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Acute Functional Adaptation to Nephron Loss: Micropuncture Studies

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The renal and proximal tubule response to contralateral kidney exclusion was studied in a variety of circumstances. Recollection micropuncture studies were performed to assess the response to contralateral kidney clamping in the normal or a remnant kidney of the dog. Acute clamping of the contralateral kidney for a normal and unilateral remnant kidney resulted in marked reduction in proximal TF/P inulin ratios in the experimental kidney reflecting a 15 percent reduction in fluid reabsorption. Mean fractional excretion of sodium, potassium and water increased significantly in remnant kidney dogs but no significant change was observed in normal dogs except for potassium excretion. The marked reduction in proximal reabsorption occurred as soon as 5-15 minutes after contralateral kidney clamping and was compensated by distal reabsorption. Acute obstruction of the contralateral ureter results in a similar markedly reduced proximal tubular reabsorption. The reduction in proximal reabsorption induced by contralateral clamping occurred in the presence of reduced perfusion pressure and volume expansion and to some extent with renal denervation. When prostaglandin E_2 or acetycholine were infused prior to contralateral kidney clamping, proximal reabsorption remained at control levels and the contralateral clamping response was blocked. Similar blockade occurred after treatment with indomethacin. Acute reduction in nephron mass causes a marked depression of proximal tubular sodium and fluid absorption not obviously accounted for by hemodynamic-physical factors and humoral factors may be involved. The level of distal reabsorption to increased proximal delivery following contralateral clamping, determines the net urinary excretion.

INTRODUCTION

Acute reduction in nephron mass either by uninephrectomy and tying both poles of the remaining kidney or chronically (1) clipping one kidney and acutely removing the other (2) or simply clamping the vascular pedicle of the contralateral kidney will lead to increase in water and sodium excretion. This increment in salt and water excretion can be accounted for by depressed proximal reabsorption [1,3]. Giebisch et al. [2] considered that elevation in both blood pressure and blood flow might explain the decreased proximal tubule reabsorption. Our present study was designed to examine the acute reduction of nephron mass by acutely clamping the pedicle or obstructing the ureter of the right kidney, and examining the effect of such a maneuver on reabsorption in the nephron of the left kidney. These studies indicate marked and rapid reduction in proximal reabsorption after contralateral clamping. We performed further studies to explore the role of perfusion pressure, volume expansion and the renal nerves with acute reduction of renal mass. Further studies were directed at certain chemical mediators such as prostaglandin and acetylcholine in terms of their influence on the reduction in proximal reabsorption during contralateral kidney clamping.

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METHOD

Acute Contralateral Clamping and Ureteral Obstruction

Two-phase recollection micropuncture experiments were performed in three groups of mongrel dogs anaesthetized with pentobarbital (30 mg/Kg). An endotracheal tube was inserted and all dogs were ventilated with room air using a Harvard respirator. Polyethylene catheters were placed into a jugular vein for inulin and para-aminohippurate infusions, a femoral vein for blood sampling and a femoral artery for blood pressure monitoring. Both ureters were catheterized at the level of the bladder by a suprapubic incision, and the left kidney was exposed through a flank incision and prepared for micropuncture. The kidney was lifted out of the body and mounted on a lucite kidney holder attached to a metal rod. The left renal artery was cannulated with a 27-gauge needle in a retrograde fashion close to its origin for the injection of FD and C green dye. About one square centimeter of the renal capsule was removed utilizing a microcautery in order to expose the superficial cortical nephrons illuminated by a fiberoptic source. Warmed mineral oil was permitted to drop onto the decapsulated surface of the kidney to prevent dehydration of the cortex.

The site of puncture was selected by the periodic injection of 0.1 to 0.2 ml of FD and C green dye into the left renal artery. Selected late proximal tubules for micropuncture were marked with nigrosine dye to allow recollection from the same site. The oil blocks were maintained immediately distal to the puncture site to prevent retrograde recollections. Approximately 20 to 40 nl of tubule fluid were obtained and stored with the pipette tip under mineral oil until analyzed for inulin, and in some instances sodium and potassium using techniques described in our laboratory [6].

