Renal hydrogen ion secretion after release of unilateral ureteral obstruction

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THE EFFECTS OF unilateral ureteral obstruction (UUO) on renal function have been studied extensively (5, 6, 8, 11, 14, 22, 25, 30). After release of UUO, the postobstructed kidney exhibits a reduced glomerular filtration rate (GFR), renal plasma flow (RPF), and concentrating ability as well as enhanced phosphate reabsorption and decreased sodium reabsorption (5, 6, 8, 22). Studies in man and rats have also demonstrated that the postobstructed kidney has impaired hydrogen ion secretion (6, 27). Sodium reabsorption and hydrogen ion secretion are factors known to control bicarbonate reabsorption (18). One would, therefore, expect a different pattern of bicarbonate reabsorption in the postobstructed kidney (EK) as compared to the contralateral kidney (CK). Accordingly, we studied the effects of 24 h of UUO on bicarbonate reabsorption and urinary acidification.

METHODS

Twenty-eight experiments were performed on 28 female mongrel dogs. Twenty-four hours prior to the study, the dogs were anesthetized with sodium pentobarbital, 30 mg/kg iv. Through a suprapubic incision, the right ureter was identified and ligated completely. The incision was then closed and dogs were returned to their cages. Twenty-four hours later, the dogs were again anesthetized with sodium pentobarbital (30 mg/kg iv); light anesthesia, as judged by preservation of the corneal reflexes, was maintained by subsequent small doses. An endotracheal tube was inserted and connected to a Bird respirator; arterial CO₂ tension was kept between 35 and 45 mmHg by appropriate manipulation of the respirator. An arterial catheter was used to obtain arterial blood and to record and monitor blood pressure. A femoral vein catheter was used for infusions. Using a suprapubic incision, both ureters were identified and cannulated. Saline (0.9%) containing 115 μCi/liter was administered at 0.6 ml/min throughout each experiment as a marker of GFR. Saline containing 4 mg/ml of p-aminohippurate (PAH) was also administered at 0.5 ml/min throughout each experiment. An equilibration period of at least 60 min was allowed before any collection was started. Collection periods were started only when urine flow of both kidneys had stabilized. Collection periods were 10 min in duration except in group II in which collections were of 30 min duration. All blood samples collected were immediately replaced with an equal volume of 0.9% saline. Urinary losses also were replaced with 0.9% saline. Urinary losses also were replaced with 0.9% saline.

Group IA: bicarbonate loading. After two control periods, 11 dogs were infused with 0.9 M NaHCO₃ at varying rates to maintain a plasma HCO₃ concentration between 30 and 40 meq/liter. Clearance periods with HCO₃ concentration outside the range of 30–40 meq/liter were not used. After four clearance periods, the dogs were infused with 0.9% saline (10% body wt) for 100 min.

Group IB: bicarbonate loading plus phosphate infusion. Six dogs were infused with 0.9 M NaHCO₃ in order to achieve a maximally alkaline urine (urine pH 7.8–8.0). After three clearance periods, isotonic buffered phosphate solution (Na₂HPO₄ - NaH₂PO₄ in a molar ratio of 4:1) was infused at a rate of approximately 0.09

acid excretion; HCO₃ reabsorption; ureteral obstruction
Radioactive microspheres \( ( \text{MS}) \) 15 ± 5 \( \mu \text{m} \) in size labeled with \( ^{85}\text{Sr} \) or \( ^{141}\text{Ce} \) were injected into the left ventricle, and femoral arteries for blood sampling. Catheters were placed in the left ventricle (for microsphere injection) and in the femoral artery for blood sampling. Radioactive microspheres (MS) 15 ± 5 \( \mu \text{m} \) in size labeled with \( ^{85}\text{Sr} \) or \( ^{141}\text{Ce} \) were injected into the left ventricle within 10 s; a total amount of 15 \( \mu \text{Ci} \) (400,000 MS) was injected. Immediately after the administration of the microspheres, femoral arterial blood was drawn at a constant rate using a Sage pump over a 1-min period, and the volume withdrawn was noted. The first set of microspheres (\( ^{85}\text{Sr} \)) was injected 1 h after the release of the ureteral obstruction. The second set of microspheres was injected 1 h after the release of the ureteral obstruction, a second sample after 1 h of release of the ureteral obstruction, and the third after 10% body wt expansion with 0.9% saline. Three blood collections were drawn; one before the release of the ureteral obstruction, a second sample after 1 h of release of the ureteral obstruction, and the third after 10% body wt expansion with 0.9% saline (as in group I). Catheters were placed in the left ventricle (for microsphere injection) and in the femoral artery for blood sampling. Radioactive microspheres (MS) 15 ± 5 \( \mu \text{m} \) in size labeled with \( ^{85}\text{Sr} \) or \( ^{141}\text{Ce} \) were injected into the left ventricle within 10 s; a total amount of 15 \( \mu \text{Ci} \) (400,000 MS) was injected. Immediately after the administration of the microspheres, femoral arterial blood was drawn at a constant rate using a Sage pump over a 1-min period, and the volume withdrawn was noted. The first set of microspheres (\( ^{85}\text{Sr} \)) was injected 1 h after the release of the ureteral obstruction. The second set of microspheres (\( ^{141}\text{Ce} \)) was injected after 100 min of volume expansion with 0.9% saline. At the end of each experiment, both kidneys were removed.

