Subthalamic Nucleus Stimulation

Improvements in Outcome With Reprogramming

Elena Moro, MD, PhD; Yu-Yan W. Poon, RN; Andres M. Lozano, MD, PhD; Jean A. Saint-Cyr, PhD; Anthony E. Lang, MD

Background: Deep brain stimulation (DBS) is currently the most effective surgical treatment for advanced Parkinson disease (PD). Even when the electrode is well positioned in the target, the optimization of clinical results depends on careful programming of electrical parameters and changes in antiparkinsonian drug dosages.

Objective: To determine whether stable outcomes from subthalamic nucleus DBS for PD can be improved by revising stimulation parameters and drug dosages through “hands-on” involvement of a neurologist expert in both movement disorders and DBS programming.

Methods: In 44 consecutive patients with PD with long-term stable response to subthalamic nucleus DBS (mean ± SD, 3.5 ± 1.7 years), we compared scores from the Unified Parkinson’s Disease Rating Scale parts II through IV obtained immediately before and following a formal reprogramming of their stimulation. The reprogramming was performed by a neurologist expert in both PD and DBS and accompanied by further medication adjustment.

Results: In 24 patients (54.6%), the scores on the Unified Parkinson’s Disease Rating Scale parts II and III significantly improved by 15.0% and 25.9%, respectively. Anti-PD drugs were significantly reduced (by 25.9%). No improvement was observed in 16 patients (36.4%), and the conditions of 4 patients (9.1%) worsened.

Conclusions: Further improvement of parkinsonian signs can be achieved in the majority of patients even after long-term stable stimulation. Improved patient outcomes from subthalamic nucleus DBS are obtained when postoperative care is personally managed by a neurologist expert in movement disorders and DBS who is directly responsible for stimulation programming and simultaneous drug adjustments based on observed clinical responses to changing stimulation parameters.

Arch Neurol. 2006;63:1266-1272
We were interested in the clinical impact of this direct involvement of a movement disorders neurologist experienced in both the medical management of PD and DBS programming. We hypothesized that this approach to the management of DBS would result in improvement in patient outcomes, even in patients who were experiencing a stable response to their treatment following previous postoperative management that had not been directly performed by neurologists with this experience. We therefore evaluated the clinical results before and after revised programming and drug dosages in 44 patients with long-term, stable STN DBS for PD.

INITIAL POSTOPERATIVE CARE

Prior to the dedicated neurologist’s installation, programming was begun 2 to 3 weeks after surgery. Patients were asked to come to the clinic off medication (typically overnight). All electrode contacts were systematically tested at parameters of 60 µs pulse width and 130 Hz frequency in monopolar configuration, and we noted the alleviation of symptoms (especially tremor and limb rigidity). The voltage was increased in 0.5 V intervals until a stable “secondary” effect was produced (usually paraesthesia or muscle contraction). After the optimal contact was found in each side, systematic trials were begun with adjacent contacts and combinations of contact, including polarity reversal (Figure). Patients returned at daily or weekly intervals for further adjustments to programming, which was usually completed in a 3-month period. During the postoperative treatment period, stimulation parameters and particularly PD drug changes were regularly discussed with the supervising movement disorders faculty neurologist, but these individuals typically did not directly observe the efficacy or adversity of individual parameter settings. Table 1 lists the main patient characteristics before and after surgery prior to the new programming sessions. There was a significant and stable improvement in all the scores after DBS surgery, although before reprogramming, dyskinesia disability scores were no longer significantly improved (Table 2).

