Movement Disorders
Vol. 21, Suppl. 14, 2006, pp. S219–S237
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Deep Brain Stimulation: Postoperative Issues

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Abstract: Numerous factors need to be taken into account when managing a patient with Parkinson's disease (PD) after deep brain stimulation (DBS). Questions such as when to begin programming, how to conduct a programming screen, how to assess the effects of programming, and how to titrate stimulation and medication for each of the targeted sites need to be addressed. Follow-up care should be determined, including patient adjustments of stimulation, timing of follow-up visits and telephone contact with the patient, and stimulation and medication conditions during the follow-up assessments. A management plan for problems that can arise after DBS such as weight gain, dyskinesia, axial symptoms, speech dysfunction, muscle contractions, paresthesia, eyelid, ocular and visual disturbances, and behavioral and cognitive problems should be developed. Long-term complications such as infection or erosion, loss of effect, intermittent stimulation, tolerance, and pain or discomfort can develop and need to be managed. Other factors that need consideration are social and job-related factors, development of dementia, general medical issues, and lifestyle changes. This report from the Consensus on Deep Brain Stimulation for Parkinson's Disease, a project commissioned by the Congress of Neurological Surgeons and the Movement Disorder Society, outlines answers to a series of questions developed to address all aspects of DBS postoperative management and decision-making with a systematic overview of the literature (until mid-2004) and by the expert opinion of the authors. The report has been endorsed by the Scientific Issues Committee of the Movement Disorder Society and the American Society of Stereotactic and Functional Neurosurgery. © 2006 Movement Disorder Society

Key words: deep brain stimulation; Parkinson's disease; postoperative management

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Published online in Wiley InterScience (www.interscience.wiley.com). DOI: 10.1002/mds.20957

The management of a Parkinson's disease (PD) patient after deep brain stimulation (DBS) is complex and multifaceted. The questions developed and addressed in this article attempt to consider all factors that might be involved in the early management, follow-up care, and long-term follow-up of PD patients undergoing DBS. In addition, the authors attempt to define the best practices to manage commonly encountered problems and long-term complications of DBS.

METHODS

Search Strategy

A search of the literature from 1965 to May 2004 using Medline was completed and resulted in a total of 574 articles related to DBS. The multiple searches used the following key words: deep brain stimulation, Parkinson's disease, neurostimulation, thalamic stimulation, pallidal stimulation, and subthalamic stimulation. These searches were combined with those of neuropsychology, neuropsychiatry, microelectrode recording, surgical technique, complications, neuroimaging, and targeting. Articles were classified as preoperative and postoperative issues, intraoperative issues, and neuropsychological issues. A PDF file was created for each of the articles that could be obtained. This resulted in a CD containing copies of 522 of the articles, which was distributed to each member of the writing committee. During the writing process, the authors added an additional 87 articles in order to provide background references for various issues, techniques, and measurement scales discussed in the articles or to include articles published after May 2004 that were determined to be critical in completely addressing a particular issue.

Process of Generating Clinical Recommendations

The writing committee was composed of neurologists, neurosurgeons, neuropsychologists, neuropsychiatrists, and researchers with experience and expertise in DBS for Parkinson's disease. The committee was composed of experts from multiple centers in North America and Europe. The natural division of important issues related to DBS was applied and participants were assigned based on their interests and expertise to separate preoperative, surgical/intraoperative, or postoperative issues committees. The steering committee formulated a series of questions related to all aspects of these three areas, which were then reviewed and refined by the committee members. Committee members were then assigned specific questions to answer utilizing the literature compiled as described above. The responses were then reviewed by the steering committee at a separate meeting and it was decided to organize responses by first reviewing the "available data", followed by sections providing "conclusions", "pragmatic recommendations," and "points to be addressed" by future research. It is generally agreed that there is insufficient evidence in the currently available literature to formulate definitive recommendations and conclusions for most of the questions and issues covered in the articles. Therefore, subsequently, the entire group discussed and revised the recommendations during a 3-day meeting. At this meeting, the whole group (members of all three working committees) provided feedback and agreed on the final conclusions to be drawn from available data as well as the formulation of pragmatic recommendations recognizing that many of the questions could not be answered on the basis of currently available studies. The steering committee members then reformulated each section and redistributed the document to their entire working committee for final approval. Whenever there were opposing or conflicting opinions, further discussion followed and consensus was reached or outstanding disagreement was specifically addressed in the paper.

1. GENERAL MANAGEMENT ASPECTS

1.1. General Aspects of Early Postoperative Management

1.1.1. Are There General Treatment Recommendations for the Early Postoperative Period?

Available Data

There are no formal studies on immediate postoperative patient management. General health recommendations include the systemic administration of antibiotics intraoperatively and up to the first postoperative week and standard monitoring such as ECG, blood pressure, and vital signs.[1] Some patients have postoperative transitory confusion or agitation,[2–8] particularly if mild preoperative cognitive impairment was present,[2] for which atypical antipsychotics have been used.[9] In many centers,

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a postoperative MRI or CT scan is done before stimulator implantation to verify electrode position and to rule out surgical complications.[3,10–13] There is limited information on the postoperative reintroduction of antiparkinsonian medication.[14]

Conclusions

Standard postoperative care is sufficient providing the surgery is uncomplicated; postoperative management in the intensive care unit is not a necessity.

Pragmatic Recommendations

Postoperative antiparkinsonian treatment should be restarted as soon as possible to relieve discomfort and limit the risk of acute dopaminergic withdrawal and malignant hyperthermia. The postoperative levodopa equivalent dose (LED) should be the same as the presurgical dose and composed primarily of levodopa to facilitate simple titration during programming initiation.

