Bilateral subthalamic nucleus (STN) deep brain stimulation (DBS) significantly improves primary motor symptoms of idiopathic Parkinson’s disease (PD) including bradykinesia, rigidity, and tremor. STN-DBS has also been consistently shown to reduce the incidence of motor fluctuations, dyskinesia and levodopa requirements [1]. In past years a moderate-to-marked gain in body weight (BW) after STN-DBS has been reported [2-10]. This event may lead to a pathological increase in body mass index in some patients [2,6,9], exposing them to obesity-related health problems [11]. Despite several studies, the mechanisms underlying BW gain after STN-DBS are still elusive [2]. Our aim was to analyze neurological, psychological and endocrinological parameters in PD patients undergoing STN-DBS to better understand the causes of the BW gain.

From 2005 to 2011 thirty-four consecutive idiopathic PD patients underwent STN-DBS surgery in our center. Twenty-eight patients were included in our analysis while six patients were excluded due to prematurely death not related to surgery [2], electrodes reimplantation for infectious complications (n=3) and lost to follow-up (n=1). Patients were admitted to our department for 3 days at T0 (1.5±0.5 months pre-surgery), after 6 and 12 months (T6, T12) post STN-DBS. BW was measured with the same scale. During the hospitalization we collected prospectively the following data: Unified Parkinson’s Disease Rating Scale version 3.0 (UPDRS) part I (mobilization, behavior and mood), part II (Activities of Daily Living), part III (motor section) and part IV (off state, on medication) and total electrodes voltage (sum of left plus right side), levodopa-equivalent daily dose (LEDD), short form health survey (SF-36), apathy evaluation scale (AES), Beck depression inventory (BDI) and Epworth Sleepiness Scale (ESS). Blood samples were obtained at 8:00 am: prolactin, FSH, LH, free and total testosterone (male only), progesterone and estradiol (female only), ACTH, cortisol, TSH, FT4 and FT3 levels were determined at the hospital laboratory. Statistical analysis was performed with SPSS software: parametric and non-parametric tests for paired samples were used as appropriate.

**Materials and Methods**

Demographic and clinical baseline characteristics

<table>
<thead>
<tr>
<th>Cohort (28 pts)</th>
<th>Gender (male/female)</th>
<th>19/9</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD duration (years), MD±SD (range)</td>
<td>12.1±4.1 (8-20)</td>
<td></td>
</tr>
<tr>
<td>Age at STN-DBS (years), MD±SD (range)</td>
<td>61.1±6.8 (47-72)</td>
<td></td>
</tr>
<tr>
<td>UPDRS part II off state, MD±SD (range)</td>
<td>43.29±7.4 (24-96)</td>
<td></td>
</tr>
<tr>
<td>LEDD, MD±SD (range)</td>
<td>1088±374 (520-2112)</td>
<td></td>
</tr>
<tr>
<td>UPDRS IV dyskinesias score (item 32+33), MD±SD (range)</td>
<td>3.3±2.2 (0-6)</td>
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</tr>
</tbody>
</table>

**Body weight gain after STN-DBS**

![Figure 1. Mean body weight at baseline (T0), after six (T6) and twelve months (T12) post STN-DBS surgery. LEDD, levodopa-equivalent daily dose. Total electrodes voltage (left-right side voltage), LEDD, mean deviation, SD, standard deviation. **Regression.**](image)

**Discussion and Conclusions**

Mechanisms behind BW gain after STN-DBS are still a matter of debate. Some groups reported a clear association with dyskinetic reduction [2-4], while others did not find such correlation, pointing to hormonal changes [5-7], daily energy expenditure reduction [8] and direct stimulation of nearby hypothalamic nuclei (centers of metabolic regulation) [4, 10]. In our cohort BW increased after STN-DBS surgery by 2.9 Kg at T6 and by 5.4 Kg at T12 (mean values). Efficacy of STN-DBS (improvement of UPDRS part III evaluated during off-medications/stimulation state respect to pre-DBS off-medication state) was about 35%. There was also evidence of a significant reduction of dyskinesias (UPDRS IV item 32±33) and LEDDs and an improvement in quality of life (pSF-36) as in activities of daily living (UPDRS part II).

**References**