Increased Resting Energy Expenditure in Hemodialysis Patients with Severe Hyperparathyroidism

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Abstract. Several metabolic derangements, including enhanced protein catabolism, have been suggested to be associated with increased circulating parathyroid hormone (PTH) in patients with secondary hyperparathyroidism (HPT). Such conditions, therefore, might lead to an increase in energy expenditure. The present study examined by indirect calorimetry the resting energy expenditure (REE) of 15 hemodialysis patients who have severe HPT (PTH = 1457 ± 676 pg/ml) and were pair-matched for age and gender to 15 hemodialysis patients with mild to moderate HPT (PTH = 247 ± 196 pg/ml). Both groups were also pair-matched for age and gender to a group of 15 healthy adult subjects (control). In six patients from the severe HPT group submitted to total parathyroidectomy, REE was determined 6 mo after the surgery. The groups were not different regarding lean body mass (LBM) measured by bioelectric impedance, serum C-reactive protein, and bicarbonate. Thyroid-stimulating hormone was within the normal range in all groups. Nonadjusted REE was significantly higher in the severe HPT group (1674 ± 337 kcal/d) compared with patients with mild to moderate HPT (1388 ± 229 kcal/d; P < 0.05). Both groups did not differ from the control group (1468 ± 323 kcal/d). When adjustment of REE for LBM was performed using the multiple regression analysis, patients with mild to moderate HPT and control subjects had significantly lower REE (−231 and −262 kcal, respectively) than that of the severe HPT group. Considering all patients together, nonadjusted REE correlated directly with LBM (r = 0.61; P < 0.01). PTH correlated strongly with LBM in the severe HPT group (r = −0.82; P < 0.01). In the multiple linear regression analysis, only LBM and PTH were independent determinants of REE (n = 30; R² = 0.47). REE decreased significantly in the six patients who were evaluated 6 mo after parathyroidectomy (from 1617 ± 339 to 1226 ± 253; P = 0.02). These results demonstrate that hemodialysis patients with severe HPT have increased REE that might be reduced after parathyroidectomy.

Reasonable evidence exists to support the notion that parathyroid hormone (PTH) is a uremic toxin (1). Indeed, in excess, the hormone can contribute to many of the cellular and metabolic abnormalities frequently observed in patients with chronic kidney disease (CKD) (2). Increased circulating PTH may cause wasting, weight loss, weakness, muscle atrophy, and negative nitrogen balance in patients with primary and secondary hyperparathyroidism (HPT) (3–5). These observations suggest that PTH may exert its effect by affecting protein metabolism and/or bioenergetics of skeletal muscle. In fact, a study has shown that PTH impairs energy production, transfer, and utilization of skeletal muscle (6). In addition, Garber (7) demonstrated in vitro that PTH enhances muscle proteolysis and increases the release of alanine and glutamine. Increased protein catabolism, therefore, might increase energy expenditure and adversely affect the nutritional status of patients with secondary HPT. Actually, improvement in one or more nutritional markers has been observed in patients with severe secondary HPT after parathyroidectomy (8,9). The resting energy expenditure (REE) of patients with CKD has been demonstrated to be lower (10,11), similar to (12,13), or greater than (14) that of healthy individuals. However, the presence of HPT and its possible relationship with energy expenditure have not been investigated in the previous studies. Thus, in the present study, we tested the hypothesis that severe HPT might lead to an increase in REE of patients undergoing hemodialysis and that parathyroidectomy could reverse that condition.

