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Glomerular hemodynamics before and after release of 24-hour bilateral ureteral obstruction

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Glomerular hemodynamics before and after release of 24-hour bilateral ureteral obstruction. Glomerular hemodynamics were studied, by micropuncture, in Munich-Wistar rats submitted to 24-hour bilateral ureteral ligation (BUL). Glomerular capillary pressure (P_G), intratubular pressure (P_T), and pressure in the first order peritubular capillaries (EAP) were measured with a servo-nulling device. Single nephron filtration fraction (SNFF) was calculated from arterial and peritubular blood protein concentrations. Single nephron glomerular filtration rate (SNGFR) was both measured by conventional micropuncture techniques and calculated from efferent arteriole blood flow and SNFF. Afferent arteriole blood flow (AABF) and resistance of afferent (R_a) and efferent (R_e) arteriole were calculated. Measurements were repeated in the left kidney after releasing the ureter. Sham operated rats were used as control. BUL caused a fall in SNGFR (from 101.8 ± 9.7 to 40.7 ± [SEM] 6.0 nl/min/kg body wt), accounted for by a rise in P_T (from 14.1 ± 0.7 to 28.9 ± 3.1 mm Hg), glomerular hemodynamics (particularly P_G and AABF) being unchanged. A marked increase in R_a (from 6.6 ± 0.7 to 10.8 ± 1.5 dynes · sec · cm^{-5}) occurred after releasing the ureter, lessening both P_G and AABF. Therefore, a low SNGFR was maintained despite the concomitant normalization of P_T.

Hémodynamique glomérulaire avant et après la levée d'une obstruction urétérale bilatérale de 24 heures. L'hémodynamique glomérulaire a été étudiée par microponction chez des rats Munich-Wistar soumis à une ligature urétérale bilatérale de 24 heures (BUL). La pression capillaire glomérulaire (P_G), la pression intratubulaire (P_T) et la pression dans les capillaires péritubulaires de premier ordre ont été mesurées au moyen d'un dispositif à zéro asservi. La fraction de filtration des néphrons (SNFF) a été calculée à partir des concentrations de protéines dans le sang artériel et péritubulaire. Le débit de filtration glomérulaire des néphrons (SNGFR) a été mesuré par les techniques habituelles de microponction et calculé à partir du débit dans l'artériole efférente et de SNFF. Le débit dans l'artériole afférente (AABF) et la résistance des artérioles afférente (R_a) et efférente (R_e) ont été calculés. Les mesures ont été faites à nouveau sur le rein gauche après la levée de l'obstruction urétérale. Des rats ayant subi un simulacre d'intervention ont servi de contrôles. BUL a déterminé une dimunition de SNGFR (de 101,8 ± 9,7 à 40,7 ± [SEM] 6,0 nl/min/kg poids humide) dont rend compte une augmentation de P_T (de 14,1 ± 0,7 à 28,9 ± 3,1 mmHg), les paramètres de l'hémodynamique glomérulaire (P_G et AABF particulièrement) n'étant pas affectés. Une augmentation importante de R_a (de 6,6 ± 0,7 à 10.8 ± 1,5 dynes · sec · cm^{-5}) se produit après la levée de l'obstacle urétéral, diminuant à la fois P_G et AABF. Un SNGFR bas est donc maintenu malgré une normalisation concomitante de P_T.

Recently, we have shown that unilateral ureteral obstruction for 24 hours produces a marked increase in afferent arteriole resistance, which is not reversed by ureteral release. The ensuing cortical ischemia accounts for a fall in glomerular filtration rate, both in the whole kidney (GFR) and in the single nephron (SNGFR) [1].

So far, the effects of 24-hour bilateral ureteral ligation (BUL) have not been adequately investigated. No measurement of glomerular filtration rate or of renal blood flow has been reported during BUL. After releasing one ureter, a decrease in both GFR and SNGFR has been demonstrated [2–5]. Scanty and controversial data are instead available on glomerular plasma flow, which has been found to be either normal [5], or consistently reduced [2].