SECOND PROGRAMMING AND DRUG CHANGES

The 44 STN DBS patients had DBS reprogramming at the time of one of their routine follow-up visits. At the time of the reassessment, patients were initially assessed first thing in the morning in the defined off condition (after 12 hours of antiparkinsonian drug withdrawal)8 and with bilateral stimulation on. The patients and the new treating neurologist had not met each other prior to their assessment. The patients were informed that the purpose of the visit was to verify the functioning of the device by the reassessment of the benefit induced by the stimulation. The patients were then assessed with the UPDRS7 parts I through IV. Afterwards, the following paradigm10,11 was used by the same neurologist for reprogramming each stimulator (Figure). First, stimulation electrical parameters were set at 130 Hz and 60 µs. Second, rigidity at the wrist was assessed with stimulation off (contralateral to the assessed limb) at baseline and after 1 minute of fixed steps of 0.5 V stimulation, progressively increased up to the occurrence of adverse effects (muscle contractions, diplopia, etc.). The detected rigidity improvement was scored using a subjective scale (percentage of improvement) compared with the baseline. When the assessment of rigidity was not possible because of its absence, resting tremor (if present) and akinesia (finger tapping) were assessed. Third, rigidity (or tremor/akinesia) improvement was evaluated at each of the 4 contacts, starting from the most cranial to the most caudal. Fourth, the therapeutic window12 and setting of the stimulation was established for each electrode independently at the contact with the best benefit at the lowest voltage. In case of bilateral stimulation (40 patients), each side was initially assessed separately and then together.

The amplitude of stimulation was left just under the threshold that induced adverse effects or 3.6 V. However, during the programming, if stimulation-induced acute dyskinesia (SID) was observed at 1 contact, that contact was chosen as the final stimulation site because of the known consistent, good prognostic value of this response for subsequent clinical improvement.13,14 In this case, the voltage was left just below the threshold for inducing disabling dyskinesia. At this point, the new electrical parameters of stimulation were left for a predetermined period of observation with particular attention to

Figure. Timeline of events after surgery with first programming and reprogramming. PD indicates Parkinson disease; UPDRS, Unified Parkinson’s Disease Rating Scale.


©2006 American Medical Association. All rights reserved.
The late onset of SID (or exacerbation of existing dyskinesia) and gait changes. The patients were advised about the possible onset or worsening of dyskinesia within the next several hours and the possible worsening of their PD signs and motor fluctuations over the following days. If a consistent improvement of the PD signs or a worsening of dyskinesia was seen soon after the new programming, anti-PD medication dosages, starting with levodopa, were reduced by 50%. After the new programming, each patient was asked not to leave the hospital area for 1 to 2 hours in case further modification of DBS settings were required because of the onset of delayed dyskinesia or unpredictable clinical worsening. Patients coming from a long distance were asked to stay a few days in the Toronto area for completion of the new programming. Patients living close to Toronto were sent home and asked to call within the next several days to report on their status and to book a new visit over the following weeks or months, according to their clinical status. Because the assessments were not blinded, each patient was supposed to be reassessed at least twice over 14 months after the new programming for further assessments.
paring the follow-up baseline state (before reprogramming and score (part III) in the off medication/on stimulation state com-

ment was defined as 15% or more reduction in motor UPDRS version 5.0, SAS Institute Inc, Cary, NC). Clinical improve-

ments subsequent to the reprogramming (StatView for Windows, repeated measurements was used when comparing the follow-

programming at the first follow-up. The Friedman test for re-

coxon signed rank test was used to compare the UPDRS scores

out improvement after reprogramming. The nonparametric Wil-

13, 14, and 15). For part III, speech, facial expression, resting
tremor, bradykinesia, gait, and postural instability were chosen
to the new programming. In particular for part II, axial items were

selected, such as speech, falling, freezing, and walking (items 5,
to the value of the changes were made in group 1.

therapy was done in group 1.

and/or adjustments of stimulation parameters and/or anti-PD

medications.

The UPDRS parts II (activities of daily living) and III (motor)
total scores were the primary outcome variable) and some individual items of parts II and IV were used to assess the response to the new programming. In particular for part II, axial items were selected, such as speech, falling, freezing, and walking (items 5, 13, 14, and 15). For part III, speech, facial expression, resting tremor, bradykinesia, gait, and postural instability were chosen (items 18, 19, 20, 22, 23, 24, 25, 26, 29, 30, and 31). For part IV, duration of dyskinesia, early morning dystonia, and off duration were selected (items 32, 35, and 39).

