SUMMARY

In this study 51 malnourished children and 10 normal control all were aged between one and 2 years, were studied in order to investigate the possible role of renin-angiotensin-aldosterone system in the pathogenesis of oedema of PEM.

Thorough clinical examination and anthropometric measurements with a detailed appraisal of the socio-economic and dietetic status was made. Laboratory determination of serum proteins, serum albumin, serum electrolytes (Na^+ and K^+) and radioimmunoassay of serum aldosterone concentration and serum renin activity were done for all the cases.

We found a significant decrease of serum proteins and serum albumin in Kwashiorkor and marasmic-Kwashiorkor cases. A significant increase in serum aldosterone in Kwashiorkor cases ($P < 0.005$), a non-significant increase in marasmic-Kwashiorkor cases ($P > 0.15$), and a significant increase in serum renin activity in both Kwashiorkor and marasmic-KWO cases ($P < 0.005$ and $P < 0.005$).

We found a positive relationship between serum aldosterone

concentration and the degree of oedema in both Kwashiorkor and marasmic-Kwashiorkor cases. The correlation coefficient r were 0.99 and 0.904 respectively.

Also a positive relationship between serum renin activity and the degree of oedema in Kwashiorkor and marasmic-Kwashiorkor cases. The correlation coefficient r were 0.843 and 0.828 respectively.

We found also a positive relationship between serum aldosterone concentration and the degree of hepatomegaly in Kwashiorkor and marasmic-Kwashiorkor cases. The correlation coefficient r were (0.82 and 0.132) respectively.

Our findings suggest that renin and aldosterone have a role in the pathogenesis of oedema as suggested before by Beitins et al. (1974), Kritzinger et al. (1972, 1974) and Worthington et al. (1977). Although the level of the hormones are affected by other factors such as protein binding in the plasma, liver catabolism and renal clearance, the significant raised levels of the hormones points clearly to their role in the pathogenesis of nutritional oedema. No one can deny the major role of the decreased serum albumin concentration in the pathogenesis of oedema of PEM as serum albumin is found persistently low in all the cases and

all of the previous studies (Whitehead et al., 1973; Mc Laren and Burman, 1976).

As for the role of potassium in the pathogenesis of oedema as has been suggested by Mc Cane et al. (1969) we found a non-significant decrease of serum potassium in the cases of PEM with oedema, but as has been pointed out by Mc Laren and Burman (1976) there is no direct correlation between total body potassium and serum potassium.