The experimental protocol consisted of a control phase of hydropenia followed by an experimental phase in which the contralateral renal artery and vein were completely occluded. Micropuncture recollections were made sixty minutes after clamping. The following experimental groups were studied.

Contralateral Kidney Clamping or Obstruction

Group I consisted of eleven normal dogs. Group II included ten "remnant kidney" animals, in which segmental infarction of one kidney was induced by ligating three-fourths to five-sixths of the main branches of the renal artery. Micropuncture experiments were carried out two weeks later and this is the only group with puncture of the distal tubule. Group III consisted of five normal dogs in which the experimental phase consisted of complete obstruction of the ureter of the opposite kidney at the level of the renal pelvis with sampling at 90–120 minutes after complete obstruction.

Role of Perfusion Pressure, Volume Expansion and Renal Nerves in Proximal Reabsorption after Contralateral Kidney Clamping

To assess the role of perfusion pressure prior to volume expansion and neurologic factors in the acute compensating response, three-phase recollection micropuncture experiments were performed in four groups of normal dogs. In Group IV a hydropenic control phase was followed by repuncture after contralateral clamping within 15 and then again at 60 minutes. Simultaneously with the contralateral clamping maneuver, an aortic clamp was adjusted so as to keep the mean arterial pressure slightly below control.

In Group V, the protocol was identical except that perfusion pressure was deliberately lowered by an aortic clamp adjusted so that the perfusion pressure was

significantly lowered during the contralateral clamping phase to 90–95 mmHg, some 20 mmHg below control.

In Group VI animals received Ringer's infusion amounting to 3 percent of the body weight during the control phase and then the contralateral clamping maneuver was carried out. In these experiments perfusion pressure was not controlled but remained unchanged. In our last group (Group VII) we assessed the role of renal nerves on the contralateral clamping response. The kidney was first denervated during the control period by stripping all the nerves along the renal vessels and painting with xylocaine. Again, two recollections were taken after contralateral clamping.

Role of Prior Infusion of Prostaglandin in E_2, and Acetycholine and Indomethacin on Contralateral Kidney Clamping

To explore the possible role of chemical mediators in inducing the observed reduction in proximal reabsorption following contralateral clamping, recollection micropuncture studies were done in another four groups of normal animals. In Group VIII Prostaglandin E_2 was infused intravenously into 9 dogs at the rate of 10 μg/min during control and after contralateral clamping. In Group IX, Acetycholine was given to 6 dogs at the rate of 33 μg/min. during both phases. In Group X, ten dogs were given an intravenous infusion of 1 mg/kg/hr indomethacin to block prostaglandin synthesis in a three-phase experiment. In Groups VIII–X recollection micropuncture was performed sixty minutes following contralateral kidney clamping.

RESULTS

Acute Contralateral Clamping and Ureteral Obstruction (Groups I–III)

A summary of clearance data from the first three groups of dogs is shown in Table 1. GFR remained unchanged, while PAH clearance fell consistently in all three

TABLE 1
Summary of Clearance Data Before and After Contralateral Clamping or Ureteral Obstruction

Expt. Group	Expt. Phase	GFR ml/min	C_{PAH} ml/min	FE_{H_2O} %	FE_{Na} %	FE_K %
Group I Normal Dogs	Control	27 ± 2.8	79 ± 7.8	1.6 ± 0.56	1.0 ± 0.21	25 ± 3.5
n = 11	Acute clamping of right kidney	30 ± 3.1	65 ± 7*	2.0 ± 0.52	1.1 ± 0.2	32 ± 3.6*
Group II Remnant Kidney Dogs	Control	10 ± 1.7	36 ± 4.3	1.5 ± 0.3	0.8 ± 0.2	28 ± 3.1
n = 10	Acute clamping of right kidney	10 ± 1.7	25 ± 4.0*	2.8 ± 0.7*	1.3 ± 0.2*	37 ± 4.5*
Group III Normal Dogs	Control	31 ± 2.4	101 ± 10	1.4 ± 0.7	1.5 ± 0.9	20 ± 5.6
n = 5	Ureteral obstruction of right kidney	27 ± 2.8	67 ± 10	1.0 ± 0.2	1.1 ± 0.3	29 ± 3.6*

GFR = glomerular filtration rate; FE_{H_2O}, FE_{Na}, FE_K = fractional percentage in urine of filtered load of water, sodium and potassium; C_{PAH} = clearance of para-aminohippurate; ± = SEM.

