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Commentary

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Nephrotic syndrome is defined as oedema accompanied by proteinuria and hypoalbuninaemia, the oedema formation being due to retention of sodium by the kidney. The classic teaching is that this sodium retention is caused by stimulation of the renin-angiotensin-aldosterone system by the low plasma oncotic pressure. There is, however, mounting evidence that this theory is incorrect and that some other mechanism, possibly intrarenal, is responsible for the sodium retention. There are now many studies showing that plasma aldosterone, plasma renin activity, and blood volume may be low, normal, or high in patients with nephrotic syndrome while they are retaining sodium. Furthermore, sodium retention continues even after blockade of the renin-angiotensin-aldosterone system by captopril.

The paper by Bohlin and Berg in this issue confirms the lack of correlation between plasma aldosterone and urinary sodium excretion. The authors also show that there was no change in urinary sodium excretion in patients retaining sodium when acutely volume expanded with albumin. This is in keeping with other studies showing that many patients continue to retain sodium when given albumin for many days despite profound suppression of the renin-angiotensin-aldosterone system.

The actual mechanism of the sodium retention in nephrotic syndrome, however, remains unclear. The finding of decreased sodium excretion only by the affected kidney in the model of unilateral aminonucleoside induced nephrosis in the rat suggests that sodium retention may well be due to an intrarenal mechanism. What this intrarenal mechanism is, however, remains unknown.

Even the site of the increased sodium reabsorption is not fully established. This study favours the distal tubule yet there are other studies suggesting that proximal sodium reabsorption may be increased.

If an intrarenal mechanism is the cause of sodium retention in nephrotic syndrome, why is the renin-angiotensin-aldosterone system stimulated in some of the patients and not in others? The most likely explanation is that those patients with more severe hypoalbuninaemia develop a low blood volume which stimulates renin release. In normal subjects, a diminution of blood volume and stimulation of the renin system is a potent mechanism causing sodium retention by the kidney. In nephrotic patients, however, there seems to be an overriding mechanism, probably intrarenal, causing sodium retention independent of the renin system. The findings of Bohlin and Berg provide further support for this theory.