evidence to support the theory, originally put forth by Jaenike (2), that the proportionately smaller fall in surface nephron GFR than in whole kidney GFR, after release of BUO, was due to a heterogeneity in the filtering capacity of both nephron populations rather than to a selective loss of JM nephron function. Further, the results of this work indicate that nephron recruitment to filtration after release of ureteral obstruction is symmetrical and is a function of time after release.

The fact that whole kidney GFR after release of UUO is the same as or better than that of the BUO group suggests that individual

TABLE III. PERCENTAGE OF FILTERING SUPERFICIAL AND JUXTAMEDULLARY NEPHRONS BEFORE AND AFTER RELEASE OF URETERAL OBSTRUCTION

	0 min			90 min			180 min		
	No. filtering	No. counted	Percentage filtering	No. filtering	No. counted	Percentage filtering	No. filtering	No. counted	Percentage filtering
		2 Rats			4 Rats			5 Rats	
SHAM									
Superficial	170	176	96.1 ± 4.0[a]	214	234	92.4 ± 3.1	345	365	94.5 ± 1.9
Juxtamedullary	95	97	97.0 ± 0.7	162	175	94.1 ± 2.2	238	256	93.2 ± 2.9
		5 Rats			5 Rats			5 Rats	
UUO									
CCK									
Superficial	212	233	90.3 ± 2.3	248	279	89.0 ± 2.5	141	170	82.5 ± 2.6
Juxtamedullary	211	229	92.5 ± 2.7	223	253	88.4 ± 2.2	119	142	83.6 ± 1.7
POK									
Superficial	27	252	10.2 ± 2.0	121	294	40.8 ± 3.6	99	175	56.0 ± 5.2
Juxtamedullary	20	239	7.5 ± 1.8	102	306	33.4 ± 3.1	67	132	51.7 ± 6.8
		5 Rats			9 Rats			8 Rats	
BUO									
Superficial	65	358	18.8 ± 1.7	308	574	53.2 ± 3.4	543	745	72.4 ± 4.5
Juxtamedullary	59	316	17.2 ± 3.5	293	537	55.9 ± 5.4	332	400	75.2 ± 6.0

[a] Values are mean ± SE. See Table II for remaining abbreviations.

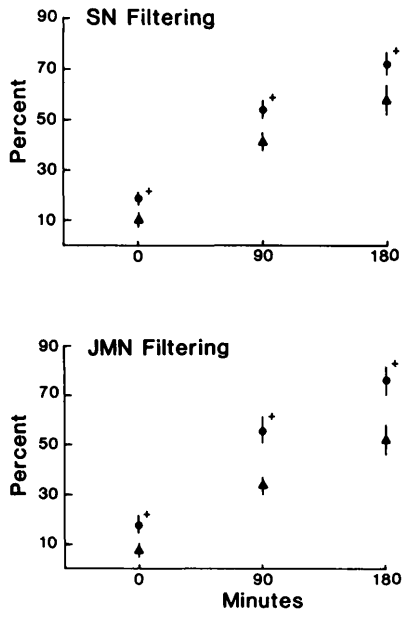

FIG. 2. Relation of the percentage of filtering superficial (SN) and juxtamedullary (JMN) nephrons and time after release of bilateral (●) and unilateral (▲) obstruction. +Values are significantly different from the respective means of the UUO group at $P < 0.01$.

nephron filtration of one or both nephron populations is better preserved in this setting than after release of BUO. Previous micropuncture studies performed in our laboratory would suggest that there is preferential preservation of JM nephron SNGFR in the postrelease kidney of the UUO group (9). In these studies, we found that although the SNGFR of surface and JM nephrons of the postobstructed kidney was lower than values obtained from shams, the SNGFR of JM nephrons decreased by less than half while surface nephron GFR fell nearly threefold. This redistribution in nephron GFR was not observed following release of BUO when SNGFR fell proportionately in both nephron populations (5). Further, SNGFR measurements of superficial nephrons were similar in these two studies (5, 9). This finding is similar to results which we reported in adult rats (8). Thus, the mechanisms for relative preservation of GFR in the two settings of obstruction appear quite different.

Although mechanisms responsible for the differences in nephron recruitment cannot be determined from the present study, it seems likely that the different hemodynamic events that occur in these two settings must be involved. Several direct (18, 19) and indirect (3, 7, 14, 20) studies of glomerular hemodynamics of the obstructed kidney indicate that a marked increase in preglomerular resistance is the predominant factor responsible for the decrease in GFR, before and shortly after release of unilateral ureteral blockade of more than 12 hr duration. These effects seem to be mediated at a local level and have been demonstrated in individual surface nephrons that have been obstructed for 24 hr (21). Further, after release of UUO preglomerular resistance actually increases initially (19). Although the duration of this increased resistance remains undefined, the present studies would suggest that alterations in preglomerular resistance predominate within 3 hr of ureteral release.

