Deep brain stimulation has become an important therapeutic option for various medically refractory movement disorders like tremor, PD, and dystonia. The modality is currently being tested in the treatment of a number of additional neurological and psychiatric diseases like epilepsy, obsessive compulsive disorders, and depression.

The stimulation systems provided by Medtronic are by far the most frequently applied systems worldwide. They consist of quadripolar electrodes (models 3389 and 3387) implanted into the intracerebral target area by stereotactic techniques. The electrodes are connected to extensions (model 7482 or 7495), which are led subcutaneously to the IPG. Single-channel IPGs (Itrel II model 7424 and Soletra model 7426) and a dual-channel IPG (Kinetta model 7428) are available. All these IPGs have an internal battery that is not rechargeable and requires replacement of the IPG when the battery fails. The longevity of batteries depends on the energy consumption required for stimulation in the individual patient and can vary markedly. The IPG is programmed telemetrically, that is, by the help of radiofrequency signals with a programmer (N’Vision Physician Programmer, Medtronic). Individual programming of the IPG consists of a careful workup to determine the most efficacious contact(s) of the quadripolar stimulation lead. A new short circuit of active with previously inactive contacts of the quadripolar stimulation lead resulted in a worsening of symptoms in 4% of replacements.

Stability of symptom control after replacement of impulse generators for deep brain stimulation

Clinical article

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Object. Impulse generators (IPGs) for deep brain stimulation (DBS) need to be replaced when their internal batteries fail or when technical problems occur. New IPGs are routinely programmed with the previous stimulation parameters. In this study, the authors evaluate the stability of symptom control after such IPG replacements.

Methods. The authors retrospectively analyzed the outcome of 56 IPG replacements in 42 patients with various movement disorders treated using DBS.

Results. Stable symptom control was found in 65% of single-channel IPG replacements and 53% of dual-channel IPG replacements. Worsening of symptoms resulted primarily from changes in stimulation effects requiring reprogramming of stimulation parameters (17% of dual-channel IPG and 25% of single-channel IPG). In 14% of dual-channel IPG replacements, instability resulted from erroneous extension adjustment with change in laterality. A new short circuit of active with previously inactive contacts of the quadripolar stimulation lead resulted in a worsening of symptoms in 4% of replacements.

Conclusions. Replacement of the IPG requires careful follow-up of patients with DBS to ensure stable symptom control. (DOI: 10.3171/2009.1.JNS081352)

Key Words • dystonia • deep brain stimulation • impulse generator • tremor

Abbreviations used in this paper: DBS = deep brain stimulation; IPG = impulse generator; PD = Parkinson disease.
Impulse generator replacement in deep brain stimulation
cline in the course of time but remains stable until complete depletion of the battery. Hence, optimized stimulation parameters need not be adjusted at the end period of the IPG’s battery life.

After replacement, the new IPG is routinely programmed with the previous stimulation parameters because the intracerebral position of an electrode is not changed. In this study, we evaluate the stability of symptom control after IPG replacement in patients with DBS for various movement disorders.

Methods

Patients undergoing DBS are regularly followed up in our outpatient clinic. Control visits are offered at intervals of 3 months to check neurostimulators for regular function including testing of impedance and current flow at the individual parameter setting and status of the battery. At first contact or for the initial programming of IPGs after implantation of DBS systems, a complete check of all electrode leads and contacts with unipolar and bipolar settings at 30 Hz, 210 μsec, and 1.5 V is performed. Irregularities like short circuits or lead fractures/disconnections are documented.

When the battery becomes low, it is recommended that patients have the IPG replaced. In this study, we recommended that IPG replacement be performed at the neurosurgical department where the system was initially implanted. In some cases, patients chose to have it replaced at other neurosurgical departments with better accessibility. In all cases, the neurosurgeon was provided with the last stimulation parameters to program the new IPG, particularly in those cases in which the battery was completely depleted and the parameters could no longer be checked telemetrically. At the first control visit after IPG replacement, patients were carefully evaluated for changes in clinical efficacy as well as possible technical problems.

Results

Between March 2004 and September 2008, replacements of 56 IPGs were necessary in 42 patients who were undergoing DBS for movement disorders. In all but 1 patient, the replacements included the first replacement after initializing DBS therapy. One patient had undergone a previous replacement before starting our study, and 11 patients had > 1 IPG replacement during the above time period. Demographic data on the IPG replacements are given in Table 1. The replacements took place at 8 different neurosurgical departments, 5 of them at university hospitals and 3 at nonuniversity hospitals. Apart from 1 patient with a postsurgical hematoma, no other adverse event related to the surgical procedure itself was observed. In this case acetylsalicylic acid was continued at the time of IPG replacement due to a recent myocardial infarction.