Materials and Methods

Patients

This cross-sectional study was performed in 15 hemodialysis patients who had severe and uncontrolled HPT (S-HPT) and had already been referred for parathyroidectomy. These patients were pair-matched for age and gender to a group of 15 clinically stable hemodialysis patients with mild to moderate HPT defined as PTH <700 pg/ml (M-HPT). Inclusion criteria for both groups were patients who were older than 18 yr and had been on hemodialysis for >3 mo. Patients with active infectious or inflammatory disease, patients who were hospitalized within 3 mo before the study, and those who were receiving steroids and/or immunosuppressive agents were excluded from the study. Eleven patients in the S-HPT group and 10 patients in the M-HPT were taking antihypertensive drugs (β-blockers: five patients in the S-HPT group and four patients in the M-HPT group).
Therapy with oral calcitriol was used in three patients in the M-HPT group and in only two patients in the S-HPT group. The patients of the M-HPT group were being treated with 1.5 and 1.0 mg of calcitriol three times a week after the dialysis session, and one patient was receiving a daily dose of 0.25 µg. The patients of the S-HPT group were receiving 2.0 and 0.75 mg of calcitriol three times a week after the dialysis session. In the remaining patients of the S-HPT group, therapy with calcitriol was not possible because of persistent hypercalcemia and/or hyperphosphatemia.

In the S-HPT group, six patients were treated with calcium acetate, whereas in the M-HPT group, 11 were taking calcium acetate. Except for two patients in the S-HPT group who underwent dialysis for 2 h extra per week, all patients of both groups were receiving dialysis 4 h thrice a week. All patients had a native fistula as the type of dialysis access. Half of the patients in the S-HPT group and 73% in the M-HPT group were using polysulphone membrane dialyzers. The remaining patients were using cellulose acetate membrane.

Both patient groups were also pair-matched for age and gender with 15 healthy adult subjects (control), who were usually the patient’s relatives or employees of the clinic. Only subjects who were in good health, were not taking medications, and had normal thyroid and renal function were included. It was possible to study prospectively six of the 15 patients with severe HPT who were submitted to parathyroidectomy 6 mo after the surgery. The study was approved by the Ethics and Research Committee of the Federal University of São Paulo.

Study Protocol
In the cross-sectional study, the subjects were initially submitted to a first interview to meet the inclusion criteria and to obtain informed consent. Within 1 wk, the healthy subjects and the patients in a nondialysis day were scheduled to come to the clinic to be submitted to measurements of REE, body composition, and blood chemistries.

REE
REE was calculated on the basis of data collected by an open-circuit ventilated canopy indirect calorimetry using a computerized metabolic system (Vmax 29n; Sensormedics, Yorba Linda, CA). The oxygen and carbon dioxide sensors were calibrated before each measurement with the use of mixed reference gases of known composition. Subjects were admitted in the clinic at 8:00 a.m. after an overnight 12-h fast. The subjects rested comfortably in the recumbent position for 30 min in a thermoneutral environment and then had a clear plastic canopy placed over their heads. They were instructed to avoid hyperventilation, fidgeting, or falling asleep during the test. Oxygen consumption and carbon dioxide production were measured at intervals of 1 min during 30 min, and the mean of the last 20-min measurement period was used to calculate REE according to the Weir’s equation without using urinary urea nitrogen (15). The respiratory quotient was calculated as the ratio between the volume of CO₂ expired and the volume of O₂ consumed.

Anthropometry
Anthropometric measurements included body weight, height, triceps skinfold thickness (TSF), and midarm muscle circumference (MAMC). Body mass index (BMI) was calculated as body weight divided by squared height (16). TSF was measured using Lange Caliper (Cambridge Instrument, Cambridge, MD). The measurements were performed on the side of the body opposite that of vascular access for hemodialysis. MAMC was calculated using the formula

\[ \text{MAMC} = \text{arm circumference} - 0.314 \times \text{TSF}. \]

Percentage standard of TSF and MAMC were obtained using the National Health and Nutrition Examination Survey percentile distribution tables adapted by Frisancho (17).

Body Composition
For the cross-sectional study, bioelectrical impedance analysis (BIA) was used to estimate lean body mass (LBM). BIA was performed with the patient in supine position using a single-frequency (50 kHz) tetrapolar technique (BIA 101 Quantum; RJL Systems, Detroit, MI). The electrodes were placed in the standard tetrapolar positions of the non-access side of the patient. A current of 800 µA was introduced at the distal electrodes, and the voltage drop was detected by the proximal electrodes. The software Fluids & Nutrition (version 3.0) provided by the manufacturer was used to estimate body water, LBM, and fat mass.

