step to clinical approach. Unfortunately, both appropriate control of hyperphosphataemia and calcitriol therapy frequently fail to control hyperparathyroidism, and parathyroidectomy is required for these patients.

Parathyroidectomy can be partial or total, with or without parathyroid fragment implant in the forearm. Several studies assessed the outcomes after each of these treatment modalities [1–4]. In patients with parathyroid fragment grafts in the forearm, functional assessment was limited to comparison of parathyroid hormone (PTH) concentrations in both arms [5–8].

As far as we know, there are no studies to assess the parathyroid graft function by analysis relating PTH secretion with serum calcium concentration after induction of hypocalcaemia. We have studied parathyroid function using this approach in a patient with hyperparathyroidism before and after total parathyroidectomy with heterotopic autotransplantation. This patient was a female aged 47 years undergoing haemodialysis for 115 months. Before total parathyroidectomy, intact PTH was 1031 pg/l (range 8–76), alkaline phosphatase 960 U/l (range 32–104), and total serum calcium 2.15 mmol/l. The X-ray of bones, scintigraphy with technetium and the bone biopsy showed hyperparathyroidism. The patient underwent parathyroidectomy. Four glands were removed with the following dimensions: upper-right 1.9 × 0.7 × 0.3 cm; upper-left 2.0 × 0.9 × 0.5 cm; lower-right 1.3 × 0.8 × 0.5 cm, and lower-left 2.0 × 0.9 × 0.5 cm.

Twenty fragments of 1 mm were implanted under the brachioradial muscle in the arm contralateral to the arteriovenous fistula. Fifteen and forty-five days after the surgery, intact PTH was 13.0 and 13.4 pg/l, alkaline phosphatase 1291 and 587 U/l, and serum calcium 2.05 and 2.1 mmol/l respectively. The histological examination shows parathyroid hyperplasia.

To assess PTH secretion before and after parathyroidectomy, the patient underwent haemodialysis with calcium dialysate zero (hypocalcaemia) and 2 mmol/l (hypercalcaemia). These studies were carried out 10 days before and 15 days after parathyroidectomy.

Figure 1 shows the relationship between ionized serum calcium and intact PTH in the arm contralateral to the graft.
Table 1. Relationship between the PTH concentration in the antecubital vein of the grafted arm and the non-grafted arm

<table>
<thead>
<tr>
<th>Months after parathyroidectomy</th>
<th>Intact PTH (pg/l)</th>
<th>R</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Grafted arm</td>
<td>Non-grafted arm</td>
</tr>
<tr>
<td>20</td>
<td>874</td>
<td>69.1</td>
</tr>
<tr>
<td>42</td>
<td>519</td>
<td>19.8</td>
</tr>
<tr>
<td>48</td>
<td>1536</td>
<td>76.8</td>
</tr>
<tr>
<td>65</td>
<td>965</td>
<td>43.0</td>
</tr>
<tr>
<td>72</td>
<td>1965</td>
<td>82.9</td>
</tr>
</tbody>
</table>

The slope of both curves was very similar suggesting that the parathyroid sensitivity to calcium was the same before and after parathyroidectomy. However, there was a shift of the sigmoidal curve to the right and upward after parathyroidectomy. Therefore, the set point shifted from 0.96 mmol/l before parathyroidectomy to 1.21 mmol/l after parathyroidectomy. Moreover, before parathyroidectomy the maximally inhibited secretion of PTH was approximately 8% of the maximal PTH secretion. It was shifted to 40% after parathyroidectomy. This behaviour of the sigmoidal curve suggests that the parathyroid graft had functional characteristics of a parathyroid adenoma, i.e. that PTH secretion was only partially blocked by the increase in calcemia.

The calcium-PTH sigmoidal curve of patients undergoing dialysis shows that the range of ionized serum calcium between maximum stimulation and inhibition of PTH secretion is very small, approximately 0.95 and 1.25 mmol/l [9]. In our patient, the range was between 0.88 and 1.32 mmol/l, before and between 0.89 and 1.24 mmol/l, after parathyroidectomy. We assume that within these ranges, we have achieved maximum and minimum stimulation to PTH secretion. If this assumption is correct the parathyroid graft had autonomous PTH secretion that was only partly blocked by increased calcium concentration.

Long term studies have showed that for patients undergoing subtotal or total parathyroidectomy with autotransplantation, one third of patients keep normal function of the parathyroid, one third develop hypoparathyroidism, and one third have hyperparathyroidism again [4].

PTH secretion is directly proportional to the parathyroid mass. On the other hand, hypocalcaemia, hyperphosphataemia and deficiency of active vitamin D are powerful stimuli of PTH secretion. It is possible that the presence of these factors associated with implantation of cells coming from an adenomatous portion of the parathyroid gland increase the probability of hyperparathyroidism after parathyroidectomy. Moreover, before parathyroidectomy the maximal PTH secretion. It was shifted to 40% after parathyroidectomy. This behaviour of the sigmoidal curve suggests that the parathyroid graft had functional characteristics of a parathyroid adenoma, i.e. that PTH secretion was only partially blocked by the increase in calcemia.

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Recently Walgenbach et al. [8] suggested that recurrence of graft dependent hyperparathyroidism is indicated by a PTH gradient between PTH concentration in the antecubital vein of the grafted arm versus non-grafted arm in excess of 20. Table 1 shows that in our patient this relationship was higher than 20 after the 42nd month after parathyroidectomy. We emphasize that PTH concentrations in the non-grafted arm were in normal range throughout. This observation suggests that whether manifest hyperparathyroidism develops, depends on the mass of parathyroid gland implanted in to the brachioradial muscle.