The results of hemodynamic studies performed in the presence or shortly after release of BUO are quite different from those reported in the postrelease kidney of the unilaterally obstructed group. In this setting, several factors appear to be responsible for the decrease in GFR. First, hydrostatic pressure within the tubule lumen is higher than in controls (1, 22). Second, pre- and postglomerular resistances fall significantly or change only slightly during obstruction. As a consequence of these two factors net ultrafiltration pressure is reduced (2, 22, 23). These changes are distinctly different from those measured before and immediately after release of UUO and strongly suggest that factors which accumulate in the blood during the anuric phase have a major role in the hemodynamic events which occur at the nephron level in this setting. The profile of nephron recovery in these two settings suggests that the presence of uremia has a more profound effect on recovery of nephron filtration. That is, although more than 70% of nephrons counted were filtering 3 hr after release, whole kidney GFR changed by less than 20%. On the other hand, the GFR of the postrelease kidney of the UUO group at this time interval was greater with 25% fewer nephrons.

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1. Yarger WE, Aynedjian HS, Bank N. A micropuncture study of postobstructive diuresis in the rat. J Clin Invest **51**:625–637, 1972.
2. Jaenike JR. The renal functional defect of postobstructive nephropathy. J Clin Invest **51**: 2999–3006, 1972.
3. Harris RH, Yarger WE. Renal function after release of unilateral ureteral obstruction in rats. Amer J Physiol **227**:806–815, 1974.
4. Wilson DR. Nephron functional heterogeneity in the postobstructive kidney. Kidney Int **7**:19–26, 1975.
5. Buerkert J, Head M, Klahr S. Effects of acute bilateral ureteral obstruction on deep nephron and terminal collecting duct function in the young rat. J Clin Invest **59**:1055–1065, 1977.
6. Hanssen OE. A histochemical method for evaluation of excreted ferrocyanide in isolated tubules of the mouse kidney. Acta Pathol Microbiol Scand **44**:363–371, 1958.
7. Jaenike JR. The renal response to ureteral obstruction: A model for the study of factors which influence glomerular filtration pressure. J Lab Clin Med **76**:373–382, 1970.
8. Buerkert J, Alexander E, Purkerson ML, Klahr S. On the site of decreased fluid reabsorption after release of ureteral obstruction in the rat. J Lab Clin Med **87**:397–410, 1976.
9. Buerkert J, Martin D, Head M, Prasad J, Klahr S. Deep nephron function after release of acute unilateral ureteral obstruction in the young rat. J Clin Invest **62**:1228–1239, 1978.
10. Fuhr J, Kaczamarczyk J, Kruttgen C-D. Eine einfache colorimetrische method zur inulinbestimmung fur nieren-clearance-unterschungen bei stoffwechselgesunden und diabetikern. Klin Wochenschr **33**:729–730, 1955.
11. Fawcett JK, Scott JE. A rapid and precise method for the determination of urea. J Clin Pathol (Lond) **13**:156–159, 1960.
12. Snedecor GW, Cochran WG. Statistical Methods. Ames, Iowa State Univ Press, 6th ed, 1967.
13. Suki WN, Guthrie AG, Martinez-Maldonado M, Eknoyan G. Effects of ureteral pressure elevation on renal hemodynamics and urine concentration. Amer J Physiol **220**:38–43, 1971.
14. Yarger WE, Griffith LD. Intrarenal hemodynamics following chronic unilateral ureteral obstruction in the dog. Amer J Physiol **227**:816–826, 1974.
15. Gillenwater JY, Westervelt FB Jr, Vaughan ED Jr, Howards SS. Renal function after release of chronic unilateral hydronephrosis in man. Kidney Int **7**: 179–186, 1975.
16. Better OS, Tuma S, Kedar S, Chaimowitz C. Enhanced tubular reabsorption of phosphate. Arch Intern Med **135**:245–248, 1975.
17. Better OS, Arieff AI, Massry SG, Kleeman CR, Maxwell MH. Studies on renal function after relief of complete unilateral obstruction of three months' duration in man. Amer J Med **54**:234–240, 1973.
18. Dal Canton A, Stanziale R, Corradi A, Andreucci VE, Migone L. Effects of acute ureteral obstruction on glomerular hemodynamics in rat kidney. Kidney Int **12**:403–411, 1977.
19. Dal Canton A, Corradi A, Stanziale R, Maruccio G, Migone L. Effects of 24-hour unilateral ureteral obstruction on glomerular hemodynamics in rat kidney. Kidney Int **15**:457–462, 1979.
20. Yarger WE, Schocken DD, Harris RH. Obstructive nephropathy in the rat. J Clin Invest **65**:400–412, 1980.
21. Arendshorst WJ, Finn WF, Gottschalk CW. Nephron stop-flow pressure response to obstruction for 24 hours in the rat kidney. J Clin Invest **53**:1497–1500, 1974.
22. Dal Canton A, Corradi A, Stanziale R, Maruccio G, Migone L. Glomerular hemodynamics before and after release of 24-hour bilateral ureteral obstruction. Kidney Int **17**:491–496, 1980.
23. Tanner GA. Effects of kidney tubule obstruction on glomerular function in rats. Amer J Physiol **237**:F379–F385, 1979.

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