For the six patients who were evaluated prospectively after parathyroidectomy, it was possible to analyze the body composition before and after the surgery by using dual-energy x-ray absorptiometry (DEXA). LBM and fat mass were determined by DEXA using the Hologic QDR model 4500 densitometer (Hologic, Waltham, MA).

Biochemical Parameters
All subjects had blood drawn under fasting condition. The control group underwent determinations of serum creatinine, C-reactive protein (CRP), and thyroid-stimulating hormone (TSH). On a nondialysis day the patients had blood drawn for determination of serum creatinine, bicarbonate, phosphorus, calcium, and alkaline phosphatase (colorimetric method; normal <270 U/L for men and <240 U/L for women).

Intact PTH was determined by a two-site chemiluminescent enzyme-labeled immunometric assay (Diagnostic Products Corporation, Los Angeles, CA; normal range, 10 to 65 pg/ml). We chose this method to determine PTH because it is used widely in the clinical setting, although it has been shown that this assay can overestimate PTH concentration. Moreover, it is still controversial whether the new assays that measure the whole molecule of PTH (1 to 84) reflect more accurately the bone histology compared with the intact PTH.

High-sensitivity CRP (normal value, <0.5 mg/dl) was determined by immunochemiluminescence. TSH was measured using an immunofluorimetric assay (normal range, 0.3 to 4.0 mU/L). Serum albumin was analyzed using bromocresol green technique (normal range, 3.4 to 4.8 g/dl).

Statistical Analyses
Statistical analysis was performed using True Epistat software (Epistat Services, Richmond, TX). Variables were checked for normality. Data are shown as mean ± SD, except for nonnormally distributed variables, for which geometric mean and median are shown. ANOVA complemented with Duncan test was used to compare the three groups for normally distributed continuous variables and Kruskal-Wallis ANOVA for nonnormally distributed variables. For comparisons between two groups, the paired t test was used for normally distributed continuous variables and Mann-Whitney test for nonnormally distributed variables. To test REE differences among the three groups adjusting for LBM multiple linear regression analysis was used. Spearman or Pearson correlation analysis was performed to determine the association between two variables considering respectively nonnormally or normally distributed variables. Multiple linear regression analysis was also used to investigate the independent determinants of REE and of LBM. To test a possible interaction between the variables used in the multiple regression analysis model.
a variance inflation factors test was used. The significance level for the REE comparison among the groups was fixed in 0.05, and the power was fixed in 0.80. Differences with \( P < 0.05 \) were considered statistically significant.

### Results

Table 1 depicts the main demographic and biochemical characteristics of the patients. The study was performed with eight male and seven female subjects in each group. S-HPT patients had been on dialysis for a longer period of time compared with M-HPT patients. One patient of the S-HPT group had type 2 diabetes as a comorbidity; however, he was in good blood glucose control. According to the Kt/V, patients of both groups were receiving an adequate dialysis dose. PTH was significantly and markedly elevated in the S-HPT group. TSH was in the normal range in the three groups. Although not significant, CRP tended to be higher in the S-HPT group. Only two patients in the S-HPT group had markedly elevated CRP concentration (9.1 and 7.6 mg/dl) despite that no signs of clinical inflammation had been detected. As the analysis excluding these two patients did not modify the results regarding REE, they were maintained in the study. Serum albumin was similar and in the normal range in both groups of patients. Total serum calcium and phosphorus were not significantly different comparing the two groups of patients. Alkaline phosphatase was significantly higher in S-HPT patients. The anthropometric parameters of the three groups are demonstrated in Table 2. Standard percentage of MAMC was significantly different comparing the two groups of patients. Alkaline phosphatase was significantly higher in S-HPT and the M-HPT groups (0.87 \( P < 0.05 \)). No difference in nonadjusted REE was observed between the control group (1468 \( \pm \) 323 kcal/d) and the patient groups. When adjustment of REE for LBM was performed using the multiple regression analysis, M-HPT patients and control subjects had REE significantly lower (\(-231\) and \(-262\) kcal, respectively) than that of the S-HPT group (Table 3, Figure 1). The respiratory quotient was significantly higher in the S-HPT and the M-HPT groups (0.87 \( P < 0.05 \) and 0.92 \( P < 0.06 \), respectively) as compared with the control group (0.81 \( P < 0.05 \)).