STATISTICAL ANALYSIS

A series of t tests were performed to test for preoperative and postoperative differences between the patients with and without improvement after reprogramming. The nonparametric Wilcoxon signed rank test was used to compare the UPDRS scores and items before and after surgery and before and after the reprogramming at the first follow-up. The Friedman test for repeated measurements was used when comparing the follow-ups subsequent to the reprogramming (StatView for Windows, version 5.0. SAS Institute Inc. Cary, NC). Clinical improvement was defined as 15% or more reduction in motor UPDRS score (part III) in the off medication/on stimulation state comparing the follow-up baseline state (before reprogramming and drug adjustment) with the postreprogramming state.15

RESULTS

At the subsequent assessments after the reprogramming (mean, 5.0 months; range, 1 hour to 14 months), the patients could be divided into 3 groups. Group 1 had 24 patients (54.6%) who showed further clinical improvement (Table 3). Group 2 contained 16 patients (36.4%) who were unchanged (the motor UPDRS score changed by −14% to +9%; some of these patients had experienced unscored transient improvement) (Table 4). Group 3 contained 4 patients (9.1%) who were worse (>10% increase in the motor UPDRS score or subjective worsening) (Table 4). Patients who improved with reprogramming were significantly younger and had a better motor UPDRS score before surgery compared with patients who did not improve after reprogramming (Table 1). After surgery and before reprogramming, patients with further improvement had a total UPDRS IV score and dyskinesia duration significantly worse than the group without improvement (Table 2).

The most important changes made in the electrical parameters and settings of stimulation with the new programming are outlined in Table 5. Overall, the majority of the changes were made in group 1.

GROUP 1

All patients had changes in their stimulation settings after the new programming. Clinical assessment of the patients’ motor conditions showed a significant improvement of the motor UPDRS with 29.3% of the reductions at the first assessment following the new programming (Table 3). The improvement was significant for resting tremor, rigidity, and bradykinesia. The improvement was not significant for the other items. Thirteen patients (54.2%) developed transient SID after the new programming. Stimulation-induced acute dyskinesia occurred in 11 of these patients after we changed the previous contacts of stimulation (8 from bipolar to monopolar, 3 from 2 to 1 contact in monopolar). Subthalamic nucleus stimulation was still able to

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Baseline (n = 24)</th>
<th>First Follow-up (n = 24)</th>
<th>P Value†</th>
<th>Last Follow-up (n = 22)</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total UPDRS part III score in off meds/on stim (range 0-108)</td>
<td>35.8 (12.3)</td>
<td>25.3 (11.0)</td>
<td>.001</td>
<td>26.5 (11.3)</td>
<td>.03</td>
</tr>
<tr>
<td>Speech score (range 0-4)</td>
<td>1.9 (0.9)</td>
<td>1.7 (0.7)</td>
<td>.68</td>
<td>1.8 (0.6)</td>
<td>.77</td>
</tr>
<tr>
<td>Face expression score (range 0-4)</td>
<td>1.9 (0.8)</td>
<td>2.0 (1.0)</td>
<td>.89</td>
<td>1.8 (0.7)</td>
<td>.77</td>
</tr>
<tr>
<td>Resting tremor score (range 0-22)</td>
<td>2.4 (3.2)</td>
<td>1.0 (2.0)</td>
<td>.002</td>
<td>1.3 (1.8)</td>
<td>.008</td>
</tr>
<tr>
<td>Rigidity score (range 0-20)</td>
<td>7.2 (4.1)</td>
<td>4.0 (2.0)</td>
<td>.001</td>
<td>4.5 (3.6)</td>
<td>.01</td>
</tr>
<tr>
<td>Bradykinesia score (range 0-36)‡</td>
<td>18.9 (6.3)</td>
<td>11.5 (6.3)</td>
<td>.003</td>
<td>12.3 (6.1)</td>
<td>.03</td>
</tr>
<tr>
<td>Gait score (range 0-4)</td>
<td>1.7 (1.0)</td>
<td>1.3 (1.0)</td>
<td>.22</td>
<td>1.2 (1.0)</td>
<td>.12</td>
</tr>
<tr>
<td>Postural instability score (range 0-4)</td>
<td>1.4 (1.1)</td>
<td>1.0 (1.0)</td>
<td>.31</td>
<td>1.2 (1.0)</td>
<td>.52</td>
</tr>
<tr>
<td>Total UPDRS part II score in off meds/on stim (range 0-52)</td>
<td>20.0 (7.5)</td>
<td>16.7 (9.0)</td>
<td>.04</td>
<td>17.0 (8.0)</td>
<td>.04</td>
</tr>
<tr>
<td>Speech score (range 0-4)</td>
<td>1.9 (0.9)</td>
<td>1.8 (1.0)</td>
<td>.77</td>
<td>1.9 (1.0)</td>
<td>.89</td>
</tr>
<tr>
<td>Falling score (range 0-4)</td>
<td>1.2 (1.4)</td>
<td>0.9 (1.2)</td>
<td>.22</td>
<td>0.7 (1.0)</td>
<td>.12</td>
</tr>
<tr>
<td>Freezing score (range 0-4)</td>
<td>1.0 (1.0)</td>
<td>0.7 (1.0)</td>
<td>.03</td>
<td>0.9 (1.0)</td>
<td>.12</td>
</tr>
<tr>
<td>Walking score (range 0-4)</td>
<td>2.0 (1.0)</td>
<td>1.3 (1.2)</td>
<td>.01</td>
<td>1.6 (1.0)</td>
<td>.04</td>
</tr>
<tr>
<td>Total UPDRS part IV score in on stim (range 0-23)</td>
<td>5.9 (3.0)</td>
<td>3.6 (2.8)</td>
<td>.001</td>
<td>4.0 (2.0)</td>
<td>.02</td>
</tr>
<tr>
<td>Dyskinesia duration score (range 0-4)</td>
<td>0.9 (0.9)</td>
<td>0.7 (0.2)</td>
<td>.22</td>
<td>0.7 (0.9)</td>
<td>.52</td>
</tr>
<tr>
<td>Dyskinesia disability score (range 0-4)</td>
<td>0.4 (0.5)</td>
<td>0.2 (0.4)</td>
<td>.003</td>
<td>0.2 (0.7)</td>
<td>.03</td>
</tr>
<tr>
<td>Off duration score (range 0-4)</td>
<td>1.2 (0.8)</td>
<td>1.0 (0.9)</td>
<td>.28</td>
<td>1.0 (1.0)</td>
<td>.30</td>
</tr>
<tr>
<td>LEDD, mg</td>
<td>805.6 (498.9)</td>
<td>552.9 (365.7)</td>
<td>.001</td>
<td>596.6 (369.6)</td>
<td>.004</td>
</tr>
</tbody>
</table>