The present study was designed to evaluate the glomerular hemodynamics and to measure SNGFR in the rat kidney both before and after release of 24-hour bilateral ureteral obstruction.

Methods

Sixteen nonfasted Munich-Wistar rats with glomeruli on the kidney surface [6] were anesthetized lightly with ether. The abdomen was opened via a midline laparotomy, and both the ureters were ligated in nine rats about 1 cm above the bladder, and the other seven rats were sham-operated and used for control. The rats were housed in their cages and left without food and water for 24 hours, when they were anesthetized again with sodium

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pentobarbital (Nembutal®, 60 mg/kg body wt, i.p.) and prepared for micropuncture, as previously described [7]. After the surgical preparation, an i.v. infusion of bicarbonate-saline solution (sodium chloride, 110 mEq/liter; sodium bicarbonate, 28 mEq/liter; potassium chloride, 5 mEq/liter) containing inulin (5%) was begun at an infusion rate of 0.01 ml/min. The infusion rate was increased to 0.02 ml/min after releasing the left ureter in six rats in which postobstructive studies were performed. One hour was allowed for equilibration before starting micropuncture measurements.

Ureteral obstruction studies. In nine rats with 24-hour BUL, glomerular capillary pressure (P_G), intratubular pressure (P_T), and pressure in the first order peritubular capillaries (so-called efferent arteriole pressure, EAP) were measured with a servo-nulling device (Instrumentation for Physiology and Medicine, San Diego, California) and recorded on a dual-channel recorder (Hewlett-Packard, 7702 B) together with blood pressure from a femoral artery (BP). Timed, complete collections of tubular fluid were obtained for measurement of SNGFR [8]. The number of collections (and of SNGFR measurements) was limited because a long time (5 to 10 min) was required to obtain adequate volumes of tubular fluid. Moreover, the oil block injected into the tubule sometimes did not move downstream so that collection was impossible. Therefore, SNGFR was calculated also from the efferent arteriole plasma flow (EAPF) and single nephron filtration fraction (SNFF) according to the equation

$$\text{SNGFR} = \frac{\text{EAPF}}{1 - \text{SNFF}} - \text{EAPF} \qquad (1)$$

This method was previously validated in our laboratory [8] and required timed, complete collections of blood from the welling points of superficial efferent arterioles, for direct measurement of efferent arteriole blood flow (EABF) from which EAPF was calculated.

Postobstructive studies. In six rats, when the measurements of the obstructive period were completed, the left ureter was clamped just below the pelvis and cannulated proximally to the ligation with polyethylene (PE-50) tubing (Clay Adams) connected to a pressure transducer. The clamp was then released, and the ureteral pressure (P_U) was recorded. The obstruction was relieved in the left kidney by cutting the PE-50 catheter used for P_U measurement. One hour was allowed before starting the following measurements: *(a) GFR and sodium excretion rate ($U_{Na}V$) of the left kidney.* Urine was collected under mineral oil; arterial blood samples were obtained from the femoral artery at the beginning and at the end of clearance periods. *(b) Micropuncture measurements.* Timed, complete collections of tubular fluid and of peritubular blood were carried out, and micropressures were recorded as during the obstruction.

Control studies. Measurements as described above were performed also in the left kidney of seven control rats, 24 hours after sham-operation.

Analytical determinations and calculations. Urine volume was obtained by weight. The volumes of fluid and blood collected from proximal tubules and efferent arterioles, respectively, were estimated from the length of the fluid column in a calibrated constant-bore quartz tubing of approximately 100 μ I.D. (Friedrick and Dimmock, Millville, New Jersey). The concentration of chemical inulin in tubular fluid was measured by the microfluorescent method of Vurek and Pegram; chemical inulin concentrations in plasma and urine were determined by the diphenylamine method. Plasma protein concentration was measured by Lowry's method; a microadaptation of the same method was used to measure protein concentration in blood collected from efferent arterioles. Sodium concentration in urine was measured by flame photometry [6].