Considering all patients together, nonadjusted REE correlated directly and significantly with LBM (\( r = 0.61 \); \( P < 0.01 \)) and BMI (0.54; \( P < 0.05 \)). No significant correlation was found between REE and serum calcium, serum phosphorus, CRP, or bicarbonate. A significant correlation was observed

### Table 1. Main demographic and laboratory characteristics of the patients and control subjects

<table>
<thead>
<tr>
<th></th>
<th>S-HPT (n = 15)</th>
<th>M-HPT (n = 15)</th>
<th>Control (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (m/f)</td>
<td>8/7</td>
<td>8/7</td>
<td>8/7</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>38.8 ( \pm ) 12.0</td>
<td>39.5 ( \pm ) 11.8</td>
<td>39.4 ( \pm ) 11.8</td>
</tr>
<tr>
<td>Length on HD (mo)</td>
<td>62.8 (72)(^b)</td>
<td>13.4 (12)</td>
<td>–</td>
</tr>
<tr>
<td>Kt/V</td>
<td>1.41 ( \pm ) 0.3</td>
<td>1.37 ( \pm ) 0.2</td>
<td>–</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>7.4 ( \pm ) 1.5</td>
<td>8.4 ( \pm ) 1.9</td>
<td>0.8 ( \pm ) 0.2(^c)</td>
</tr>
<tr>
<td>PTH (pg/ml)</td>
<td>1302 (1171)(^b)</td>
<td>169 (194)</td>
<td>–</td>
</tr>
<tr>
<td>TSH (mUI/l)</td>
<td>1.71 ( \pm ) 1.33</td>
<td>1.60 ( \pm ) 1.20</td>
<td>1.77 ( \pm ) 0.88</td>
</tr>
<tr>
<td>C-reactive protein (mg/dl)</td>
<td>0.72 (0.84)</td>
<td>0.45 (0.53)</td>
<td>0.1 (0.07)(^d)</td>
</tr>
<tr>
<td>Albumin (g/dl)</td>
<td>4.04 ( \pm ) 0.5</td>
<td>4.30 ( \pm ) 0.3</td>
<td>–</td>
</tr>
<tr>
<td>HCO(_3) (mmol/L)</td>
<td>23.3 ( \pm ) 2.8</td>
<td>24.2 ( \pm ) 3.1</td>
<td>–</td>
</tr>
<tr>
<td>Calcium (mg/dl)</td>
<td>10.0 ( \pm ) 0.8</td>
<td>10.1 ( \pm ) 0.7</td>
<td>–</td>
</tr>
<tr>
<td>Phosphorus (mg/dl)</td>
<td>6.5 ( \pm ) 1.6</td>
<td>5.5 ( \pm ) 1.5</td>
<td>–</td>
</tr>
<tr>
<td>Alkaline phosphatase (U/L)</td>
<td>766 ( \pm ) 629</td>
<td>181 ( \pm ) 81(^b)</td>
<td>–</td>
</tr>
</tbody>
</table>

\(^a\) Values are expressed as mean \( \pm \) SD. BMI, body mass index; TSF, triceps skinfold thickness; MAMC, midarm muscle circumference; LBM, lean body mass.

\(^b\) \( P < 0.001 \) S-HPT versus M-HPT.

