Reabsorption of Inorganic Phosphate in the Rat Kidney*

I. Saturation of Transport Mechanism

II. Suppression of Fractional Phosphate Reabsorption
Due to Expansion of Extracellular Fluid Volume

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Summary. Clearance methods were employed to study reabsorption of inorganic phosphate in the rat kidney.

Plasma inorganic phosphate concentration was raised stepwise by phosphate infusions. Reabsorption of inorganic phosphate increased and finally reached a maximal value (TmPO4), which, however, decreased during further phosphate infusions to about 60% of this value. This decline of TmPO4 during phosphate infusions could be identified as the result of the phosphate load per se rather than of the volume load. No evidence was obtained for net secretion of inorganic phosphate.

In experiments at endogenous phosphate levels, fractional reabsorption of inorganic phosphate was decreased by expansion of extracellular fluid volume from

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86% in the control to 75% (P < 0.001). A positive correlation was found between fractional reabsorption of water and fractional reabsorption of inorganic phosphate, which suggests a common link between sodium and inorganic phosphate reabsorption at different degrees of extracellular fluid volume expansion.

**Key-Words:** Renal Physiology — Electrolyte Physiology — Inorganic Phosphate Reabsorption.

Renal inorganic phosphate reabsorption, although definitely characterized as a process with limited transport capacity (Tm\(_{\text{PO}_4}\)) in man (Thompson and Hiatt [16]) and dog (Pitts and Alexander [12]), has been described for the cat (Eggleton and Shuster [4]) and for the rat (Crawford et al. [2]) as a mechanism, which does not exhibit saturation kinetics. In 1964, however, Strickler et al. [15] from their micropuncture experiments suggested, that inorganic phosphate transport in the rat kidney might be saturable as well.

Because of this controversy the present clearance experiments were designed to examine whether a Tm\(_{\text{PO}_4}\) in the rat kidney exists or not. During progress of these experiments Ginn and Shanbour [8] published data, which conclusively demonstrate limitation of PO\(_4\) reabsorption in the rat. The results of the present study confirm Ginn's findings insofar as a Tm\(_{\text{PO}_4}\) was established also.

In contrast to Ginn's findings, however, in the present experiments after Tm\(_{\text{PO}_4}\) was achieved by phosphate infusions at a rate sufficient to saturate the transport mechanism, this maximal reabsorptive capacity was not stable rather than it decreased during further increments of plasma phosphate concentration. This decrease could be the result of increased parathyroid activity as a response to the elevated plasma phosphate concentration. An alternative possibility could be that during phosphate infusion an expansion of extracellular fluid volume was produced, which *per se* suppressed reabsorption of inorganic phosphate as described recently for the proximal tubule by Frick et al. [7]. In order to differentiate between these two possibilities, additional experiments were performed, which excluded the volume mechanism and therefore pointed to increased plasma phosphate concentration as the cause of the decreased Tm\(_{\text{PO}_4}\) during phosphate infusions. In a third series of experiments, however, expansion of extracellular fluid volume without phosphate loading could be demonstrated to suppress fractional reabsorption of inorganic phosphate.

**Methods**

Female rats of the Sprague Dawley strain (270—300 g b.w.), kept on a normal diet (Altromin) with water ad libitum, were anesthetized with Inactin (Promonta) intraperitoneally (10 mg/100 g b.w.) and tracheotomized. The right femoral artery and vein were cannulated for blood sampling and infusions, respectively. The urinary