SNGFR, single nephron filtration fraction (SNFF), effective filtration pressure at the afferent end of the glomerulus (EFP_a), flows and resistances of single afferent and efferent arterioles were calculated as described in our previous papers [1, 8].

Results are presented as the means ± SEM.

Results

Ureteral obstruction studies (Table 1). P_G was similar in control and in obstructed rats. In the latter, P_T was increased from 14.1 ± 0.7 to 28.9 ± 3.1 mm Hg ($P < 0.005$). The rise in P_T was responsible for a reduction in both hydrostatic pressure gradient across glomerular capillaries (ΔP_G) and EFP_a. During BUL, no difference was found in afferent arteriole resistance (R_a) nor in afferent arteriole blood flow (AABF). SNGFR fell from 101.8 ± 9.7 to 40.7 ± 6.0 nl/min/kg body wt ($P < 0.001$). The latter value was calculated as the mean of averaged measurements of SNGFR in single rats, with conventional technique and with efferent arteriole blood collection. As shown in Table 2, in fact, similar results were obtained with the two methods. Afferent arteriole plasma flow (AAPF) being unchanged, the decrease in SNGFR accounted for a reduction in SNFF, which was paralleled by a fall in both hema-

Table 1. Pressures, flows, protein concentrations, hematocrits, and resistances in control conditions and after 24 hours of bilateral ureteral obstruction[a]

	Control	Ureteral obstruction	*P*
Body wt, *g*	181.4 ± 11.6	208.3 ± 9.5	NS
BP, *mm Hg*	106.4 ± 3.9	112.2 ± 3.2	NS
P_G, *mm Hg*	45.8 ± 1.0 (*N* = 26)	49.4 ± 3.4 (*N* = 31)	NS
P_T, *mm Hg*	14.1 ± 0.7 (*N* = 39)	28.9 ± 3.1 (*N* = 48)	<0.005
EAP, *mm Hg*	16.8 ± 1.0 (*N* = 29)	18.8 ± 2.8 (*N* = 37)	NS
ΔP_G, *mm Hg*	31.7 ± 1.3	20.0 ± 1.9	<0.001
EFP_a, *mm Hg*	16.6 ± 1.8	5.4 ± 1.8	<0.001
SNGFR, *nl/min/kg body wt*	101.8 ± 9.7 (*N* = 42)	40.7 ± 6.0 (*N* = 68)	<0.001
AABF, *nl/min/kg body wt*	493.3 ± 53.9	444.3 ± 28.1	NS
AAPF, *nl/min/kg body wt*	255.2 ± 25.2	246.5 ± 18.0	NS
EABF, *nl/min/kg body wt*	391.6 ± 49.2	403.6 ± 24.7	NS
EAPF, *nl/min/kg body wt*	154.3 ± 21.9	205.9 ± 15.7	NS
P_a, *g/dl*	4.8 ± 0.2	4.7 ± 0.1	NS
P_e, *g/dl*	8.29 ± 0.44 (*N* = 33)	5.76 ± 0.22 (*N* = 45)	<0.001
π_a, *mm Hg*	14.7 ± 0.9	14.8 ± 0.7	NS
π_e, *mm Hg*	34.0 ± 2.9	19.2 ± 1.1	<0.001
$\pi_e/\Delta P_G$	1.09 ± 0.10	0.99 ± 0.07	NS
Hct_a, %	0.47 ± 0.02	0.45 ± 0.01	NS
Hct_e, %	0.61 ± 0.02	0.49 ± 0.01	<0.005
SNFF	0.41 ± 0.04	0.16 ± 0.02	<0.001
R_a, *dynes · sec · cm*$^{-5}$	5.88 ± 0.77	5.80 ± 0.68	NS
R_e, *dynes · sec · cm*$^{-5}$	3.50 ± 0.31	3.05 ± 0.30	NS

