Dear Sir,

There is still ongoing debate whether hypertension arising in renal allograft recipients on cyclosporin A (Cy-A) relates to stimulation of the renin-angiotensin-al-dosterone system (RAAS).

In animal experiments Cy-A has been demonstrated to enhance renin content and release from renal cortical slices and to increase plasma renin activity (PRA) [1–3]. In contrast, however, low or low-normal PRA was found in 5 of 7 hypertensive patients treated with Cy-A for renal transplantation [4].

We did multiple measurements of PRA and aldosterone in 11 Cy-A-treated renal allograft recipients (table 1). 10 of 11 patients were hypertensive and successfully treated with labetalol, nifedipine, and dihydralazine. We could not find changes in PRA and aldosterone up to 3 weeks after transplantation:

<table>
<thead>
<tr>
<th>Weeks after transplantation:</th>
<th>Plasma renin activity, ng/ml/h</th>
<th>Plasma aldosterone, pg/ml</th>
<th>Plasma creatinine, mg/100 ml</th>
<th>Blood pressure, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.31 ± 0.05</td>
<td>114 ± 27</td>
<td>72 ± 18</td>
<td>140/90</td>
</tr>
<tr>
<td>2</td>
<td>0.30 ± 0.03</td>
<td>72 ± 18</td>
<td>72 ± 18</td>
<td>140/90</td>
</tr>
<tr>
<td>3</td>
<td>0.32 ± 0.05</td>
<td>72 ± 18</td>
<td>72 ± 18</td>
<td>140/90</td>
</tr>
</tbody>
</table>

Although therapy with β-blockers has to be taken into account, there does not seem to be profound stimulation of basal PRA in our patients shortly after initiation of Cy-A therapy.

May be pharmacological stimulation of renin release by inhibiting angiotensin-converting enzyme will give more insight into the activity of the RAAS in renal allograft recipients. This
approach could also help to single out those patients whose posttransplantation hypertension might be improved by a converting enzyme inhibitor.

References