Glomerular filtration rate, blood and urinary electrolyte determinations, and statistical analyses were performed as previously reported (18). Titratable acidity and ammonium were measured by the Formalin titrimetric method as described by Cunarro and Weiner (9). Plasma renin activity was measured by radioimmunoassay as described by Haber et al. (12).

**RESULTS**

Table 1 shows a summary of data obtained in the bicarbonate-loaded dogs. During control and bicarbonate loading, GFR was significantly lower in the experimental kidney than in the control kidney. Volume expansion led to a significant increase in GFR in the EK but not in the CK; after volume expansion there was no significant difference in GFR of the two kidneys. RPF was lower in the EK than in the CK, but this difference was significant only during bicarbonate loading. After volume expansion, RPF did not change significantly in either the CK or the EK. There was no significant difference in filtration fraction between the CK and the EK. During control, fractional sodium excretion, although slightly greater in the EK was not significantly different from the CK (3.3 ± 1.2 vs. 1.9 ± 0.6). Figure 1 plots bicarbonate reabsorption against plasma bicarbonate concentration during bicarbonate loading and volume expansion. As can be seen during bicarbonate loading, bicarbonate reabsorption is significantly higher in the EK than in the CK (2.59 ± 2.28 meq/100 ml of GFR, \( P < 0.01 \)). EK \( y = 3.05 - 0.01x \) and CK \( y = 1.31 + 0.03x \). The 95% confidence limits do not overlap. Figure 2 plots bicarbonate reabsorption against fractional chloride excretion. During bicarbonate loading, bicarbonate reabsorption for any given fractional chloride excretion was higher in the EK than in the CK. Volume expansion (Figs. 1 and 2) led to a significant depression of bicarbonate reabsorption in both kidneys. After volume expansion, there was no significant difference in bicarbonate reabsorption between the EK and CK.

**TABLE 1. Bicarbonate reabsorption after unilateral ureteral obstruction**

<table>
<thead>
<tr>
<th>Urine Flow</th>
<th>GFR</th>
<th>RPF (C_{\text{crea}})</th>
<th>Filtration Fraction</th>
<th>Plasma HCO(_3)</th>
<th>HCO(_3) Reabsorption</th>
<th>(C_{\text{crea}})/GFR</th>
<th>(C_{\text{crea}})/GFR</th>
<th>UKV</th>
<th>UpH</th>
<th>UP_{\text{U}}</th>
</tr>
</thead>
<tbody>
<tr>
<td>ml/min</td>
<td>mg/liter</td>
<td>mg/liter</td>
<td>(GFR)</td>
<td>%</td>
<td>µg/ml per 100 ml GFR</td>
<td>mmol/kg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EK</td>
<td>C</td>
<td>1.9 ± 0.2</td>
<td>17.1 ± 2.70</td>
<td>54.7 ± 12.6</td>
<td>0.31 ± 0.03</td>
<td>20.2 ± 0.80</td>
<td>19.4 ± 0.80</td>
<td>3.4 ± 1.40</td>
<td>7.9 ± 2.00</td>
<td>64.2 ± 9.40</td>
</tr>
<tr>
<td></td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.05</td>
<td>NS</td>
<td>P &lt; 0.05</td>
<td>P &lt; 0.001</td>
<td>NS</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.05</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.05</td>
</tr>
<tr>
<td>BL</td>
<td>2.2 ± 2.3</td>
<td>20.1 ± 2.00</td>
<td>56.5 ± 7.5</td>
<td>0.40 ± 0.02</td>
<td>32.7 ± 0.43</td>
<td>25.9 ± 0.70</td>
<td>4.9 ± 1.00</td>
<td>21.0 ± 0.40</td>
<td>103.5 ± 15.0</td>
<td>7.80 ± 0.03</td>
</tr>
<tr>
<td>VE</td>
<td>7.5 ± 1.2</td>
<td>23.8 ± 3.70</td>
<td>63.4 ± 11.0</td>
<td>0.38 ± 0.03</td>
<td>32.9 ± 0.43</td>
<td>18.9 ± 1.20</td>
<td>22.9 ± 2.10</td>
<td>39.6 ± 4.60</td>
<td>110.5 ± 9.30</td>
<td>7.69 ± 0.03</td>
</tr>
</tbody>
</table>