[a] Abbreviations used are: BP, arterial blood pressure; P_G, glomerular capillary pressure (hydrostatic); P_T, intratubular pressure; EAP, pressure in the first order peritubular capillary (so-called efferent arteriole pressure); P_G, hydrostatic pressure gradient across glomerular capillaries; EFP_a, effective filtration pressure at the afferent end of the glomerulus; SNGFR, single nephron filtration rate; AABF, afferent arteriole blood flow; AAPF, afferent arteriole plasma flow; EABF, efferent arteriole blood flow; EAPF, efferent arteriole plasma flow; P_a, systemic arterial protein concentration; P_e, oncotic pressure in afferent and efferent arteriole; Hct_a and Hct_e, hematocrit in afferent and efferent arteriole; SNFF, single nephron filtration fraction; R_a and R_e, resistance of single afferent and efferent arteriole. All values are means ± SEM. The results are given as the mean values of the averaged measurements in single rats. NS denotes not significant ($P > 0.05$) with unpaired Student's *t* test. Numbers in parentheses are numbers of observations.

tocrit (Hct_e) and protein concentration (P_e) at the exit of the efferent arteriole. Consequently, also peritubular capillary oncotic pressure (π_e) was lowered. Ureteral obstruction caused no change in efferent arteriole resistance (R_e) or in EABF. At the efferent end of the glomerulus, glomerular pressure equilibrium was taking place in control conditions as well as during BUL, the ratio $\pi_e/\Delta P_G$ not being different from 1 ($P > 0.1$). Finally, BUL caused a rise in P_U, from 5.9 ± 0.9 to 25.8 ± 2.7 mm Hg ($P < 0.005$).

Table 2. Average SNGFR in single rats, measured with conventional micropuncture technique and from Equation 1[a]

	SNGFR, *nl/min/kg body wt*		
Rat no.	Conventional technique	Equation 1	Average
1	22.8 (*N* = 3)	25.6 (*N* = 3)	24.2
2	60.1 (*N* = 4)	47.5 (*N* = 3)	53.8
3	30.4 (*N* = 3)	18.6 (*N* = 5)	24.5
4	51.2 (*N* = 4)	66.8 (*N* = 4)	59.0
5	23.9 (*N* = 2)	29.3 (*N* = 5)	26.6
6	36.7 (*N* = 4)	46.3 (*N* = 4)	41.5
7	22.1 (*N* = 4)	17.9 (*N* = 4)	20.0
8	43.1 (*N* = 3)	46.9 (*N* = 4)	45.0
9	64.5 (*N* = 5)	78.5 (*N* = 4)	71.5
Mean ± SEM	39.4 ± 5.4	41.9 ± 7.0	40.7 ± 6.0

[a] Numbers in parentheses are numbers of observations.

Postobstructive studies (Tables 3 and 4). The relief of the ureter caused a fall in P_T, from 27.5 ± 2.5 to 10.8 ± 1.1 mm Hg ($P < 0.0005$). P_G decreased, but to a lesser degree than P_T, so that both ΔP_G and EFP_a were increased. A marked rise in R_a (from 6.7 ± 0.8 to 10.8 ± 1.5 dyne · sec · cm^{-5}) determined a fall in both AABF and AAPF. SNGFR was maintained low, measuring 32.6 ± 6.1 nl/min/kg body wt during BUL, vs. 41.1 ± 5.2 nl/min/kg body wt in the postobstructive conditions ($P > 0.05$). The increase in SNFF was accompanied by a rise in both Hct_e and P_e. R_e was slightly augmented, but the difference did not attain statistical significance ($P > 0.05$).

Although GFR was markedly reduced in the released kidney of bilaterally obstructed rats, in the latter both urinary volume and urinary sodium excretion rate were higher than they were in the control kidney of sham-operated rats (Table 4).

