Dear Sir,

We read with great interest the article ‘Effect of Captopril on Blood pressure, Renal Function, the Electrolyte Balance and the Renin-Angiotensin System in Bartter’s Syndrome’ (Nephron 33: 274–278, 1983) by Aurell and Rudin. They concluded that ‘captopril had no effect on the mechanism leading to hypokalaemia in Bartter’s syndrome’. This conclusion is only correct for short-term treatment. We treated 2 patients having this disease with captopril on a long-term base. Like in the patients of Aurell and Rudin, no effect on serum potassium excretion was noticed during the first days of treatment; the long-term results were different.

In both patients serum potassium increased from values below 3.0 mmol/l to 3.5 mmol/l after 1 month, and this level has maintained for 18 and 6 months, respectively. The plasma aldosterone concentration was reduced in both patients to 25–50% of control levels.

The findings in our 1st patient are published elsewhere [1], those of our 2nd patient are shown in table I. Thus, it is possible to increase serum potassium levels in patients with Bartter’s syndrome by long-term treatment with captopril, probably by a sustained decrease in plasma aldosterone concentration.

Table I. Patient with Bartter’s syndrome treated by 25 mg captopril three times daily

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<tr>
<th>sK</th>
<th>PRA</th>
<th>PAC</th>
<th>BP</th>
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| = Serum potassium; PRA = upright plasma renin activity (reference value: 0.15–1.0 pmol/l/s); PAC = upright plasma aldosterone concentration (reference value: 0.10–1.0 nmol/l; BP = blood pressure.

References