**C** values comparing the two control, the two bicarbonate-loading, and the two volume-expanded periods

**CK**

<table>
<thead>
<tr>
<th>C</th>
<th>NS</th>
<th>P &lt; 0.01</th>
<th>NS</th>
<th>P &lt; 0.01</th>
<th>P &lt; 0.01</th>
<th>NS</th>
<th>P &lt; 0.01</th>
<th>NS</th>
<th>P &lt; 0.01</th>
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<th>P &lt; 0.01</th>
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<tbody>
<tr>
<td>NS</td>
<td>NS</td>
<td>P &lt; 0.01</td>
<td>NS</td>
<td>P &lt; 0.01</td>
<td>NS</td>
<td>NS</td>
<td>P &lt; 0.01</td>
<td>NS</td>
<td>P &lt; 0.01</td>
<td>NS</td>
<td>P &lt; 0.01</td>
<td>NS</td>
</tr>
<tr>
<td>NS</td>
<td>NS</td>
<td>P &lt; 0.01</td>
<td>NS</td>
<td>P &lt; 0.01</td>
<td>NS</td>
<td>NS</td>
<td>P &lt; 0.01</td>
<td>NS</td>
<td>P &lt; 0.01</td>
<td>NS</td>
<td>P &lt; 0.01</td>
<td>NS</td>
</tr>
</tbody>
</table>

**VE**

| NS | NS | NS | NS | NS | NS | NS | NS | NS | NS | NS | NS | NS |

**Values are means ± SE.** C, control period; BL, bicarbonate loading period; VE, volume-expanded period.
Bicarbonate loading and volume expansion led to a significant increase in fractional phosphate excretion (\(\frac{C_{pco_2}}{GFR} \times 100\)) in both kidneys. Notice, however, that fractional phosphate excretion was significantly lower in the EK during control, bicarbonate loading, and volume expansion (Table 1, Fig. 3). Absolute phosphate excretion was also significantly lower in the EK than in the CK. Urinary Pco_2 during bicarbonate loading was significantly higher in the EK than in the CK (Fig. 1) (EK \(y = 4.36 - 0.07x\) and CK \(y = 1.59 + 0.01x\)). The 95% confidence limits overlap. Urine pH during control was significantly higher in the EK than in the CK. Urinary Pco_2 during bicarbonate loading was significantly higher in the CK (Fig. 3).

Fractional chloride excretion (\(\frac{C_{cl}}{GFR} \times 100\)) increased significantly in both kidneys after volume expansion; there was no significant difference in (\(\frac{C_{cl}}{GFR} \times 100\)) between the two kidneys (Fig. 3).
account for differences in U-B Pco₂ between the EK and CK.

**Group II.** Fractional sodium excretion \((\frac{C_{Na}}{GFR} \times 100)\) was significantly higher in the EK than in the CK after DOCA administration \(\frac{C_{Na}}{GFR} \times 100\) 4.0% ± 0.97 and 0.6% ± 0.23 for EK and CK, respectively, \(P < 0.01\) (Fig. 5). Administration of Na₂SO₄ led to a significant decrease in urine pH in both kidneys; the mean lowest urine pH in the EK was 6.31 and 5.1 in the CK (Table 2). There was no titratable acid excretion in the EK; this is presumably due to very low excretion of phosphate by the EK (19). Urine phosphate concentration was significantly lower in the EK than in the CK. Ammonium excretion increased significantly in both kidneys following Na₂SO₄ administration, but it was significantly higher in the CK. Potassium excretion after Na₂SO₄ administration was significantly higher in the CK than in the EK (Table 2).