Table 3. Pressures, flows, resistances, protein concentrations, and hematocrits during 24-hour bilateral obstruction and the postobstructive period[a]

	Obstructive period	Postobstructive period	*P*
BP, *mm Hg*	113.3 ± 9.2	109.2 ± 4.4	NS
P_G, *mm Hg*	46.7 ± 3.3 (*N* = 19)	37.0 ± 1.9 (*N* = 17)	<0.01
P_T, *mm Hg*	27.5 ± 2.5 (*N* = 29)	10.8 ± 1.1 (*N* = 29)	<0.005
EAP, *mm Hg*	16.9 ± 2.2 (*N* = 21)	11.5 ± 1.9 (*N* = 18)	<0.005
ΔP_G, *mm Hg*	19.5 ± 2.0	26.2 ± 1.4	<0.01
EFP_a, *mm Hg*	4.7 ± 1.5	11.7 ± 1.3	<0.01
SNGFR, *nl/min/kg body wt*	32.6 ± 6.1 (*N* = 26)	41.1 ± 5.2 (*N* = 22)	NS
AABF, *nl/min/kg body wt*	421.6 ± 30.9	292.7 ± 45.4	<0.005
AAPF, *nl/min/kg body wt*	228.2 ± 19.6	157.9 ± 20.7	<0.005
EABF, *nl/min/kg body wt*	388.9 ± 27.3	251.9 ± 42.8	<0.005
EAPF, *nl/min/kg body wt*	195.6 ± 17.4	113.6 ± 17.2	<0.005
P_a, *g/dl*	4.86 ± 0.24	4.84 ± 0.27	NS
P_e, *g/dl*	5.70 ± 0.34 (*N* = 21)	6.66 ± 0.27 (*N* = 18)	<0.025 <0.005
π_a, *mm Hg*	15.1 ± 1.0	15.1 ± 1.6	NS
π_e, *mm Hg*	19.0 ± 1.7	23.9 ± 1.5	<0.025
$\pi_e/\Delta P_G$	0.99 ± 0.06	0.91 ± 0.07	NS
Hct_a, %	0.46 ± 0.01	0.45 ± 0.02	NS
Hct_e, %	0.49 ± 0.02	0.53 ± 0.02	<0.025
SNFF	0.14 ± 0.02	0.27 ± 0.03	<0.025
R_a, *dynes · sec · cm*$^{-5}$	6.68 ± 0.76	10.84 ± 1.51	<0.005
R_e, *dynes · sec · cm*$^{-5}$	3.13 ± 0.20	4.79 ± 0.91	NS

[a] Abbreviations are defined in Table 1. All values are means ± SEM. The results are given as the mean values of the averaged measurements in single rats. NS denotes not significant with paired Student's *t* test. Numbers in parentheses are numbers of observations.

Discussion

In the present study, 24 hours after bilateral ureteral ligation, a fall in SNGFR was observed, accounted for by a decrease in effective filtration pressure. The latter was entirely due to a rise in P_T, glomerular hemodynamics, and particularly P_G being unmodified.

The reduction in SNGFR persisted in the postobstructive period, but the reason was quite different. In fact, after releasing the ureter, we noted a striking increase in afferent arteriole resistance, which decreased both P_G and glomerular plasma flow. The decrease in P_G was responsible for a lower than normal effective filtration pressure, which undoubtedly contributed to impair SNGFR. EFP_a, however, was raised with respect to the previous, that is, obstructive setting, because the reduction of P_G was overwhelmed by a fall in P_T. Therefore, the maintenance of a low SNGFR was mainly accounted for by the decrease in glomerular plasma flow, in accordance to the high plasma-flow dependence of SNGFR in the Munich-Wistar rat [9], especially when filtration pressure equilibrium is taking place [10]. Because the latter obtained both in control and in obstructed rats, before and after releasing the ureter, a definite value of the ultrafiltration coefficient (K_f) could not be calculated [11]. Thus, we cannot exclude that some decrease in K_f contributed to reduce SNGFR.