Confusional states generally last a few days and can be easily managed with sedatives such as benzodiazepines and atypical antipsychotics such as clozapine or quetiapine. Neuroimaging is highly recommended immediately following surgery to confirm electrode location and to rule out intracranial adverse events.

Points To Be Addressed

The relationship between postoperative confusional states and patient age, length of surgery, number of electrode passes, microrecording, and stimulation should be investigated to determine predictive risk factors and to allow optimal surgical planning.

1.1.2. When Should DBS Programming Be Started?

Available Data

With the initial introduction of DBS surgery, external electrode interrogation was commonly performed 7 to 10 days postoperatively to determine clinical response. The stimulator was then implanted if efficacy was demonstrated. External stimulation has since been largely abandoned.[15]

Prior to the initiation of stimulation, surgical tracts may cause microlesioning effects, which may result in transitory symptom improvement. To get an accurate representation of symptoms, the majority of centers prefer to delay programming until parkinsonian signs return,[1,16] tissue heals,[17] and microlesioning effects wear off.[18] Practices among centers vary and the duration of delay and the extent of hospitalization vary. For instance, stimulation assessment 1 to 2 weeks after surgery[10,16,19,20] or hospital discharge after 1 week with stimulation initiation 3 to 4 weeks after surgery[1,18,21,22] has been reported. Hospitalization during the stimulation and medication titration period[17] has also been described. Hospital discharge 4 to 7 days after surgery has been reported; prior to discharge, stimulation was initiated at preset parameters and the patient was provided with guidelines for stimulation titration and medication reduction with access to a nurse specialist and a follow-up visit 6 weeks after surgery.[23]

Conclusions

Management approaches are variable and do not appear to affect patient outcome. The decision regarding the timing of postoperative programming practices is dependent on available resources and convenience at individual centers.

Pragmatic Recommendations

The time course for the resolution of microlesioning effects is an important issue for stimulation initiation but is not well established. As a result, the patient should be made aware of the potential for fluctuations in stimulation effects in the first several months. Waiting for the resolution of microlesioning effects should be balanced with the need to relieve patient symptoms.

1.1.3. How to Proceed in Assessing Stimulation Effects and Optimizing Stimulation Parameters?

Available Data

The assessment of clinical features when optimizing stimulation parameters is not generally reported in papers on DBS efficacy. A few papers address the criteria to assess stimulation effects,[17,24,25] which include wrist rigidity, tremor (if present), bradykinesia, and gait instability.

Conclusions

There are no formal studies assessing stimulation effects. Assessment strategies are not uniform, although some items are common to several centers as discussed below.

Pragmatic Recommendations

Stimulation effects are generally assessed in two separate stages. In the first stage, a preliminary interrogation using a monopolar configuration is performed to determine efficacy and side effects of each contact. The contact with the greatest efficacy and fewest side effects is selected. Stimulation intensity is slowly increased using commonly accepted pulse widths and frequencies. In the second stage, a clinical assessment is performed, during

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which antiparkinsonian medications and stimulation parameters are adjusted as necessary.

After selection of the optimal contact, symptom reduction should be verified. Rigidity, a sign time-locked with stimulation,[17] is the most useful acute parameter to assess given its consistency, reproducibility, and independence from patient cooperation. A provocation maneuver such as elevating the contralateral arm can be performed should baseline rigidity be insufficient. Tremor, if present, can be used as a secondary target symptom. Bradykinesia is less reliable given its potential interaction with patient motivation and fatigue. Response of this feature to stimulation occurs with variable latency and small changes can be difficult to detect and quantify.

The aims of long-term stimulation are to control symptoms and to improve features such as *on–off* phenomena, dyskinesias, and sleep disturbances. Levodopa and subthalamic nucleus (STN) stimulation have similar and additive effects, but the latter has the advantage of continuous and nonfluctuating symptom control. The optimal goal should be the gradual reduction of drug doses with optimized stimulation.

If satisfactory results are not achieved or significant discrepancies exist between the effects of levodopa and bilateral STN DBS, electrode positioning should be checked and technical problems should be ruled out.[26]

1.1.4. Why Is the Screening Session Important and How Should It Be Approached?

Available Data and Conclusions

There are no formal studies on the initiation of stimulation. The available data are based on expert opinion.[17,24–26] Stimulation effects are assessed in two stages as discussed above. The first determines the effects and side effects of each contact using a monopolar configuration. One contact is then selected and the stimulation strength is slowly increased using commonly accepted pulse widths and frequency parameters.

Pragmatic Recommendations

The first priority in programming is to determine the optimal electrode contact by interrogating each contact for efficacy and side effects. The optimal contact ideally has the widest therapeutic window defined as the amplitude interval between the lowest voltage required to elicit clinical effects and the lowest voltage eliciting unwanted side effects. The type of side effect elicited has differing implications. For instance, the elicitation of dyskinesia predicts a favorable long-term outcome indicative of a well-placed electrode, whereas other side effects may be due to the current spread to surrounding structures outside of the STN. These other side effects include contralateral gaze deviation, hemibody muscle contraction, dysesthesias, dysconjugate ocular deviation, postural instability, gait ataxia, sweating, and mydriasis. The selection of the appropriate contact requires differentiating between transitory and persistent side effects.

Hierarchy of the Four Contacts

The determination of which contact to utilize for chronic stimulation requires a careful bilateral assessment of the efficacy of each contact on parkinsonian symptoms while taking into account the therapeutic window. While a range of symptoms may be assessed, the target symptom may differ depending on the nuclei selected for stimulation: for example, tremor for ventral intermediate nucleus (Vim); rigidity, tremor, and bradykinesia for STN; dyskinesias, rigidity, and bradykinesia for globus pallidus internus (GPi).[17] The assessment of two consecutive contacts requires a sufficient duration of stimulation discontinuation to allow a return of symptoms to baseline.