*$p < 0.05$ in reference to the preceding phase.

TABLE 2
Summary of Micropuncture Data in Acute Clamping of Contralateral Kidney in Remnant Kidney Dogs

Experimental Phase	SNGFR nl/min	Proximal TF/P_{In}	Distal TF/P_{In}	Distal TF/P_{Na}	Distal TF/P_{K}
Control Period	70.9 ± 7.2	1.90 ± 0.15	5.63 ± 0.93	0.24 ± 0.04	0.64 ± 0.15
Experimental Period	69.2 ± 10.4	1.51 ± 0.07**	3.98 ± 0.39*	0.30 ± 0.03	1.34 ± 0.21*

TF/P = ratio of tubular fluid concentration to plasma concentration.
*$p < 0.05$ in reference to the preceding phase.
**$p < 0.01$ in reference to the preceding phase.
± = SEM.

groups. Contralateral clamping induced a significant rise in sodium and water excretion only in the remnant kidney dog group but the fractional potassium excretion rose significantly in every group. Micropuncture data obtained during acute clamping of the right kidney in normal dogs (Group III) showed that mean proximal TF/P inulin ratios decreased from 1.85 ± 0.09 to 1.45 ± 0.07—a highly significant reduction, reflecting a decrease in sodium and water reabsorption of 15 percent comparable to that observed after saline infusion.

Micropuncture data in the remnant kidney dogs are shown in Table 2. Mean proximal TF/P inulin ratios were significantly depressed by contralateral clamping from 1.90 to 1.51. Distal TF/P inulin ratios fell from 5.63 to 3.98, in keeping with a small diuretic response observed following contralateral clamping. It is to be noted that single nephron GFR remained constant. Mean single nephron glomerular filtration rate in the remnant kidney dogs in the two phases were 70.9 nl/min and 69.2 nl/min, respectively, and the corresponding TF/P sodium and potassium ratios were 0.24 and 0.30 and 0.64 and 1.34, respectively.

In five animals, after 90–120 minutes of complete contralateral ureteral obstruction, mean TF/P inulin declined from 1.78 ± 0.09 to 1.49 ± 0.02, representing an 11 percent decrease in fractional water reabsorption. Thus, in all these groups there was marked reduction in proximal reabsorption with reabsorption of the increased distal delivery as reflected by the increased potassium excretion.

Role of Perfusion Pressure, Volume Expansion and Renal Nerves on Response to Exclusion of the Contralateral Kidney (Groups IV–VIII)

The previous group of experiments had shown that sodium and water reabsorption in the proximal convoluted tubule is depressed immediately after clamping the contralateral kidney or complete contralateral ureteral obstruction. This change was not obviously related to changes in GFR or renal blood flow. The aim of the next group of experiments was to assess the effect of various factors that may affect proximal tubule sodium reabsorption and their relation to contralateral clamping.

In Group IV, simultaneously with the contralateral clamping mean arterial pressure was slightly below control. In Group V, the aortic clamp was adjusted so that the perfusion pressure was significantly lowered by 20 mmHg during the contralateral clamping phase. In Group VI perfusion pressure was not controlled by an aortic clamp but did not change significantly. Finally, in Group VII, blood pressure tended to fall with continued clamping during denervation.

Clearance data for this series are tabulated in Table 3. In all groups, the mean GFR

TABLE 3

Summary of Clearance Data: Effects of Changes in Mean Arterial Pressure, Volume Expansion and Renal Denervation

