Dear Sir,

The kidneys (together with the liver) are responsible for most of the metabolism of polypeptide hormones; furthermore some hormones such as calcitonin (CT) and the N-terminal fragment of parathyroid hormone (PTH) are not metabolized by the liver, hence the role of the kidneys becomes more important [1]. On the other hand, it is yet unclear, whether nonfunctioning allografts take part in hormone metabolism either in posttransplantation acute tubular necrosis or in the phase of irreversible rejection.

During the investigation of the resolution of secondary hyperparathyroidism in 12 cadaveric renal allograft recipients, we noted an interesting finding. Serum levels of immunoreactive parathyroid hormone (iPTH) and CT were significantly lower in the early posttransplantation period compared with pretransplantation values (table 1). All of these recipients suffered posttransplantation acute tubular necrosis and were treated with supportive dialysis. Therefore, we concluded that the improvements in hormone levels were due to the contribution of nonfunctioning allografts to iPTH and CT metabolism. Serum levels of these hormones were not significantly lower in 2 patients with evidence of absent blood supply, as would be expected.

In the second part of the trial, serum levels of iPTH and CT were measured in 15 patients who had irreversible rejection 1–18 months before the study, and did not have transplant nephrectomy. These values were compared with the same parameters of patients undergoing dialysis in our center, and were found to be significantly lower (table 1). Two of the dialysis patients had been transplanted three years before, and suffered rejections 2 and 4 months, respectively, thereafter. These patients’ serum iPTH and CT levels were very high, similar to the levels of

Table 1. Serum levels of iPTH and CT before and 2 weeks after cadaveric renal transplantation, and in dialyzed patients with and without rejected allografts

<table>
<thead>
<tr>
<th>Serum levels of iPTH and CT</th>
<th>Pretransplantation</th>
<th>Posttransplantation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cadaveric renal transplantation</td>
<td></td>
<td></td>
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<tr>
<td>Dialyzed patients with rejected allografts</td>
<td></td>
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</tr>
</tbody>
</table>

Tx = Cadaveric renal transplantation.

Nonfunctioning Allografts Play an Important Role in Parathyroid Hormone and Calcitonin Metabolism
other dialysis patients. The findings suggest that left rejected allografts can also take part in hormone metabolism for definite periods after irreversible rejection, but later, they do not play any role at all. Our findings may seem to be discordant with those of Ardaillou et al. [2]. They have not observed any difference between the metabolic clearance rate of CT in nephrectomized and nonnephrectomized hemodialysis patients. But the consideration of the above-mentioned allografts as potentially viable organs with at least some functioning cells, and therefore different from that of the contracted, fibrotic, endstage kidneys of the patients undergoing dialysis, may explain this apparent discordance.

References

Erratum
Sir,
I wish to indicate an error in the Editorial that I wrote on ‘Long-Term Risks of Acute Pyelonephritis’ in the March issue of Nephron [1]. I mentioned Dr. James A. Roberts’ experimental work in the primate and indicated that he had induced acute Escherichia coli renal infection in the marmoset. In fact, his studies have not been done in this New World monkey, as this species has less receptors for P-fimbriated E. coli than Old World monkeys. This is why Dr. Roberts’ remarkable results were obtained in the rhesus monkey, Mucaca mulatta, a New World monkey. He also undertook experiments in the cynomolgus monkey, Macaca fascicularis and in the baboon, Papiopapio.
I apologize for my erroneous designation. The rest of my comments concerning the risk of cortical scars after acute renal E. coli infection in the Old World primate Homo sapiens remains valid.
Reference