Polarity

Monopolar stimulation is generally selected for current delivery; it is considered the most effective option and the vast majority of patients are successfully programmed with a single contact.[27–29] Bipolar stimulation may be preferred if a narrower current to reduce the elicitation of side effects is desired.[23] Double monopolar stimulation may be warranted if a single electrode is insufficient to produce an optimal effect.[17]

Amplitude, Pulse Width, and Frequency

During the initial assessment, it is suggested that the pulse and frequency be held constant while the amplitude is assessed in increasing increments of 0.5 V from 0 to 6 V. A pulse width of 60 milliseconds is usually selected as the best compromise between clinical benefit and side effects.[30] A frequency of 130 Hz is generally used during the initial assessment while a higher frequency (185 Hz) may be used for tremor control in Vim[16,31,32] and in STN stimulation.[26]

1.1.5. How Should Stimulation and Medications Be Titrated for the Three Different Targets?

Available Data

STN stimulation initiation is more complex than that of GPi or Vim due to the need to balance medication reduction with increasing stimulation intensity.[14] Motor complications such as dyskinesias and hypokinetic fluctuations as well as psychiatric symptoms such as depres-

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sion, apathy, and hypomania may develop.[17] In contrast, with GPi and Vim stimulation, intensity can be increased in larger and more rapid increments without altering medication. With GPi DBS, antiparkinsonian drugs are either unchanged,[28,33] slightly increased,[34] or slightly decreased.[35] With Vim DBS, antiparkinsonian drugs are either unchanged[20] or slightly reduced in a minority of patients.[31,36] There are no formal studies examining which type of antiparkinsonian medications may be more effective for patients following DBS.

Conclusions

In STN DBS, increasing stimulation parameters are usually accompanied by a reduction in medication dosages. In GPi DBS and Vim DBS, medication dosages usually do not change significantly.

Pragmatic Recommendations

The goal of medication titration is to achieve a balance between optimizing stimulation effects with concomitant medication reduction and minimizing side effects from excessive reduction of systemic dopaminergic stimulation. Levodopa or dopamine agonists can be used, and there are no established criteria regarding preference. Criteria are the continuous improvement of the mobility and absence of dyskinesias or other unwanted side effects.

1.2. General Aspects of Follow-Up

1.2.1. Should Patients Be Trained to Adjust Stimulation Parameters?

Available Data

With Vim DBS, patients are usually instructed to switch OFF the stimulators at night[20,37] to spare battery life and reduce the possibility of tolerance. This is not recommended for patients with stimulators in the STN or GPi.

The Access Review Therapy Controller (Medtronic, Minneapolis, MN) allows patients with a Kinetra implantable pulse generator (IPG) (Medtronic) to make minor adjustments of stimulation parameters (particularly of the current amplitude) within the limits set by the programmer, whereas the Itrel or Soletra IPGs (Medtronic) do not allow patient adjustment of stimulation. Therefore, patient training to adjust stimulators is device-dependent and is used by some[38] but not all centers.[4,39] So far, specific behavioral problems related to self-adaptation of stimulation have not been reported.

Conclusions

In patients with Kinetra IPGs, the Access Review Therapy Controller allows for limited patient adjustment of stimulation parameters, which can enhance symptom control and allow for nocturnal battery sparing.

Pragmatic Recommendations

Patients or caregivers must know how to turn the stimulator ON and OFF with either a handheld magnet or the Access Review Therapy Controller. Patients and caregivers should also be instructed on how to check the status of the stimulator with the Access Review Therapy Controller. If the controller is not available, a transistor radio set to an AM band can be used to determine if the stimulator is ON or OFF.

In patients with Kinetra IPGs, the Access Review Therapy Controller can allow the patient to switch between preset stimulation parameters. This option can be offered to highly cooperative patients or caregivers with proven reliability in recognizing clinical features as well as a clear understanding of stimulation manipulation.

1.2.2. What Are the Criteria for Scheduling Follow-Up Visits and Telephone Contacts After Surgery?

Available Data

There are no formal criteria for scheduling follow-up visits or telephone contacts after surgery. In most centers, patients are encouraged to contact the treatment team whenever necessary.[37,40] The number of visits tend to decrease after the first postoperative year, when stimulation parameters and medication titration has stabilized.[38] In contrast, some centers do schedule regular follow-up visits at prespecified intervals.[22,29,39]

Conclusions

The frequency of interim visits at each center depends on patients' needs and on the available resources (e.g., physicians, nurses, secretaries, call centers).

Pragmatic Recommendations

Regular follow-up visits are recommended and can be customized according to individual need. Patients require follow-up for as long as they have an implanted device. Device malfunctioning, battery depletion, and ongoing PD management should be monitored. Many centers have scheduled visits at 3 and 6 months and thereafter annually.

Points To Be Addressed

An algorithm for postoperative stimulation management and cost–effectiveness analyses are potential topics for further investigation.

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1.2.3. Should OFF Stimulation Assessments Be Routinely Done and, If So, How Long Should the Stimulators Be Turned OFF Prior to the Assessments?

Available Data

There are no formal studies addressing the necessity for OFF stimulation assessments. There is evidence that 75% of all symptoms usually return after 30 minutes of stopping stimulation. After 3 to 4 hours, deterioration of motor symptoms levels off at 90%.[41] Switching OFF the stimulator overnight in patients with STN stimulators is not well tolerated.[42]

Conclusions

Turning OFF the stimulation for 30 minutes for OFF stimulation assessment is generally sufficient for most investigations.