Experimental Protocol	Experimental Phases	GFR ml/min	C_{PAH} ml/min	FF %	FE_{H_2O} %	FE_{Na} %	FE_K %	MAP (mmHg)
Group IV	I Control	30 ± 2	109 ± 20	34 ± 4	0.4 ± 0.1	0.7 ± 0.2	22.8 ± 3.2	119 ± 6
Control	II 0–15 mins	25 ± 2	73 ± 10	40 ± 5	0.4 ± 0.1	0.8 ± 0.2	27.4 ± 3.0	114 ± 5
MAP	III 45–60 mins	25 ± 3	69.9 ± 9	39 ± 4	0.5 ± 0.1	0.5 ± 0.1	32.8 ± 3.6*	111 ± 7
Group V	I Control	33 ± 3	95 ± 15	41 ± 3	0.7 ± 0.2	0.8 ± 0.2	17.6 ± 2.0	113 ± 4
Lowered	II 0–15 mins	28 ± 3	83 ± 12	35 ± 6	0.7 ± 0.1	0.6 ± 0.1	23.8 ± 2.5	94 ± 5**
MAP	III 45–60 mins	30 ± 3	78 ± 13	46 ± 4	1.0 ± 0.3	0.7 ± 0.1	30.9 ± 4.1**	93 ± 3
Group VI	I Control	37 ± 3	121 ± 19	34 ± 3	5.6 ± 1.5	3.9 ± 0.9	38.4 ± 4.0	116 ± 4
3% Volume	II 0–15 mins	36 ± 4	94 ± 10	40 ± 2	6.0 ± 1.8	4.2 ± 0.8	41.7 ± 3.8	118 ± 5
Expansion	III 45–60 mins	39 ± 3	98 ± 13	40 ± 2	6.2 ± 1.9	3.5 ± 0.8	49.1 ± 6.3**	122 ± 6
Group VII	I Control	35 ± 3	144 ± 14	24 ± 2	1.1 ± 0.2	1.2 ± 0.3	24.8 ± 1.8	120 ± 3
Denervation	II 0–15 mins	32 ± 4	118 ± 16	30 ± 3	1.9 ± 0.4	1.6 ± 0.2	30.5 ± 1.5*	124 ± 6
	III 45–60 mins	33 ± 5	117 ± 21	29 ± 3	1.6 ± 0.3*	1.0 ± 0.1	31.4 ± 2.2*	110 ± 8

$FE_{H_2O, Na, K}$ = fractional excretion of water, sodium and potassium; GFR = glomerular filtration rate; C_{PAH} = clearance of para-aminohippurate; FF = filtration fraction; MAP = mean arterial pressure; ± = SEM.

*$p < 0.05$ in reference to corresponding control phase.

**$p < 0.01$ in reference to corresponding control phase.

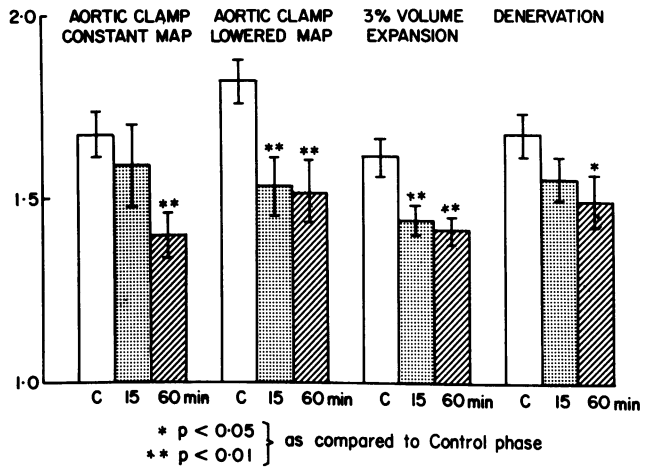

FIG. 1. Mean proximal tubule to plasma (TF/P) inulin ratios during control phase and following contralateral kidney clamping at 15 and 60 minutes in Group IV–VII.

remained unchanged, PAH clearances tended to decrease and the filtration fraction tended to increase. Fractional excretion of sodium remained unchanged. Again, a highly significant increase in fractional potassium occurred in each experimental group reflecting increased distal exchange for sodium.

Figure 1 summarizes the mean proximal TF/P inulin ratios for each of the four groups at 15 and 60 minutes after contralateral kidney clamping. It can be seen that proximal TF/P inulin fell significantly whether the perfusion pressure remained constant (Group IV) or was deliberately reduced (Group V). The response to contralateral clamping, thus, occurs despite moderately reduced perfusion pressure. In the volume expansion animals (Group VI) a significant reduction in TF/P inulin from 1.61 ± 0.04 to 1.44 ± 0.03 and 1.41 ± 0.03 during immediate and later recollections respectively. Finally in Group VII, following renal denervation, a smaller but still significant reduction in proximal TF/P inulin ratio from 1.67 ± 0.05 to 1.49 ± 0.06 occurred one hour after contralateral clamping.