Of 36 dual-channel IPG replacements, 30 (83%) took place as regular replacements before complete battery failure. One was performed together with the surgical revision of 1 electrode, which induced oculomotor symptoms at low thresholds and suboptimal symptom control in a patient with PD and bilateral subthalamic stimulation. Five dual-channel IPG replacements (14%) followed the sudden recurrence of symptoms due to a complete battery failure in 3 cases and a “power on reset” in 2 cases. The latter has been described in IPGs from a certain production series with hardware problems.1

Of 20 single-channel IPG replacements only 7 (35%) took place as regular replacements before complete battery failure occurred, whereas in the remaining 13 cases (65%) a complete battery failure with recurrence of symptoms had occurred.

The IPG replacements in 53% of dual-channel and 65% of single-channel devices were uncomplicated, that is, no change in clinical efficacy was observed after reprogramming the previous stimulation parameters. In 47% of dual-channel and 35% of single-channel IPG replacements, a worsening of symptoms required trouble-shooting and reprogramming of the IPG. The risk of reduced symptom control after IPG replacement was only slightly higher if complete IPG failure had already occurred (9 [47%] of 19) than in cases of routine IPG replacement before battery failure (15 [41%] of 37). In all these cases, the patient noticed worsening of symptom control almost immediately during the first day(s) of stimulation with the new IPG. In most cases it was initially believed by the neurosurgeon and the patient to be only transient so that patients were discharged from the neurosurgical department without further trouble-shooting.

The reasons for a need to reprogram the IPG are depicted in Table 2. In the majority of these cases (17% of dual-channel IPG replacements and 25% of single-channel IPG replacements) stimulation resulted in either reduced clinical efficacy or intolerable side effects. Although the number of cases is limited, the risk of reduced symptom

<table>
<thead>
<tr>
<th>Disease</th>
<th>No. of Patients</th>
<th>No. of Dual-Channel IPG Replacements</th>
<th>Mean Time to Exchange of Dual-Channel IPG (mos)</th>
<th>No. of Single-Channel IPG Replacements</th>
<th>Mean Time to Exchange of Single-Channel IPG (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD</td>
<td>28</td>
<td>23</td>
<td>48.8 ± 13.8</td>
<td>13</td>
<td>49.2 ± 17.4</td>
</tr>
<tr>
<td>essential tremor</td>
<td>4</td>
<td>2</td>
<td>53.5 ± 6.5</td>
<td>3</td>
<td>59.3 ± 13.7</td>
</tr>
<tr>
<td>dystonia</td>
<td>7</td>
<td>7</td>
<td>38.0 ± 15.6</td>
<td>4</td>
<td>69.3 ± 26.7</td>
</tr>
<tr>
<td>multiple sclerosis</td>
<td>2</td>
<td>2</td>
<td>31.0 ± 2.0</td>
<td>4</td>
<td>69.3 ± 26.7</td>
</tr>
<tr>
<td>cerebellar tremor</td>
<td>1</td>
<td>2</td>
<td>36.5 ± 3.5</td>
<td>20</td>
<td>54.8 ± 20.8</td>
</tr>
<tr>
<td>total</td>
<td>42</td>
<td>36</td>
<td>45.3 ± 14.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
control after IPG replacement appeared to be unrelated to the disease: 6 of these cases occurred in patients with PD, 2 in patients with essential tremor, and 3 in patients with dystonia. In 4 of these 11 patients, satisfactory symptom control was achieved after 1 or 2 appointments as outpatients, usually with a testing and observation period of several hours. The other 7 patients were hospitalized for reprogramming. With reference to the 16 electrodes involved, 8 required only a change in the stimulation amplitude (in 6 patients) or amplitude and pulse width (in 2) to achieve satisfactory symptom control. Interestingly, these changes were all reductions in the product of amplitude and pulse width by a mean of 23 ± 8%. In the other 8 electrodes, active stimulation contacts were changed to regain satisfactory symptom control.

In 11% of all IPG replacements, reduced symptom control resulted from deviations from the previous parameter setting, partly because neurosurgeons decided to start stimulation with the new IPG at lower amplitudes, and partly because the pulse width was accidentally changed. Returning to the previous setting cleared the worsening in these cases.

In 2 of 56 replacements, a newly discovered short circuit of the active electrode contact with a neighboring contact was found to induce a change in clinical efficacy. In both cases reprogramming resulted in satisfactory symptom control without the necessity of further surgical revision.

An important source of change in symptom control in this study was an erroneous channel adjustment of dual-channel IPGs with consequent change in laterality, so that contacts and stimulation parameters of the previously left-hemispheric stimulation electrode were used for the right-hemispheric electrode after replacement and vice versa. This change was not discovered before these patients presented with a worsening of symptoms in our outpatient clinic. In these cases reprogramming stimulation parameters of previously K1 for K2 and vice versa resulted in a rapid improvement and satisfactory symptom control. Apart from the 5 of 36 dual-channel replacements in Table 2, we found another such erroneous channel adjustment with unchanged clinical efficacy due to a symmetric parameter setting of both electrodes.