Abbreviations: DBS, deep brain stimulation; LEDD, levodopa equivalent daily dose; meds, medication; stim, stimulation; STN, subthalamic nucleus; UPDRS, Unified Parkinson’s Disease Rating Scale.

†Effects were measured by activities of daily living (UPDRS part II) and motor (UPDRS part III) total scores and subscores and medications (LEDD) at the first and last follow-up visits in the off medications/stimulation condition. All values except for the P values are mean (SD).

‡Bradykinesia was calculated from the sum of items 23, 24, 25, 26, and 31 from the Unified Parkinson’s Disease Rating Scale.
induce dyskinesia (often for the first time since surgery) years after the original DBS implantation (mean, 3.4 years; range, 2-7 years). Stimulation-induced acute dyskinesia started on average 9.9 hours (range, 1 minute to 48 hours) after the new programming and was severe in 7 patients. Two patients required subsequent bipolar stimulation (but leaving the new contact as cathode) to reduce dyskinesia; however, 1 of these 2 patients was able to switch to monopolar again in 1 month following levodopa dosage reduction and progressive voltage increase. The other patients with severe and moderated dyskinesia were managed through a concurrent progressive reduction or cessation of levodopa and slow progressive voltage increase. Overall, the optimization of SID required 1 to 2 weeks of balance between voltage increase and drug dosage reduction.11,16

All patients maintained their initial motor UPDRS score improvements at subsequent follow-up visits (on average, 5 follow-up visits over the next 14 months after the new programming; range, 1-16 follow-up visits) with 25.9% improvement at the last available assessment compared with the baseline (Table 3). Two patients who were assessed only on the day of the new programming because of difficulties returning for follow-up (long-distance, economic reasons, no caregiver availability, etc) confirmed ongoing stable benefit when contacted by telephone months after reprogramming.

The total UPDRS part II score in the off medication/on stimulation condition was significantly improved at the first assessment (16.5%) and last available follow-up (15.0%) (Table 3). Concerning selected items, speech and falling scores showed no significant improvement whereas freezing and walking scores improved significantly after the new programming. However, the freezing score improvement was no longer significant at the last follow-up.

After reprogramming and at last follow-up, the total UPDRS part IV and dyskinesia disability scores were significantly improved whereas dyskinesia and off duration improved but not significantly (Table 3). Four of 9 patients with off dystonia improved.