Role of PGE and Acetycholine and Indomethacin in Response to Contralateral Clamping

The effect of prior infusion of prostaglandin E_2 and acetycholine as well as pretreatment with indomethacin to inhibit prostaglandin synthesis was tested on the proximal tubule response to acute contralateral clamping.

The mean GFR remained unchanged or fell significantly, and the PAH clearance tended to decrease. After clamping fractional excretion of sodium remained unchanged from control in PGE_2 (VIII) and acetylcholine (IV) groups and rose slightly in the indomethacin (X). However, a significant increase in fractional potassium excretion was again observed in all groups. Table 5 summarizes the mean proximal TF/P inulin noted in all four groups. It can be observed that TF/P inulin ratios did not decrease when PGE_2, acetylcholine or indomethacin were infused prior to contralateral clamping, suggesting that the response to contralateral clamping in the proximal tubule is blocked by the prior and continuous infusion of these agents.

DISCUSSION

Initial experiments were designed to assess the effect of acute reduction of renal mass on proximal reabsorption in the experimental kidney after acute ligation of the

TABLE 4
Summary of Clearance Data During PGE_2, Acetylcholine and Indomethacin Infusion Before and After Contralateral Clamping

Experimental Group	Experimental Phase	GFR ml/min	C_{PAH} ml/min	FE_{H_2O} %	FE_{Na} %	FE_K %
Group VIII PGE_2 $n = 9$	PGE_2 infusion control	28 ± 3	96 ± 10	2.99 ± 0.46	1.96 ± 0.31	30 ± 2
	PGE_2 infusion contralateral clamping	24 ± 2*	69 ± 6*	3.39 ± 0.38	2.02 ± 0.12	34 ± 2
Group IX $n = 6$	Acetylcholine infusion control	34 ± 6	135 ± 36	2.3 ± 0.8	1.3 ± 0.5	27 ± 2
	Acetylcholine infusion and contralateral clamping	29 ± 5	97 ± 13	3.3 ± 1.1	1.5 ± 0.6	35 ± 3*
Group X $n = 10$	Indomethacin control	41 ± 5	115 ± 10	0.38 ± 0.05	0.55 ± 0.14	16 ± 1
	Indomethacin and contralateral clamping	34 ± 2	84 ± 8*	0.64 ± 0.13	0.76 ± 0.16*	24 ± 3*

*$p < 0.05$

Refer to Table 3 for abbreviations.

contralateral renal pedicle or ureteral obstruction. A marked decrease in proximal reabsorption occurred comparable to inhibition observed after saline infusion and other studies in our laboratories indicated similar inhibition even after pretreatment with maximal doses of desoxycorticosterone and vasopressin. Even though exclusion of the contralateral kidney consistently reduced fractional reabsorption in the proximal tubule of the experimental kidney, this was associated with a significant increase in the fractional excretion of sodium, potassium and water only in the remnant kidney studies. In normal dogs, the fractional excretion of sodium and water was not significantly increased after ligation of the contralateral renal pedicle, but potassium excretion increased significantly, likely a consequence of augmented distal

TABLE 5
Summary of Micropuncture Data During PGE_2, Acetylcholine and Indomethacin Infusion During and After Contralateral Clamping

Experimental Group	TF/P Inulin Control	TF/P Inulin Recollection	P Value
Group VII PGE_2 $n = 9$	1.69 ± 0.12	1.78 ± 0.10	<0.02
Group IX Acetylcholine $n = 6$	1.59 ± 0.11	1.63 ± 0.10	NS
Group X Indomethacin $n = 10$	1.71 ± 0.07	1.71 ± 0.12	NS

Refer to Table 2 for abbreviations.

sodium delivery and reabsorption in exchange for potassium. In the remnant kidney studies (Group II), part of the increased sodium load to distal tubules was excreted. This indicates incomplete distal reabsorption of Na in the remnant kidney, as has been noted in our previous chronic studies of progressive reduction in renal mass in dogs [6,7].