**Group III.** Total renal blood flow and renal plasma flow were slightly, but not significantly, lower in the EK (Table 3). The values of RPF calculated by the microsphere method are not significantly different from those obtained in group I dogs measured by clearance of PAH. Volume expansion did not lead to any significant change in TRBF. Comparison of the distribution of the cortical blood flow of the EK and the CK reveals a decrease in zone II and an increase in zone IV of the EK. Taking zones I and II as outer cortex and zones III and IV as the inner cortex, the EK shows, compared to the CK, a decrease in fractional blood flow to the outer cortex and a significant increase to the inner cortex. Volume expansion did not lead to any change in distribution of the cortical blood flow either in the EK or the CK.

There was no significant difference in plasma renin activity (Table 3) between the EK and the CK in any period. Volume expansion led to a significant decrease in PRA in both kidneys.

**DISCUSSION**

Our data demonstrate that the postobstructed kidney had a higher bicarbonate reabsorptive rate than did the contralateral kidney. Volume expansion depressed bicarbonate reabsorption in both kidneys and abolished the difference in bicarbonate reabsorption between the two kidneys.

Other investigators have demonstrated enhanced phosphate reabsorption by the postobstructed kidney which is not abolished by volume expansion or parathyroid hormone administration (8, 22). Our data also demonstrate that phosphate reabsorption is higher in the EK during control, bicarbonate loading, and volume expansion. Phosphate and bicarbonate, substances mainly reabsorbed in the proximal tubule, are coupled to sodium reabsorption (8, 18). This suggests that proximal reabsorption is enhanced in the postobstructed kidney and is in perfect agreement with micropuncture experiments in rats, which showed a high proximal TF/P inulin ratio in the postobstructed kidney of the rat (14). This suggestion is further strengthened by the finding that the postobstructed kidney has impaired distal hydrogen ion secretion, as evidenced by the lack of rise in urinary Pco₂ during HCO₃ loading and impaired urinary acidification during sodium sulfate administration. Thus, enhanced distal bicarbonate reabsorption cannot account for the increased maximal reabsorptive rate of this substance.

The low urinary Pco₂ in the EK, however, deserves

**TABLE 2. Effect of sodium sulfate administration**

<table>
<thead>
<tr>
<th></th>
<th>Mean Lowest pH</th>
<th>UNH,V</th>
<th>Titratable Acidity</th>
<th>UKV</th>
<th>Urine PO₄</th>
<th>Phosphate Excretion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>mg/min per 100 ml per GFR</td>
<td>mg/min per 100 ml</td>
<td>mg/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>77.5 ± 14.90</td>
<td>6.7 ± 2.52</td>
</tr>
<tr>
<td>EK</td>
<td>7.06 ± 0.10</td>
<td>0.1 ± 0.07</td>
<td>0</td>
<td>NS</td>
<td>0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>P &lt;</td>
<td>0.02</td>
<td>0.01</td>
<td>0.05</td>
<td>NS</td>
<td>0.01</td>
<td>0.16</td>
</tr>
<tr>
<td>CK</td>
<td>6.31 ± 0.28</td>
<td>52.0 ± 12.50</td>
<td>19.7 ± 7.80</td>
<td>85.2 ± 6.10</td>
<td>130.4 ± 33.78</td>
<td>0.03 ± 0.09</td>
</tr>
<tr>
<td>Na₂SO₄</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>111.0 ± 16.10</td>
<td>4.3 ± 1.34</td>
</tr>
<tr>
<td>EK</td>
<td>6.53 ± 0.10</td>
<td>24.3 ± 0.80</td>
<td>0</td>
<td>NS</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>P &lt;</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>0.01</td>
<td>0.02</td>
<td>0.47 ± 0.10</td>
</tr>
<tr>
<td>CK</td>
<td>5.10 ± 0.10</td>
<td>95.5 ± 12.00</td>
<td>49.2 ± 5.10</td>
<td>175.4 ± 14.00</td>
<td>33.9 ± 10.34</td>
<td>0.47 ± 0.10</td>
</tr>
<tr>
<td>P values of control vs. Na₂SO₄</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>ER P &lt;</td>
<td>0.01</td>
<td>0.05</td>
<td>NS</td>
<td>0.02</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>CK P &lt;</td>
<td>0.01</td>
<td>0.05</td>
<td>0.01</td>
<td>0.001</td>
<td>0.02</td>
<td>NS</td>
</tr>
</tbody>
</table>