The levodopa equivalent daily dose17 was significantly reduced after the new programming (−31.4%) and only mildly increased over long-term follow-up (−25.9%) (Table 3). Two patients were able to stop receiving levodopa after the new programming. One of them was left receiving a dopamine agonist and the other was left receiving amantadine.

GROUP 2

Five of 16 patients in this group (31.2%) did not require any changes in their stimulation settings after the new programming. Three patients had unilateral STN DBS. Clinical assessment after the new programming did not show any significant difference in this group of patients at initial and subsequent follow-up visits (on average, 3 follow-up visits over the next 14 months after the new programming; range, 1-8 follow-up visits) in their UPDRS scores or levodopa equivalent daily dose compared with their baseline condition (Table 4). Only 1 patient experienced new SID during the reprogramming.

GROUP 3

Table 4 shows the main clinical characteristics of these 4 patients. Reprogramming resulted in further worsening of speech and gait in all 4 patients, requiring a return to their original settings.

---

### Table 4. Main Results in the 2 Groups of Patients Who Did Not Improve After the Reprogramming

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Group 2 (n = 16)</th>
<th>Group 3 (n = 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>First Follow-up</td>
</tr>
<tr>
<td>Total UPDRS part III score off meds, mean (SD)</td>
<td>34.2 (11.4)</td>
<td>34.9 (10.7)</td>
</tr>
<tr>
<td>(range 0-108)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total UPDRS part II score off meds, mean (SD)</td>
<td>19.1 (6.2)</td>
<td>18.8 (7.6)</td>
</tr>
<tr>
<td>(range 0-52)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total UPDRS part IV score, mean (SD) (range 0-23)</td>
<td>3.8 (3.0)</td>
<td>3.2 (3.8)</td>
</tr>
<tr>
<td>Dyskinesia duration score (range 0-4)</td>
<td>0.4 (0.5)</td>
<td>0.4 (0.6)</td>
</tr>
<tr>
<td>Dyskinesia disability score (range 0-4)</td>
<td>0.3 (0.5)</td>
<td>0.4 (0.5)</td>
</tr>
<tr>
<td>Off duration score (range 0-4)</td>
<td>0.8 (0.8)</td>
<td>0.6 (0.9)</td>
</tr>
<tr>
<td>LEDD, mean (SD), mg</td>
<td>726.2 (582.0)</td>
<td>718.7 (593.3)</td>
</tr>
</tbody>
</table>

Abbreviations: LEDD, levodopa equivalent daily dose; meds, medications; NA, not available in every patient; UPDRS, Unified Parkinson’s Disease Rating Scale.

*Difference compared with the baseline (ie, before vs after reprogramming for both first and last follow-up).
We postulated that personal performance of stimulation programming and drug changes by a movement disorders neurologist with extensive experience both in DBS and in the pharmacological management of PD would improve the outcomes of clinically patients benefiting from STN DBS whose initial postoperative care did not include this direct hands-on involvement of a DBS/movement disorders neurologist.

A further significant benefit was obtained in more than half of these 44 consecutive patients with long-term follow-up after STN DBS. These patients had previously undergone DBS programming by a movement disorders team member experienced in this task with supervision by a movement disorders neurologist. However, stimulation parameters and drug dosages had not been made on the basis of direct observations by this physician. Notably, these patients were relatively stable from the motor point of view after surgery, and their stimulator programming settings had been established within the first postoperative year. Indeed, the need for major changes in stimulation settings (ie, change of contact or polarity) or significant drug adjustments after the first year has not been reported in long-term follow-up studies.1,2 Instead, some studies have described a progressive loss of the stimulation benefit despite further adjustments of the stimulation parameters (ie, voltage and pulse width increase) and increases of the medication dosages.1,2 In contrast, patients in group 1 showed a further significant improvement of the motor signs and significant anti-PD drug dosage reduction after an average of 3 years of stimulation at the previous settings.

This experience raises a number of issues. First, there is a major need for neurologists trained in movement disorders who know how to manage patients with DBS. In-depth knowledge of DBS programming and contemporary management of anti-PD medication allows optimization of DBS therapy.1,10 Indeed, many of these patients could have had greater improvement from DBS several years earlier, further reduced their medications,18 and reduced the costs to the health care system.19,20 However, any discussion of cost saving must take into consideration the need for more intensive involvement of the movement disorders neurologist in the postoperative care of the patients and the need for appropriate adjustments in current remuneration schedules.