\(^c\) \( P < 0.001 \) control versus S-HPT and M-HPT.

\(^d\) \( P < 0.001 \) control versus M-HPT.
between PTH and length on dialysis ($r = 0.76; P < 0.01$). Moreover, PTH correlated inversely with LBM ($r = -0.34; P = 0.06$) and MAMC ($r = -0.58; P < 0.01$). Considering only patients of the S-HPT group, the correlations between PTH and LBM ($r = 0.82; P < 0.001$; Figure 2) and PTH and MAMC ($r = 0.76; P < 0.001$) were even stronger. In the multiple regression analysis, only PTH was the independent determinant of LBM (Table 4). PTH did not correlate with total calcium, and the correlation between PTH and serum phosphorus was of borderline significance ($r = 0.34; P = 0.06$). In the multiple linear regression analysis, we tested the variables of length on dialysis, CRP, LBM, and PTH as independent determinants of REE. The only independent determinants of REE were LBM and PTH ($n = 30; r^2 = 0.47$, Table 5). Because this result could have occurred as a consequence of the high degree of correlation between PTH and LBM, we tested the collinearity between these two variables in the multiple linear regression model and no collinearity was found between PTH and LBM in the model (variance inflation factors $= 1.148$).

The patients who were evaluated after 6 mo of parathyroidectomy were four women and two men, who had a mean age of $38.2 \pm 14.1$ yr and length on hemodialysis of $63.5 \pm 18.6$ mo. All of them were submitted to total parathyroidectomy. As expected, PTH concentration decreased significantly (Table 6). CRP did not change. LBM did not change, and body fat mass increased significantly from $14.1 \pm 5.7$ to $16.1 \pm 6.2$ kg ($P = 0.04$). REE decreased significantly 6 mo after parathyroidectomy (from $1617 \pm 339$ to $1226 \pm 253$ kcal/d; $P = 0.02$; Table 6), corresponding to a reduction of $23.1 \pm 14.2\%$. More important, the decrease in REE was observed in all six patients.

**Discussion**

The results of this study demonstrate that hemodialysis patients with severe secondary HPT have markedly increased REE when compared with patients with mild to moderate HPT. The difference was maintained even adjusting REE for LBM. It is noteworthy that besides LBM, a well-known determinant of REE, PTH was an independent determinant of REE as demonstrated by multiple regression analysis. The relationship between severe HPT and increased REE is also confirmed by the analysis of REE after parathyroidectomy, because the decrease in PTH was accompanied by a marked reduction in REE in all six patients studied. As far as we are concerned, this is the first study that analyzes the effect of elevated PTH concentration on energy expenditure in this population.

The precise mechanisms involved with the increased REE observed in the present study cannot be identified clearly.
However, considering the demonstrated toxic effects of PTH excess on various organs and body systems, some possibilities can be raised. Besides bone, several lines of evidence indicate that skeletal muscle is also a target organ for PTH. In fact, muscle dysfunction and wasting are common features of clinical states with PTH excess such as primary and secondary HPT (3–5). It seems that excessive PTH also affects bioenergetics of skeletal muscle, impairing energy production, transfer, and utilization (6). In addition, the hormone may enhance muscle proteolysis and increase release of alanine and glutamine in vitro (7). Indeed, negative nitrogen balance and net protein loss were observed in patients with primary HPT (18,19). PTH also plays a role in the development of glucose intolerance by interfering with the ability of the pancreatic β cells to augment insulin secretion appropriately in response to the insulin-resistant state (20,21). Impaired peripheral glucose utilization and cellular energy supply may affect protein metabolism (20). Although we have not evaluated any parameter related to protein metabolism, the inverse correlation found between PTH and markers of muscle mass, such as LBM and MAMC, and that PTH was an independent determinant of LBM may suggest indirect evidence regarding the action of PTH in muscle (Figure 2, Table 4). Although bioelectrical impedance may have some limitations related to the hydration status, studies have demonstrated good agreement with DEXA for the measurement of LBM (22).