Pragmatic Recommendations

Motor evaluations in the OFF stimulation condition are not necessary for routine evaluations. Certain circumstances (e.g., complaints of side effects such as dystonia, dyskinesias, and postural instability that may be related either to stimulation or to poor symptom control) may require evaluation of the patient OFF stimulation. A suprathreshold levodopa challenge OFF stimulation predicts the maximal achievable benefit and reveals levodopa-resistant motor problems. Motor signs should improve to the same degree with both levodopa and stimulation. Reasons for any discrepancies should be explored (e.g., improper lead location, choice of stimulation parameters, and the behavioral state of the patient). Routine evaluations in the OFF stimulation condition can only be justified in the context of research protocols.

2. MANAGEMENT OF COMMON POSTOPERATIVE PROBLEMS AND OPTIMIZATION IN THE POSTOPERATIVE PERIOD

2.1. Management of Common Postoperative Problems

2.1.1. Management of Weight Gain

Available Data

Weight gain has been reported to occur after surgical therapy for PD. An incidence of 6% to 100% has been reported for STN DBS[6,29,40,43–46] and of 26% to 96% for GPi DBS.[47] There are no reports on weight gain following Vim DBS. The extent of weight gain may be higher in STN DBS[48,49] than in GPi DBS.[47] The etiology of weight gain is unclear and has been postulated to be due to an effect of DBS on hypothalamic centers, changes in dopaminergic reduction, and a decrease in energy expenditure due to amelioration of dyskinesias.[48,49]

Conclusions

Weight gain is a common phenomenon following DBS for PD.

Pragmatic Recommendations

Candidates for a surgical procedure may be given preoperative nutritional counseling to prevent excessive weight gain. Uncontrolled long-term observations suggest that the weight of most patients normalizes after 1 or 2 years.

2.1.2. Management of Dyskinesias

Available Data

STN DBS can trigger initial choreiform, ballistic, or dystonic movements that may resemble levodopa-induced dyskinesias.[50,51] The appearance of dyskinesias assists in the confirmation of appropriate electrode location in the STN.[52–54] Dyskinesias usually disappear with chronic STN stimulation[50] and are markedly improved in the long term.[9,22,29,55,56] Given the similar effect of STN stimulation and medication, postoperative reduction of dopaminergic medication is regarded as an important goal in many studies.[9,56–58]

GPi DBS has not been reported to induce dyskinesias and there are no reports of an amplification effect of medication and stimulation. On the contrary, GPi DBS is believed to exert a direct antidyskinetic effect[34,59] with greater antidyskinetic efficacy suggested in posterior–ventrally located contacts than dorsal contacts.[27] Vim DBS has been reported to have minimal[60–62] or no impact on levodopa-induced dyskinesias.[20]

Conclusions

STN DBS may aggravate levodopa-induced dyskinesias in the immediate postoperative period. There are no formal studies on the adjustment of stimulation parameters and dopaminergic medications to prevent or manage postoperative dyskinesias in STN DBS. Management is based on single-case reports and expert opinion. GPi DBS exerts a direct antidyskinetic effect, whereas Vim DBS has little influence on levodopa-induced dyskinesias in PD.

Pragmatic Recommendations

In the immediate postoperative period following STN DBS, simplification of the medication regimen is advisable given the complexity of the interactions between stimulation and medications. Monotherapy with immediate release

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levodopa rather than dopamine agonists or controlled release formulations can facilitate ease of management. Levodopa has a short half-life and is easier to titrate to manage *off* phase symptoms and dyskinesias. During the postoperative hospitalization period, the patient is reassessed and optimal levodopa dosage is decided based on the degree of Parkinsonism and the presence or absence of dyskinesias. The contact that produces dyskinesias is generally the most effective contact for the relief of akinetic–rigid symptoms. Since stimulation-induced dyskinesias are worsened by levodopa, initial programming should be performed before the morning medication dose. Low dosages of levodopa can be resumed if, after a few hours following programming, no dyskinesias appear. The optimal contact is initially set to an amplitude of 0.5 V, a frequency of 130 Hz, and a pulse width of 60 milliseconds. The amplitude is increased by 0.2 to 0.5 V at 1- to 2-day intervals titrated to adequate symptom relief or slight dyskinesias. Initial programming generally lasts 1 to 3 weeks. In the subsequent weeks, a reduction of stimulation effect may occur due to habituation. A slight increase in the amplitude or an increase in dopaminergic therapy can resolve this change in stimulation effect. Patients with severe preoperative dyskinesias from low levodopa dosages may experience severe hyperkinesias with stimulation initiation or small increases in stimulation. This can be avoided with a fairly rapid reduction in medication at the time of programming. Increasing stimulation by smaller increments (0.1 V) and waiting for longer intervals (several days) in between each change of amplitude may further allow for induced dyskinesias to subside. As such, several weeks of programming may be necessary for optimization. In rare cases, the therapeutic window between optimal symptom control and the dyskinesia threshold is sufficiently narrow that dyskinesias persist despite slow adjustment. Should this occur, either a more distally or proximally placed contact with a broader therapeutic window or a bipolar stimulation mode should be selected. Following the postoperative optimization of stimulation parameters, levodopa may be replaced with a longer-acting dopamine agonist.

In GPi DBS and Vim DBS, stimulation-induced dyskinesias usually do not occur. Therefore, stimulation parameters can be set to the optimal range without the need for balancing medication and stimulation.

