

Thesis Title      Effect of Diuresis on Ammonium Excretion  
after Unilateral Ureteral Obstruction in  
Rats

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#### ABSTRACT

The present studies indirectly investigated the site of tubular defect that leads to a reduction of ammonium excretion by the damaged kidney after 24 hours of unilateral ureteral obstruction (UUO) in rat. After the released of UUO, the kidney was unable to lower urine pH. The urinary pH of post-obstructed kidney (POK) was 7.28 as compared to 6.11 in the contralateral control kidney (CCK). The fractional bicarbonate excretion of the POK was 10 times the value of the CCK. The rate of ammonium excretion ( $U_{[NH_4]}V$ ) and ammonium index ( $U_{[NH_4]}V/GFR$ ) were both markedly reduced. Thus, the impaired urine acidification was due to reduction in hydrogen ion secretion, bicarbonate reabsorption and ammonium excretion. Induction of diuresis by loop diuretic, furosemide, resulted in increasing ammonium index in both kidneys. However, the Henle's loop of the POK may be

defective since the degree of the increasing was smaller than those observed in the CCK. Normal saline infusion also lead to increasing ammonium indices in both CCK and POK. However, this increased ammonium index the POK after saline loading was only 37% of the CCK in the normal condition and it was less than the corresponding value after furosemide infusion. Thus, there may be a reduction in ammonium generation and secretion at proximal tubule after UO. After right nephrectomy (RNx), both methods of diuresis induction resulted in a marked increase in GFR and also ammonium indices. It was suggested that the ammonium index of the POK is in part depending on GFR, which may vary with the number of functioning nephron. Thus, the reduction of ammonium excretion after 24 hrs UO be due to the reduction in ammonium generation and secretion at proximal tubule. Furthermore, ammonium index is increased irrespect of the methods of diuresis induction which strengthen the conclusion that the defective site of ammonium excretion after 24 hrs UO may be mainly at proximal tubule and the reabsorption at loop of Henle may not be the major contributing factor in this condition.