Since the depression of proximal TF/P inulin ratios occurred very rapidly after the reduction in renal mass, a change in renal hemodynamics could be involved. To explore this possibility a separate study, as previously reported, was done in which renal blood flow was assessed by PAH extraction ratios and Xenon wash-out were studied following contralateral clamping. Total renal blood flow and its distribution remained unchanged [8].

The second set of experiments was undertaken to investigate the role of factors known to affect proximal tubule sodium reabsorption, such as changes of perfusion pressure, saline infusion and denervation, in the responses to contralateral renal exclusion. Again, GFR remained unchanged and the PAH clearance tended to decrease with a small but insignificant increase of the filtration fraction in each instance. Such an increase should increase proximal reabsorption since the filtration fraction affects the peritubular protein concentration. More direct measurements of peritubular protein concentration and hydrostatic pressure will be necessary to evaluate the role of "physical forces." Again, the urinary excretion of sodium remained unchanged from control in each instance, whereas the potassium excretion rate was significantly augmented after contralateral clamping. As before, increased delivery of proximal tubular fluid into the loop of Henle and distal tubule segment was accompanied by reabsorption, partly in exchange for potassium.

To investigate the possible role of perfusion pressure as a cause of reduced reabsorption, an aortic clamp was employed to maintain or lower mean arterial pressure. This study demonstrated that clamping the contralateral kidney rapidly and significantly reduced proximal tubule sodium and water reabsorption whether the perfusion pressure remained constant or was deliberately reduced by 20 mmHg. The clamping response appears to be mediated by factors other than the perfusion pressure.

In the volume expanded group, reduction in proximal reabsorption following contralateral clamping could still be demonstrated in the presence of modest volume expansion, suggesting that contralateral clamping may affect a site of proximal reabsorption not responsive to saline infusion or augment the effect of volume expansion. In the denervated kidneys, a smaller reduction in proximal TF/P inulin ratio was still observed, indicating that the response could not be abolished by denervation. Thus, changes in hemodynamic-physical factors as well as neurological factors did not appear to influence the adaptive response. On the other hand, chemical or humoral mediators may be important in rapidly changing proximal reabsorption when nephron mass is reduced.

An evaluation of a potential role of prostaglandins seemed an attractive possibility. Several recent studies have shown that PGE_2 is abundantly synthesized in the kidney [9] and Lee and Artallah [10] using a radioimmunoassay technique showed that PGA_2 is present in the renal medulla and in renal venous blood in high concentration. It has also been shown that a renal artery clamp [11], or renal ischemia [12] increased the amount of prostaglandin-like substances in renal venous effluent. When prostaglandins E and A were infused into the renal artery, renal blood flow and sodium excretion increased in anaesthetized dogs [13,14,15,16]. Since free water clearance increased during prostaglandin infusion, prostaglandins appear to depress sodium reabsorption from the proximal tubule. In addition, micropuncture studies

[13] suggest a distal site of action of prostaglandins. In the light of all these studies, it seemed possible that response to contralateral clamping in the proximal tubule could be mediated by a release of prostaglandin-like substances following the clamping maneuver. To explore this possibility, the animals in the last series were infused with PGE_2 prior to and after contralateral kidney clamping. Such infusions blocked the proximal response, suggesting that either prostaglandin-like substances block the factors involved in reducing proximal reabsorption after contralateral clamping, or in fact are released during contralateral clamping and are already exerting their own effect. However, it is known that infusion of other vasodilators can elicit a response similar to that of prostaglandins [16], suggesting a non-specific hemodynamic effect. Therefore, acetylcholine was observed to blunt the proximal tubule response to contralateral clamping. Finally, the administration of an inhibitor of prostaglandin synthesis, indomethacin, blocked the contralateral response to clamping suggesting further considerations of a role of prostaglandin. However, the interrelationships between the marked decrease in proximal reabsorption after contralateral kidney clamping and a specific role of prostaglandin as well as other vasodilators appear unclear at the present time and deserve further evaluation.

In summary, the marked reduction in the proximal tubule reabsorption indicates that an important compensatory mechanism is present to rapidly adjust proximal reabsorption when functioning renal mass is suddenly reduced. These studies do not obviously point to an hemodynamic mechanism but rather to an important role of a chemical mediator, possibly the prostaglandins.

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