2.1.3. Management of Axial Symptoms

Available Data

Postoperative deterioration of axial symptoms has been reported as a specific adverse effect of STN stimulation,[4,8,28,31,36,46,63–67] although the incidence appears to be low. This includes persistent impairments of postural stability,[4] a transient decline of balance,[28] and increased falls.[46] STN DBS generally results in the postoperative improvement of axial symptoms compared to both *on* and *off* medication preoperative baselines.[3,42,46,68–71] A less favorable outcome of STN DBS on axial symptoms compared to the preoperative *on* medication baseline was described in one study.[72] A greater magnitude of preoperative levodopa response and a lower preoperative severity of axial symptoms have been correlated with greater postoperative improvements of axial symptoms.[73] One study suggested that improvement of axial symptoms could be achieved by adjustment of medication and stimulation due to the observed synergistic effects.[69] Long-term deterioration of axial symptoms after STN DBS has been reported.[22,29,39] However, rather than a stimulation-related effect, this observed decline in postural stability, gait, and speech after 2 to 5 years has been attributed to the underlying disease progression and effects of nondopaminergic systems and pathways outside the nigrostriatal system.

Impairments in axial symptoms have been reported as specific adverse effects of GPi stimulation. These include a transient increase in freezing,[3,35] a transient impairment in balance,[28] falling,[46] and persistent gait akinesia.[74] On average, most studies report that axial symptoms are improved following GPi DBS compared to the preoperative state.[3,28,35,42,46,74–76] A long-term deterioration of axial symptoms including gait initiation failure[74] and loss of gait and postural stability[77,78] has been reported in several studies.

In Vim DBS, balance problems have been reported at 3% to 7.5%,[31,36,60,63–67,79] with greater frequency associated with bilateral stimulation.[31,60] The symptoms are reversible with stimulation parameter changes.[60,63]

Conclusions

Stimulation-induced deterioration of axial symptoms has been reported as a specific side effect following STN, GPi, and Vim DBS. The extent of postoperative axial symptom improvement may be estimated by the extent of the preoperative levodopa response. Assuming an adequate preoperative levodopa response, optimization of axial symptoms can be achieved by adjustments of medications and stimulation parameters. Observed declines of axial symptoms with chronic stimulation are likely a result of nondopaminergic lesions that cannot be reversed with medications or stimulation.

Pragmatic Recommendations

To manage the side effect of stimulation-induced axial deterioration, either the active contact or the stimulation parameters should be changed. For STN DBS, a more

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proximally or laterally located contact away from the prerubral field should be selected. If insufficient *off* period symptom improvement prevents a contact change, then a change to a bipolar electrode configuration should be considered. For Vim DBS, a more proximal contact to limit current spread to the cerebellothalamic fiber tracts may be selected to minimize deterioration of axial symptoms. If tremor control is insufficient, a bipolar configuration should be attempted.

If the postoperative improvement of axial symptoms is suboptimal despite a favorable preoperative levodopa response, each contact should be reassessed for efficacy in the relief of *off* period symptoms. Selection of a more effective contact may be indicated. If adjustment of stimulation parameters is not adequate for symptom control, dopaminergic medication may be slowly increased while carefully assessing for the development of dyskinesia.

Worsening axial symptoms unresponsive to the above medication and stimulation manipulations are likely due to increasing nondopaminergic lesions. Therapeutic approaches are thus limited and supportive care should be provided.

2.1.4. Management of Speech Dysfunction

Available Data

Hypophonia or dysarthria following initiation of STN DBS and Vim DBS has been described as a common problem. In STN DBS, the prevalence of speech dysfunction as a stimulation-induced side effect ranges from 4% to 17%.[7,22,29,39,40,55,80–82] Dysarthria is the most common adverse effect of Vim DBS with a prevalence ranging from 5% to 25%.[36,60,63–67,79] Dysarthria is more frequent in bilateral Vim DBS or when DBS is applied contralateral to a previous thalamotomy.[60,64] In contrast, speech difficulties following GPi DBS have been infrequently reported.[3,46,83]

The etiology of dysarthria after DBS may be multifactorial, but the proximity of the STN and Vim to the internal capsule and current spread into the internal capsule is considered the most important factor.[17,84,85] It has been shown that STN DBS does not in itself produce speech dysfunction.[86] Even in those patients who had preoperative levodopa-responsive speech disturbances and had an initial postoperative benefit from STN stimulation, worsening of the speech abnormality and resistance to treatment over time have been shown in several studies.[22,29] This has been attributed to the increase of dysfunction of nondopaminergic pathways with progression of the disease. In those patients with preoperative levodopa-resistant speech difficulties, STN DBS is not effective in alleviating the dysfunction.[4,29,69] There is no study available specifically focusing on the management of speech dysfunction following DBS.

Conclusions

Speech dysfunction is a common stimulation-induced adverse effect of STN DBS and Vim DBS. It rarely occurs in GPi DBS. Deterioration of speech function in the long-term has been reported in several studies as a result of the progression of the disease.

Pragmatic Recommendations

If speech dysfunction occurs after initiation of STN DBS or Vim DBS, speech and oral control should be evaluated without medications in the ON and OFF stimulation conditions. If speech worsens when stimulation is turned ON, this indicates a stimulation-induced speech disturbance. These symptoms may be relieved by reducing the amplitude of stimulation or by focusing the electrical field with a bipolar configuration. As there is no habituation to this side effect, some patients accept a mild disturbance in speech in exchange for benefit to the other symptoms of PD.[17] If there is a relatively low threshold for the induction of pseudobulbar dysarthria and a narrow therapeutic window despite optimization, it suggests that the electrode may be placed too far laterally, thereby recruiting corticobulbar fibers and repositioning of the electrode should be considered. If the speech difficulty is not affected by altering stimulation, a levodopa challenge with the stimulator turned ON may be diagnostic. If giving a suprathreshold dose of levodopa results in an improvement in the speech deficit, it suggests that stimulation is suboptimal with an excess postoperative reduction in medications. In those patients with preoperative levodopa-resistant speech difficulties or declining speech function caused by progression of the disease, DBS is not effective in alleviating the dysfunction.[4,29,69] In those patients, supportive measures such as the Lee Silverman Voice Treatment[87,88] should be initiated.

