Proximal and distal tubule cells were examined for potential difference across the cell membrane by means of microelectrodes for the peritubular side and luminal side. The effect of the administration of mineralcorticoids on the potential difference (P. D.) were also studied.

Rats weighing 250-350 gm. were anesthetized with thiopental sodium; the left kidney was exposed and held tight in a cup-shaped holder to arrest the respiratory movements of the organ. In measuring the potential, the electrode was advanced slowly and impaled in a cell, so that the potential dropped abruptly down to a level approximately 60 to 70 mV. negative with respect to the extracellular space (baseline); it remained stable during the period of the impalement (30-60 sec.). On further pushing the electrode, the potential difference was abruptly reduced to about 15 to 25 mV. negative with respect to the extracellular space.

With P. D. of -60 to -70 mV. the tip of the electrode was considered to be inside of the cell; with P. D. of -15 to -25 mV., it was thought to be located within the tubular lumina.

For the identification of the site of puncture either with the distal or with the proximal tubule, indigocarmin was injected, for a distal tubule acquires a distinct blue color, while proximal tubules remain colorless.

In untreated rats, the mean peritubular P. D. was -65 mV. (S.D. ±10 mV.) for proximal tubules and -63 mV. (S.D. ±11 mV.) for distal tubules. The mean value of the transtubular P. D. was -19 mV. (S.D. ±4 mV.) in the proximal tubule and -23 mV. (S.D. ±8 mV.) in the distal tubule. There was no distinct difference in peritubular P. D. between proximal and distal tubules. On the other hand, standard deviation of the measured transtubular potential difference was larger for the distal tubules than for the proximal tubules. The reason for this may be that there is a greater variety of functional states of nephron in distal tubules than in the proximal tubules.

The effect of the administration of mineralcorticoids on P. D. was investigated. Firstly, with desoxycorticosterone glucoside (D.C.G.) injected subcutaneously in two doses 18 hours and 6 hours before the experiments, the peritubular P. D. became larger with the given dose of D.C.G.

With the injection of D.C.G. in dose of 8 mg., P. D. was significantly increased by 10 mV. compared with the control. These results apparently indicate that there
is a positive correlation between dosage of D.C.G. and the peritubular P.D. Secondly, P.D. was measured in untreated rats, then D.C.G. was injected intravenously in a dose of 1 mg. and changes in P.D. were studied in the course of time. In this case, increased peritubular and slightly increased transtubular P.D. resulted both in proximal and distal tubules.

Concerning the acting mechanism of mineralcorticoids on renal tubules, it has been said that they act on the distal tubules, however a theory was advanced by Nicholson that they act on the proximal tubules. This issue remains yet to be settled. The present results show that corticoids act on both proximal and distal tubules. It was shown by Giebisch that the tubular P.D. is due to a potassium diffusion potential. If this is really the case, it is suggested from our results that mineralcorticoids act directly on sodium-potassium exchange pump located in the tubular cell membrane and thus increase the peritubular P.D.; this further suggests that mineralcorticoids activate the pump mechanism and thereby promote both the tubular reabsorption of sodium and excretion of potassium.