### Discussion

Our study demonstrates an unexpected high rate of IPG replacements followed by reduced efficacy of DBS and the need for reprogramming. It stresses the importance of a careful follow-up after IPG replacements and close interdisciplinary communication between neurologist and neurosurgeon.

Among the sources of reduced symptom control are those that can be avoided by careful testing of the new IPG immediately after the replacement. One is the technical function and, in the case of dual channel IPGs, the verification of proper laterality of the electrode leads. The technical check should include careful impedance measurements to exclude short circuits and disconnections that will be of significance if active contacts are involved. An erroneous connection of the extensions to the channels of dual-channel IPGs is easily recognizable if the channels are tested at appropriate amplitudes for lateralized side effects.

If IPGs are tested in this way and technical problems can be excluded, stimulation parameters should be set at the previous setting even if they appear relatively high. In 11% of the replacements the worsening of symptoms was merely due to an unprovoked reduction of the stimulation amplitude and was improved after getting back to the previous setting.

Only in the case of new stimulation-induced side effects should the stimulation amplitude be reduced appropriately and a further programming recommended by the neurologist. With these measures, the hospital discharge of patients with reduced symptom control after IPG replacement would have been decreased from 40 to 20% in this study.

However, there are still 20% of IPG replacements with no obvious technical problem, but still there is a change in efficacy. The most likely reason for such changes in stimulation effects is an altered current flow at the active

### Table 2: Symptom control after IPG replacement

<table>
<thead>
<tr>
<th>Type of IPG Replacement</th>
<th>Unchanged</th>
<th>Reduced (side effects or reduced symptom control)</th>
<th>Reduced (previous stimulation parameters not continued)</th>
<th>Reduced (erroneous lead adjustment w/ change in laterality)</th>
<th>Reduced (short circuit after replacement)</th>
</tr>
</thead>
<tbody>
<tr>
<td>dual-channel regular</td>
<td>19</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>after failure</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>total</td>
<td>19 (53)</td>
<td>6 (17)</td>
<td>5 (14)</td>
<td>5 (14)</td>
<td>1 (3)</td>
</tr>
<tr>
<td>single-channel regular</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>NA</td>
<td>0</td>
</tr>
<tr>
<td>after failure</td>
<td>10</td>
<td>2</td>
<td>1</td>
<td>NA</td>
<td>1</td>
</tr>
<tr>
<td>total</td>
<td>13 (65)</td>
<td>5 (25)</td>
<td>1 (5)</td>
<td>NA</td>
<td>1 (5)</td>
</tr>
<tr>
<td>total</td>
<td>32 (57)</td>
<td>11 (20)</td>
<td>6 (11)</td>
<td>NA</td>
<td>2 (4)</td>
</tr>
</tbody>
</table>

* NA = not applicable.
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electrode contact with a change of the volume of tissue activated. The current flow can change if the electrode impedance changes after the IPG replacement. Both parameters—current flow and impedance—are poorly controlled by the current IPGs, particularly the dual-channel IPG Kinera. It will be of interest for future developments of DBS systems whether better control of all electrical parameters including current flow and impedance will improve patient safety at the time of IPG replacement.

Our study focused on the issue of symptom control after IPG replacement and not battery longevity. Battery longevity in this study is underestimated because of a bias toward patients with earlier replacements. The IPG replacement should always be attempted before complete battery failure occurs to avoid sudden recurrence of symptoms, which can be particularly hazardous in patients with PD. The high rate of battery failures before IPG replacement observed in this study is partly due to irregular follow-up visits in patients with stable symptom control and often long distances between home and outpatient clinic. Another issue is that the decrease in battery capacity is not linear but shows an often rapid decline at the end. Almost all patients in this study were trained to use a hand-held therapy controller (Access Therapy Controller for Kinera model 7428, Access Review Therapy Controller for Itrel II model 7424, and Soletra model 7426) to check the IPG battery. Nevertheless, particularly in the case of single-channel IPGs, this did not prevent the high rate of sudden failures.

To improve patient safety, a more rigorous follow-up, particularly at the expected end of an IPG’s life, is recommended. Furthermore, technical improvements like rechargeability, better precision in measuring the battery capacity, and better control of battery function by patients themselves will be helpful.

Conclusions

The IPG replacements in our study resulted in reduced clinical efficacy of DBS in 40% of cases. Approximately 20% could have been avoided by a careful technical check of the system including impedances of active electrode contacts, proper laterality of extensions at dual channel IPGs, and programming of stimulation parameters identical to the previous setting. These measures take only a couple of minutes and can avoid undue time intervals with reduced symptom control between discharge from neurosurgical departments and trouble-shooting/reprogramming in the neurological departments.

In a remaining group of ~ 20%, changes in stimulation efficacy were most likely due to changes in the impedance and, consequently, the volume of tissue activated. In these cases, a more thorough testing for optimal stimulation parameters would be necessary, including possible changes in stimulation contacts.

Disclosure

Niels Allert has received honoraria from Medtronic for lecturing and consulting services. The authors report no other conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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