UNH,V, ammonium excretion; UKV, potassium excretion.
H⁺ SECRETION AFTER URETERAL OBSTRUCTION

TABLE 3. Renal hemodynamics after unilateral ureteral obstruction

<table>
<thead>
<tr>
<th></th>
<th>RBF (ml/min)</th>
<th>RPF (ng/ml per 3 h)</th>
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<tbody>
<tr>
<td></td>
<td>Zone 1</td>
<td>Zone 2</td>
</tr>
</tbody>
</table>

Further comment. Halperin et al. (13) have suggested that distal H⁺ secretion can be assessed by the urinary Pco₂ levels provided that the following criteria are met: a) urine must be alkaline; b) a sufficient amount of buffer must be present in the urine; c) carbonic anhydrase must be absent in the lumen of the distal nephron; d) the lower urinary tract must be relatively impermeable to carbon dioxide. During HCO₃ loading, bicarbonate excretion was significantly lower in the EK than in the CK, but there was no difference in the urine pH between the two kidneys. It is possible that the low urinary Pco₂ in the EK may be attributable to the lower bicarbonate excretion rather than to defective H⁺ secretion. This is highly unlikely for the following reasons: a) during HCO₃ loading, a high urinary Pco₂ can be demonstrated with different levels of bicarbonate excretion, provided the urine is alkaline (20); b) Portwood et al. (21) demonstrated in man that U-B Pco₂ increases sharply as bicarbonate excretion increases up to 300 μeq/min; beyond this level further increase in bicarbonate excretion was not accompanied by any additional increase in urinary Pco₂. c) In acute thoracic inferior vena cava constriction with bicarbonate loading, we found that despite a very low bicarbonate excretion U-B Pco₂ was not different from that seen in normal dogs provided that the urine was alkaline (unpublished observations). Thus, despite different levels of bicarbonate excretion, a high urinary Pco₂ can be achieved provided the urine is alkaline. It seems, therefore, unlikely that the difference in bicarbonate excretion could account for the low urinary Pco₂ in the EK.

Kennedy et al. (16) demonstrated, in vitro, that dehydration of H₂CO₃ was very rapid when nonbicarbonate buffer was absent. Kennedy et al. also demonstrated, in dogs, that there is a direct relationship between Pco₂ and buffer concentration in urine (17). It could be argued that low urinary Pco₂ from EK is due to the low urinary phosphate concentration. This is highly unlikely for the following reasons: a) Portwood et al. (21), Rector et al. (23), and Dorman et al. (10) have demonstrated that in the presence of very low phosphate excretion, comparable to that seen in this study, urinary Pco₂ rises during HCO₃ loading; Dorman et al. (10) also demonstrated that changes in urinary phosphate concentration of 100-fold were not associated with any significant change in urinary Pco₂, and 2) in dogs, subjected to thyroparathyroidectomy, with very low phosphate excretion we found that urinary Pco₂ rose during HCO₃ loading, as compared to the normal kidney, was the same, or less, but certainly not higher.
Sodium sulfate, however, led to normal acidification in the contralateral kidney but failed to elicit the same response in the postobstructed kidney. The reason why ammonium chloride resulted in acidification in the postobstructed kidney of the rat, whereas sodium sulfate failed to elicit the same response in the postobstructed kidney of the dog is unclear.

The mechanism responsible for the acidifying defect observed after release of unilateral ureteral obstruction is not clear. Since the postobstructed kidney shows functional evidence of damage to the deep nephrons, e.g., impaired concentrating ability, it is also possible that the impaired acidification may be due to defective bicarbonate handling in the deep nephrons. This is not likely for the following reasons: 1) maximal bicarbonate reabsorption was higher in the postobstructed kidney, and 2) after release of acute ureteral obstruction, Wilson (28) found no consistent change in nephron filtration rate between superficial and deep nephrons.