A systematic approach is mandatory in the management of DBS patients, including rigorous clinical assessment of benefit and adverse effects induced by the stimulation. If the initial programming uses this methodology applied by a neurologist experienced in programming and PD pharmacology and the electrode is well positioned, the subsequent management will be easy and will not require excessive time. Stimulation parameters and medication dosages do not significantly change over the years once the optimal benefit has been obtained.1,2 However, extensive knowledge of the interaction between anti-PD medications and DBS is mandatory, particularly their additive effects on dyskinesia11,13 and mood.11,21 Indeed in group 1, after optimization of the SID by means of parameter and anti-PD drug adjustments, patients showed significant dyskinesia reduction compared with baseline. However, in the short-term, accentuation of dyskinesia in 54% of patients required careful adjustments of stimulation parameters and medication, further emphasizing the importance of having an experienced neurologist directly managing such cases. Generally, the experienced clinician can manage SID by tapering levodopa and increasing the voltage, although in a small proportion of patients it can be a considerable challenge.

Monopolar stimulation is the first choice in STN DBS whereas bipolar stimulation may be useful in the temporary management of dyskinesia or in case of persistent adverse effects induced by low-voltage monopolar stimulation (which is usually insufficient for a satisfactory clinical benefit). In the latter case, however, the positioning of the electrode is generally suboptimal.

The improvement in rigidity during the programming and the occurrence of SID are good predictive signs of amelioration of the other parkinsonian signs (such as akinesia, freezing, and walking) and anti-PD drug dosage reduction. However, group 2 did not obtain any improvement following changes in settings and one could conclude that the previous programming was already optimized. Since there were no important baseline clinical differences between groups, it is possible that the positioning of the electrodes in the STN was less favorable in group 2 and possibly more so in group 3 compared with group 1,7,22

This was not a prospective study designed to evaluate 2 different approaches to postoperative care. Instead, we took advantage of the opportunity that presented to us in changing faculty coverage and expertise in our DBS program. Limitations of our study are that the UPDRS assessments were not done in a double-blinded fashion and without videotaped protocol. However, we believe that several factors support the validity and reliability of our unblinded observations. The patients were assessed by a neurologist they did not know and were not prepared for, or they did not expect to have any important changes in their parkinsonism with this re-evaluation of their DBS response. Similarly, the neurologist did not know the patients beforehand and did not expect to induce significant improvement or worsening in their parkinsonism. Additionally, the majority of the patients were followed up for more than 1 year after reprogramming and assessed several times.

Our experience emphasizes the importance of the hands-on involvement of a movement disorders neurologist familiar with DBS programming and postoperative drug adjustments in the direct management of DBS cases, at least in the first few months after surgery when stimulation and medication are not yet optimized.

Accepted for Publication: April 29, 2006.
Published Online: July 10, 2006 (doi:10.1001/archneur.63.9).
Correspondence: Elena Moro, MD, PhD, Movement Disorders Center, Toronto Western Hospital, 399 Bathurst
Author Contributions: Study concept and design: Moro, Lozano, and Lang. Acquisition of data: Moro, Poon, Lozano, and Saint-Cyr. Analysis and interpretation of data: Moro, Lozano, and Lang. Drafting of the manuscript: Moro, Poon, Lozano, and Saint-Cyr. Critical revision of the manuscript for important intellectual content: Lozano and Lang. Statistical analysis: Moro. Administrative, technical, and material support: Moro, Poon, Lozano, and Lang. Study supervision: Lozano and Lang.

Financial Disclosure: Dr Moro has occasionally received honoraria from Medtronic for lecturing at meetings. Dr Lozano has received honoraria from Medtronic for consulting services. Dr Lang has received research support from Medtronic. Ms Poon and Dr Saint-Cyr have reported no conflicts of interest.

Funding/Support: This study was partially funded through a Center of Excellence grant from the National Parkinson Foundation and a grant from Medtronic in support of fellow and nurse salaries.

Acknowledgment: We thank Drs Paul Krack and Janis M. Miyasaki for their comments. Dr Moro had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

REFERENCES