Table 4. Multiple linear regression analysis using LBM as dependent variable (adjusted $R^2 = 0.17$)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>SEM</th>
<th>$P$ Value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTH</td>
<td>−0.0076</td>
<td>0.0035</td>
<td>0.041*</td>
<td>−0.015 to −0.0003</td>
</tr>
<tr>
<td>Length on dialysis</td>
<td>0.065</td>
<td>0.081</td>
<td>0.435</td>
<td>−0.102 to −0.23</td>
</tr>
<tr>
<td>CRPa</td>
<td>1.71</td>
<td>1.03</td>
<td>0.109</td>
<td>−0.41 to −3.83</td>
</tr>
<tr>
<td>Intercept</td>
<td>48.4</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*CRP, C-reactive protein.

Table 5. Multiple linear regression analysis using REE as dependent variable (adjusted $R^2 = 0.47$)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>SEM</th>
<th>$P$ Value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBM</td>
<td>20.27</td>
<td>3.909</td>
<td>0.000019*</td>
<td>12.25 to 28.29</td>
</tr>
<tr>
<td>PTH</td>
<td>0.16</td>
<td>0.0586</td>
<td>0.011*</td>
<td>0.0396 to 0.28</td>
</tr>
<tr>
<td>Length on dialysis</td>
<td>−2.32</td>
<td>1.78</td>
<td>0.202</td>
<td>−5.99 to 133</td>
</tr>
<tr>
<td>CRPa</td>
<td>17.34</td>
<td>23.4</td>
<td>0.468</td>
<td>−30.96 to 65.43</td>
</tr>
<tr>
<td>Intercept</td>
<td>459.1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*CRP, C-reactive protein.

Table 6. Laboratory parameters, body composition, and REE of the six patients who underwent parathyroidectomy

<table>
<thead>
<tr>
<th></th>
<th>Pre-PTX</th>
<th>6 Months Post-PTX</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTH (pg/ml)</td>
<td>1405 (1459)</td>
<td>8.30 (3.75)</td>
<td>0.03</td>
</tr>
<tr>
<td>CRP (mg/dl)</td>
<td>0.42 (0.45)</td>
<td>0.19 (0.12)</td>
<td>0.22</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>22.9 ± 4.2</td>
<td>23.7 ± 4.1</td>
<td>0.09</td>
</tr>
<tr>
<td>Body fat mass (kg)</td>
<td>14.1 ± 5.7</td>
<td>16.1 ± 6.2</td>
<td>0.04</td>
</tr>
<tr>
<td>LBM (kg)</td>
<td>44.1 ± 10.3</td>
<td>43.5 ± 9.3</td>
<td>0.38</td>
</tr>
<tr>
<td>REE (kcal/d)</td>
<td>1617 ± 339</td>
<td>1226 ± 253</td>
<td>0.02</td>
</tr>
</tbody>
</table>

*Values are expressed as mean ± SD and geometric mean (median). PTX, parathyroidectomy.

However, considering the demonstrated toxic effects of PTH excess on various organs and body systems, some possibilities can be raised. Besides bone, several lines of evidence indicate that skeletal muscle is also a target organ for PTH. In fact, muscle dysfunction and wasting are common features of clinical states with PTH excess such as primary and secondary HPT (3–5). It seems that excessive PTH also affects bioenergetics of skeletal muscle, impairing energy production, transfer, and utilization (6). In addition, the hormone may enhance muscle proteolysis and increase release of alanine and glutamine in vitro (7). Indeed, negative nitrogen balance and net protein loss were observed in patients with primary HPT (18,19). PTH also plays a role in the development of glucose intolerance by interfering with the ability of the pancreatic β cells to augment insulin secretion appropriately in response to the insulin-resistant state (20,21). Impaired peripheral glucose utilization and cellular energy supply may affect protein metabolism (20). Although we have not evaluated any parameter related to protein metabolism, the inverse correlation found between PTH and markers of muscle mass, such as LBM and MAMC, and that PTH was an independent determinant of LBM may suggest indirect evidence regarding the action of PTH in muscle (Figure 2, Table 4). Although bioelectrical impedance may have some limitations related to the hydration status, studies have demonstrated good agreement with DEXA for the measurement of LBM (22).