2.1.5. Management of Muscle Contractions

Available Data

Tonic muscle contraction has been reported to be a common phenomenon in patients when increasing the stimulation parameters to suprathreshold values.[24] Based on electrophysiological findings in patients with STN DBS, it is probably also a result of current spread into the internal capsule.[84] Contractions are often first visible in the contralateral face or hand and may progress to the proximal and lower limb with higher amplitudes.[17] The

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prevalence of muscle contractions as a chronic adverse effect in the different nuclei is low and only single cases have been reported.[22,39] In one case, revision of the electrode was necessary and led to resolution.[39]

Conclusions

Muscle contractions may occur in all targets when increasing stimulation amplitude to suprathreshold values. Persistence of contractions chronically in DBS patients is infrequent.

Pragmatic Recommendations

As corticospinal fibers form the lateral border of the STN, diffusion of the current laterally may be diminished by limiting the amount of tissue influenced by stimulation using strategies such as bipolar configuration and reduction of amplitude. Tetanic muscle contraction does not habituate and stimulation parameters should be set at least 10% below the threshold of visible muscle contractions.[17] If the therapeutic window is narrow, it suggests that the electrode is placed too far laterally and repositioning of the electrode may need to be considered. Diagnostic tests need to be performed to distinguish between tonic muscle contraction and dystonia.

2.1.6. Management of Paresthesia

Available Data

In STN DBS, paresthesias contralateral to the stimulation electrode have been reported as a common side effect when increasing the stimulation amplitude.[6,8,22,39,42,43,55,81] In most cases, paresthesias rapidly habituate. Paresthesias have been attributed to excitation of medial lemniscal fibers that run posteroventrally to the STN.[17,85]

In Vim DBS, dysesthesias are experienced by the majority of patients[67] when the stimulator is switched ON or the amplitude of stimulation is increased. They usually habituate within seconds, but may persist as a mild unpleasant and tingling sensation in up to 10% of the patients.[63] Dysesthesias probably result from current spread to the adjacent ventrocaudal nucleus of the thalamus or lemniscal fibers entering the thalamus.[89]

In GPi DBS, dysesthesias are not a frequent problem. There is no literature available to provide evidence for a standard approach to managing dysesthesias.

Conclusions

In STN DBS and Vim DBS, paresthesias are a common adverse effect and usually habituate.

Pragmatic Recommendations

In most cases, paresthesias habituate and resolve within seconds to minutes. If, however, the paresthesias are persistent, modification of stimulation parameters or a change in electrode configuration may be required. If manipulations of parameters are not successful, repositioning of an electrode may be necessary.

2.1.7. Management of Eyelid and Ocular Disturbances

Available Data

Eye movement abnormalities include ocular deviation and eyelid apraxia. Ocular deviation can result in double or blurred vision and may cause adduction or upward or downward eye deviation of the ipsilateral eye as well as conjugate movements of both eyes.[17,90] Ipsilateral ocular deviation has been attributed to current spread into oculomotor nuclei or nerve fibers and is not a symptom that habituates.[17,85] In STN DBS, the overall incidence of severe eye movement disturbances appears to be low. In one patient, revision of the electrode was regarded necessary because of otherwise intractable double vision[80] and in another case surgery was aborted due to visual disturbances during surgery.[39] Ipsilateral eye deviation has not been reported to occur in GPi DBS or Vim DBS.

Conjugate eye movements are frequently observed when stimulating at low thresholds within the STN. Conjugate ocular movements have been reported to occur frequently in STN DBS and may be due to influence of the oculomotor basal ganglia loop or current diffusion to supranuclear fibers.[17] However, the incidence of this adverse effect is not available in the current literature. There is one case report of contraversive eye deviation during GPi DBS. This has been attributed to an excitation of fibers in the internal capsule.[91]

Eyelid opening apraxia has been reported following STN DBS in several studies with a prevalence between 1.8% and 30%.[4,6,22,29,54,55] The incidence of eyelid apraxia following GPi DBS appears to be significantly lower than in STN DBS, and only one case has been reported.[83] It has not been reported following Vim DBS. The etiology of eyelid opening apraxia has not been clearly defined. The association, however, with blepharospasm suggests a kind of focal dystonia.[92] Apraxia of lid opening may occur early after surgery and improve, or it may persist in the long term.[29] Botulinum toxin injections every 2 to 3 months are an effective method of controlling this symptom,[93,94] further supporting the possibility of pretarsal blepharospasm. Transient side effects such as ptosis, blurred vision, and dry eyes may occur.[93,95]

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Conclusions

Eye movement abnormalities following surgery include monocular ipsilateral eye deviation, conjugate eye movements, and eyelid opening apraxia.

Pragmatic Recommendations

Monocular eye deviation suggests an electrode that is placed too far medially. If lowering stimulation parameters, changing contacts, or switching to bipolar configuration is inadequate in terms of optimizing PD control without inducing eye deviation, then the electrode needs to be repositioned. Conjugate eye deviation commonly habituates. However, if this is persistent, further adjustment of stimulation parameters should be considered. Because apraxia of eyelid opening can occur even with relatively low stimulation parameters, adjusting stimulation does not necessarily resolve the problem. An effective treatment of apraxia of eyelid opening is botulinum toxin injections in the orbicularis oculi muscle.

2.1.8. Management of Visual Disturbances

Available Data

Visual disturbances in terms of homonymous hemianopias have been described following pallidotomy as a result of lesioning of the optic tract.[96] Optic tract responses in terms of phosphenes have been utilized to facilitate intraoperative guidance of the electrode,[97] as the presence of phosphenes suggests excitation of the optic tract. Permanent visual disturbances have not been described in GPi DBS, STN DBS, or Vim DBS.

Conclusions

In GPi DBS, phosphenes may occur because of current spread to the adjacent optic tract.