The mechanism whereby phosphate and bicarbonate reabsorption are enhanced in the EK is not clear. It has been suggested that the enhanced phosphate reabsorption is secondary to a decrease in filtered load of phosphate (8, 22). It is also possible that the enhanced HCO3-, reabsorption is due to disruption of glomerular tubular balance. A recent study, however, demonstrated that a decrease in GFR, comparable to that seen in our study, does not disrupt the glomerular-tubular balance for bicarbonate (4).

It is interesting to note that volume expansion corrected the enhanced bicarbonate reabsorption but failed to correct the enhanced phosphate reabsorption. This suggests that there is a difference in glomerular-tubular balance for bicarbonate and phosphate.

Bercovitch et al. (5) and other investigators have demonstrated a sodium leak in the postobstructed kidney along with enhanced free water clearance and decreased concentrating ability. Our data demonstrate a sodium leak only during deoxycorticosterone administration. Thus, the site of the leak is distal to the diluting segment, since proximal reabsorption is enhanced as well as free water clearance.

Our data, as well as that of other investigators, show that K+ excretion (14, 30) and urinary acidification (27) were markedly impaired in the EK. Harris and Yarger (14) demonstrated that the postobstructed kidney of the rat exhibits a decreased distal delivery of Na and decreased K excretion. Potassium secretion is dependent on distal sodium delivery; Harris and Yarger (14) attributed the reduced K excretion of the postobstructed kidney to the decreased sodium delivery rather than to a defect in the distal secretory mechanism for potassium. Our findings are more compatible with the latter hypothesis because the reduced potassium excretion was not corrected by increasing distal delivery with volume expansion or sodium sulfate administration. It has recently been demonstrated that Na-K-ATPase in outer medulla is decreased in the postobstructed kidney (29).

Outer medullary Na-K-ATPase is increased in rats fed a high-K diet, and it is thought to be important in K adaptation (24). It is possible that the decrease in Na-K-ATPase may play a role in the impaired potassium excretion by the postobstructed kidney.

The EK thus presents a generalized disorder of the distal nephron, as demonstrated by decreased sodium reabsorption after DOCA administration, impaired potassium and H+ secretion, as well as decreased concentrating ability. The postobstructed kidney showed redistribution of cortical blood flow from the outer cortex to the inner cortex as compared to the contralateral kidney. This finding is identical to those reported in UUO and during elevation of the ureteral pressure (3, 30). Saline expansion has also been demonstrated to cause a redistribution of cortical blood flow identical to that seen in UUO (7). Saline did not cause any further redistribution of blood flow in the EK nor did it affect the distribution of cortical blood flow in the CK. Several possibilities may explain this observation. First, Yarger and Griffith (30) have demonstrated that UUO causes redistribution of cortical blood flow in the EK as well as the contralateral kidney; it is possible that saline administration failed to produce redistribution because cortical blood flow, in both kidneys, was already redistributed by UUO. Second, it has been suggested that saline causes redistribution by producing interstitial edema of the outer cortex (15). This would result in decreased radioactivity per gram of tissue and would lead to an apparent redistribution to the inner cortex. It is possible that UUO caused interstitial edema in the EK (by a direct effect) and in the CK by volume expansion. Saline administration might thus fail to produce further redistribution.

It has been suggested that renin release is stimulated by a decrease in sodium load to or by decreased sodium transport across the macula densa. Ureteral obstruction is known to result in increased renin release by these two mechanisms (26). We were unable to demonstrate high PRA from the obstructed kidney, despite evidence suggestive of decreased sodium delivery and decreased transport of sodium in the distal nephron. We cannot explain the finding of a normal PRA from the EK.

In summary, the postobstructed kidney exhibits enhanced proximal H+ secretion and a generalized defect of the distal nephron characterized by impaired H+ and K excretion as well as decreased Na reabsorption after administration of exogenous mineralocorticoids.

This research was supported by the following grants: 1) Veterans Administration Central Office Grant 7083-04; 2) Veterans Administration Basic Institutional Support Grant 3024-01; 3) Veterans Administration Research Associate Grant 022-01.

J. A. L. Arruda is a Research Associate at the West Side Veterans Administration Hospital in Chicago, Ill.

Received for publication 2 June 1975.
H+ SECRETION AFTER URETERAL OBSTRUCTION

1239