Elevated protein catabolism was also suggested in a previous study of our group. A higher blood urea nitrogen and protein equivalent of nitrogen appearance have been observed when hemodialysis patients who had moderate to severe HPT (PTH = 898 [439 to 2120 pg/ml]; median and range) were compared with hemodialysis patients who did not have HPT or had mild HPT (PTH = 155 [64 to 288 pg/ml]; median and range) and were pair-matched for age and gender and consumed similar amounts of protein and energy (23). Therefore, although we cannot exclude other possible mechanisms involved, it seems reasonable to speculate that protein catabolism caused by excessive PTH might be implicated in the higher REE. Indeed, the association between increased energy expenditure and elevated rates of whole-body and muscle proteolysis was observed by Ikizler et al. (24) in patients during hemodialysis, suggesting that, at least in part, protein catabolism contributes to increased energy expenditure.

Considering the higher REE and a possible elevated protein catabolism, we should expect to find differences in the nutritional status among the studied groups. A significant reduction in percentage standard of MAMC and a tendency to a lower TSF thickness was observed in the severe HPT patients when compared with those with moderate HPT. In addition, the mean values of these two parameters were indicative of malnutrition only in the severe HPT group (Table 2). However, we cannot exclude the possible influence of the longer length on dialysis of S-HPT patients on the decline of nutritional parameters.

If we consider the excess of PTH responsible for the deterioration of nutritional parameters of the patients, then an...
improvement in the nutritional status after effective treatment of secondary HPT would be expected. Only two previous studies have analyzed this issue. The first one, a retrospective study, observed a weight gain of more than 5% in 53% of the hemodialysis patients during the first year after total parathyroidectomy. No changes in albumin or protein equivalent of nitrogen appearance had been found (9). The second study was a prospective one in which 34 patients were followed for 12 mo after parathyroidectomy. A gradual improvement in albumin concentration and an increase in body weight were found after 12 mo. However, LBM did not change (8). Similar findings were observed in the present study regarding the six patients who were evaluated after parathyroidectomy. The decrease in REE was accompanied by a significant increase in fat mass, and the lack of change in LBM might be a consequence of the relatively short period of time after the surgery, and, more important, it seems that nutritional supplementation and/or physical activity is necessary for promoting muscle protein accretion in hemodialysis patients (25).

In accordance with the majority of the previous studies (12,13), the REE of our hemodialysis patients with mild to moderate HPT was not different from that of healthy subjects. Therefore, it seems that a condition of markedly elevated PTH concentration is required to cause an elevation of REE.

Although the present study has evaluated a small number of patients and has the limitation of being cross-sectional, we could speculate that the proposed recommendation of 30 to 35 kcal/kg per d for hemodialysis patients might not be sufficient to attend the energy requirement of patients with severe HPT. The same can be speculated regarding protein recommendation. Thus, until an effective treatment of the HPT is reached, a careful monitoring of the nutritional status of these patients should be used to guarantee the supply of sufficient amounts of energy and nutrients to overcome the possible higher requirements. Furthermore, a prospective study would be necessary to prove the relationship between malnutrition and HPT.

In conclusion, the results of the present study demonstrate that patients with severe HPT have increased REE and that the treatment of that comorbidity can result in a decrease in REE. However, further long-term studies with a greater sample size are needed not only to confirm our results but also to determine the underlying causes of this finding.

Acknowledgments

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References


**Access to UpToDate on-line is available for additional clinical information at [http://www.jasn.org/](http://www.jasn.org/)**