Pragmatic Recommendations

Though phosphenes usually habituate, if persistent, a more dorsal contact should be chosen for chronic stimulation.

2.1.9. Management of Behavioral and Cognitive Problems

Available Data

Behavioral and cognitive problems have been reported with DBS of all three targets. However, the incidence of these problems appears to be higher in STN DBS than in GPi DBS or Vim DBS. In STN DBS, the most common neuropsychiatric side effect in the immediate postoperative period is transient confusion with an incidence between 1% and 36%.[7,29,42,44,46,55,81,98–101] In the follow-up period, hypomania, depression, apathy, and suicide risk have been reported in several studies. Postoperative hypomania was reported in 4% to 15% of STN DBS patients in four studies[29,55,102,103] and postoperative depression has been reported in 1.5% to 25% of patients.[6,29,40,43,46,55,82,98,99,104,105] Overall group depression scores have been reported to improve at 3 and 12 months in multiple studies.[46,102,106,107] However, the clinical relevance has not been ascertained. A number of uncontrolled series have documented suicide attempts and/or suicides following STN DBS.[29,108] The base rate of suicide in elderly PD patients has been reported to be either similar to[109] or one-tenth the rate of the general population.[110] The base rate of suicide in younger patients with severe PD who present for surgery is not known but is likely to be higher. Within the first three postoperative months, apathy, which can respond to dopaminergic medication, occurs, although the incidence is not known. In contrast, permanent apathy was identified in 12% of patients, in whom 80% had associated decreases on frontal lobe measures.[29]

In STN DBS, the literature regarding neuropsychological outcome reports mixed outcomes. In carefully selected patients, most groups have reported relatively little cognitive morbidity,[28,29,107,111–116] with improvements of unknown clinical relevance in some areas.[30,111,117–119] In contrast, other studies have reported declines. The most robust finding across studies appears to be a decline in word fluency.[102,107,111,112,120–124] However, a minority of studies have documented declines in verbal memory[102,111,123,125] and selected measures of executive function.[123,125]

In GPi DBS, case reports suggest that postoperative neuropsychiatric symptoms can occur.[126,127] However, one study suggested postoperative psychiatric symptoms in GPi DBS are less frequent than after STN DBS as 3 of 16 STN versus 0 of 11 GPi patients developed depression requiring psychiatric treatment.[46] Overall improvements in group depression and anxiety scores have been documented, although the clinical relevance is not known.[46,112,128] One study that differentiated between primary and secondary anxiety suggested that GPi stimulation improved primary anxiety symptoms.[128]

GPi DBS is relatively safe from a neuropsychological standpoint.[76,112,129] Decrements in verbal fluency can occur,[35,125,130,131] but often resolve by 12 months.[46] In some cases, a variety of other minor cognitive changes may occur after unilateral GPi DBS,[132] while in rare instances of bilateral GPi DBS, significant executive dysfunction may ensue.[118]

In Vim DBS, studies suggest neuropsychological changes are usually minimal after surgery.[133–136] There are no reports on psychiatric outcomes.

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Conclusions

In STN DBS, hypomania, depression, and apathy can occur in the period following initiation of stimulation. Suicidal ideation has been reported. In the long term, depression, apathy, and neuropsychological changes have been noted.

In GPi DBS, there is an apparently lower incidence of postoperative psychiatric issues than in STN DBS. However, this may be influenced by cohort effects, lack of systematic observation, or exclusion of high-risk patients. Insufficient studies exist to allow determination of the incidence of cognitive deficits after unilateral or bilateral GPi DBS. The procedure is unlikely to result in major cognitive morbidity, but verbal fluency and executive function changes can occur.

In Vim DBS, there are insufficient data to draw conclusions about the occurrence of postoperative psychiatric symptoms. The procedure appears relatively safe from a cognitive standpoint.

Pragmatic Recommendations

In the initiation/stabilization period, clinicians should assess for postoperative depression, suicidal ideation, hypomania/mania, and apathy and institute appropriate treatment as needed. In the long term, patients should be assessed for depression, dementia, apathy, and hallucinations. Patients should have a complete postoperative neuropsychological assessment whenever clinically indicated. In particular, patients with preoperative vulnerabilities and apparent cognitive decline or behavioral changes such as impulse control, poor judgment, pathological gambling, or hypersexuality should be monitored.

Points To Be Addressed

The differential role of disease, stimulation, and medications in neurobehavioral symptoms needs to be further studied. This also includes sexual dimensions or specific female/male-related patient problems of patient life and age-related psychosocial problems.

2.2. When Is the Optimal Response to Be Expected, Do Patients Develop Tolerance, and How Should Secondary Deterioration Be Improved?