The normality of glomerular hemodynamics during BUL stands in interesting contrast to the changes observed during unilateral ureteral ligation (UUL) in our laboratory [1]. As shown in Table 5, in fact, after 24 hours of UUL, a marked increase in afferent arteriole resistance takes place, accounting

Table 4. Urine flow, sodium excretion and inulin clearance from the left kidney of control (C) and previously obstructed (PO) rats[a]

	Inulin clearance *ml/min*	V *μl/min*	$U_{Na}V$ *μEq/min*
C	0.878 ±0.064	3.4 ±0.3	0.63 ±0.04
PO	0.217 ±0.024	6.5 ±0.5	1.48 ±0.09
P	<0.01	<0.05	<0.05

[a] Values are means ± SEM. Abbreviations used are: V, urine flow; $U_{Na}V$, urinary sodium excretion.

Table 5. Comparison of the effects of unilateral (UUL) and bilateral (BUL) ureteral ligation on glomerular hemodynamics[a]

	P_T	P_G	AAPF	R_a	SNGFR
24-hr UUL	=	↓	↓↓	↑↑	↓↓
24-hr BUL	↑↑	=	=	=	↓↓

[a] Abbreviations are defined in Table 1. Symbols are ↑, increased; =, unchanged; ↓, decreased. Number of arrows reflect the entity of the change.

for a fall both in glomerular capillary hydrostatic pressure and in glomerular plasma flow. Therefore, the cause of the reduction in GFR is quite different in BUL and UUL, being a rise in P_T in the former and a rise in R_a in the latter. The reason for this difference is not apparent from our studies. It has been suggested that the increase in R_a during UUL is accounted for by a reduced prostaglandin delivery to the superficial arterioles, secondary to a fall in distal tubular flow rate [1]. Prostaglandins, in fact, enter Henle's loops from the medullary interstitium, moving to the cortex via the distal tubular fluid [12]. Tubular flow rate was not measured in our experiments, nor was it reported by others, during BUL. After releasing the ureter, however, a marked decrease in proximal fractional reabsorption was found, maintaining distal tubular flow despite a fall in SNGFR [3, 5]. Although the preobstructive and postobstructive conditions cannot be soundly compared, because the relief of the obstruction modifies the hydrodynamic pressure gradient along the urinary spaces (that is, the driving force for tubular flow), it is possible that the cortical migration of prostaglandins was not as reduced during BUL as it was during UUL. Alternatively, at least two other vasodilating mechanisms may be operating in BUL, but not in UUL. The first possibility is that an extracellular volume expansion may occur in 24 hours of anuria. To prevent this possibility, however, we held our rats with BUL without water and food during the obstruction and we infused them at a low infusion rate (0.01 ml/min) to replace surgical fluid losses during micropuncture. Furthermore, no difference in blood protein concentration nor in hematocrit was found between control and obstructed rats, which would reasonably exclude in the latter an extracellular volume expansion. The second possibility is that some unidentified vasodilating substance, which is normally excreted in urine, is retained during BUL. The existence of such a substance was shown by the experiments of Harris and Yarger [13] and is suggested also by the diuretic and vasodilating effects of urine intravenously reinfused in the rat [14].

In the present study, a polyuria was taking place after releasing the ureter. Previous studies have shown that such a postobstructive diuresis is due to a fall in sodium reabsorption both in the proximal tubule and in the loop of Henle, raising distal sodium delivery [5]. According to the tubuloglomerular feedback hypothesis [15], this sudden increase in sodium chloride delivery to the distal tubule can well account for the marked increase in afferent arteriole resistance observed soon after releasing the ureter, as first suggested by Wright [16]. Alternatively, the ureter-obstructed kidney may be producing a vasoconstrictor material, as shown in isolated perfused rabbit kidneys by Morrison, Nishikawa, and Needleman [17]. Such a constrictor—identified as thromboxane A_2—may be antagonized in the anuric condition, as previously discussed, and manifest its effects only after ureteral release.

Finally, we emphasize that the afferent vasoconstriction was not accompanied by any change in efferent arteriole resistance. This appears in agreement with our previous studies showing that superficial efferent arterioles behave as passive vessels; that is, they are unable both to dilate and to constrict in response to neural and/or humoral stimuli [7].

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