Available Data

STN DBS studies with a follow-up period up to 5 years have been published. In studies with a follow-up period of 3 to 6 months,[3,11,42,69,99,137,138] an improvement in *off* and *on* Unified Parkinson's Disease Rating Scale (UPDRS) activities of daily living (ADL) and motor subscores has been reported to be between 44% and 72% and 50% and 70%, respectively. The LED was reduced between 37% and 80% and dyskinesias were reduced by 57% to 83% compared to baseline. There were no reports of reduced efficacy of STN DBS during this short follow-up period. Studies covering a period of 12 months[4,6,8,14,28,40,44,46,70,80–82,100,101,104,105,139,140] showed an improvement in UPDRS ADL and motor scores between 32% and 78% and 33% and 67%, respectively. LED was reduced by 19% and 69% and dyskinesias decreased between 63% and 91%. In some of these studies,[4,6,8,28,44,46,82,101,104,139] the evaluations were performed at different time periods. In the first year, there was no deterioration in the benefit achieved with stimulation. Analysis of subitems of the UPDRS motor section showed a beneficial response from stimulation on rigidity, akinesia/bradykinesia, tremor, and axial symptoms that was sustained for 12 months following surgery.[4,8,46,82] Studies with a follow-up of 24 months reported a reduction of UPDRS ADL and motor scores between 27% and 55% and 27% and 63%, respectively.[7,9,22,39,45,55] LED was reduced by 32% to 81% and dyskinesias were reduced by 46% to 90%. There was no significant deterioration in benefit to motor and ADL subscores or dyskinesias over time. The reduction in LED requirements was sustained for the 24-month period. Subgroup analysis found a deterioration in the initial benefit to axial symptoms at the time of the last evaluation.[22] One study[39] also observed a reduction in the efficacy of STN DBS on postural instability after 24 months compared to the 3- and 12-month assessments. In two other studies,[9,55] improvement of axial symptoms by stimulation was robust after 2 years. The results of a 5-year prospective study of 42 consecutive patients[29] showed an improvement of the UPDRS ADL and motor subscales by 54% and 49% compared to the preoperative state. But there was a significant worsening of both UPDRS subscales after 5 years compared to the 1-year assessment. Worsening of the UPDRS motor scores was due to a significant decline of the stimulation effect on akinesia/bradykinesia, speech, postural stability, and gait, whereas the improvement of tremor and rigidity was sustained. There was a stable reduction of dyskinesias by 52% and LED by 63% after 5 years.

In GPi DBS, there are 11 studies[3,24,28,35,42,46,74,76,139,141,142] that suggest a robust improvement in motor symptoms and dyskinesias after 1 year. However, one study that continued for 2 years[74] noted that two out of three patients had recurrence of motor fluctuations between 9 and 15 months, and one-third had gait initiation failure that was not responsive to alterations in stimulation parameters. Another study that examined the acute effects of stimulation[141] assessed patients an average of 2.8 years following surgery and found that there was still

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significant improvement in *off* period akinesia/bradykinesia, rigidity, and postural instability, suggesting that there is continued benefit from GPi DBS. Conversely, reemergence of symptoms after several months of GPi DBS has also been reported.[78] Two of five patients had undergone further bilateral STN surgery with an excellent result 6 months following the second surgery, and another two who had loss of efficacy with GPi DBS were in the process of undergoing STN DBS. This outcome occurred in another series of 11 patients who had received bilateral GPi DBS and were followed for up to 5 years.[77] Though all features of the UPDRS scores except speech and swallowing were improved at 1 year, at 3 years effects on bradykinesia, gait, and postural stability were lost and by 5 years only rigidity was still improved compared to baseline. Of four patients at 2 and 3 years who lost benefit from GPi DBS, marked improvement recurred following subsequent placement in the STN.

In Vim DBS, studies with a follow-up period from 1 month to 8 years report good long-term abolition or significant reduction of tremor in PD patients.[16,20,30,31,36,60,61,63,64,67,79,143–147] In most studies, no evidence of tolerance has been reported.[16,20,30,36,60,63,67,79,143–145,147] However, some investigators found some tolerance development ranging from 5%[31,146] to 20%[61,64] of treated patients. Stimulation parameters are usually increased during the first 6 to 12 weeks[36,79] and stable afterward. Only nonsignificant changes have been reported in the follow-up period. In most studies, Vim DBS has not been reported to improve symptoms other than tremor.[31,60,63,79] In some studies, a mild amelioration of rigidity,[16,144,146] dyskinesias,[61] or akinesia[147] has been reported.

Conclusions

Patients generally develop an optimal response between 1 and 6 months following surgery. Only minor improvements have been reported after this period of time, though in individual cases a longer adjustment may be necessary to achieve the best clinical outcome.

In STN DBS, the main effects are substantially preserved over a period up to 5 years. The long-term beneficial effect of STN DBS includes both *off* and *on* motor symptoms of PD. Nevertheless, some patients can have worsening primarily of axial symptoms.

In GPi DBS, tolerance does not develop in the first postoperative year; however, it may occur later. However, the target for DBS within the GPi is less defined than for the STN and the present studies are not based on documented locations for the electrodes. In Vim DBS, the effect on tremor is sustained in the long term in the majority of patients. Other features such as bradykinesia, rigidity, and dyskinesias show none or only mild improvement and may even decline with the natural progression of PD.

Pragmatic Recommendations

Most patients who are properly diagnosed with idiopathic PD who have good placement in the target structure and a confirmed response to levodopa should demonstrate improvement in parkinsonian symptomatology during the initial programming session. Continued adjustments in medication and stimulation parameters are generally necessary over the first several months after implantation. In general, the vast majority of PD patients should be optimized within 6 months following implantation. Continued adjustments in medication and stimulation will be necessary as the disease progresses.

In STN DBS patients with worsening of axial symptoms, an increase in medication or stimulation should be attempted. However, this approach may be unrewarding and supportive care may be the only alternative. This includes assessment of swallowing function with appropriate interventions such as softening consistency of diet, chin tuck maneuvers, and other measures to ensure prevention of aspiration, as well as physical and occupational therapy with a focus on gait training and postural stability. As postural instability may result in a significant fall risk, supplemental safety measures such as a cane, walker, or wheelchair should be considered. Psychological support may also be helpful to patients. An aggressive and significant decline of stimulation effect on all parkinsonian symptoms accompanied by a loss of levodopa responsiveness may argue for an alternate cause of Parkinsonism. In those cases, further escalation of stimulation or medication is unlikely to yield any benefit. Instead, supportive care with a focus on the particular symptom is essential in caring for these patients.

In GPi DBS patients, reemerging symptoms should prompt an adjustment of stimulation parameters, including a change in electrode configuration, amplitude, frequency, and pulse width. Medication adjustment may be necessary with higher doses of dopamine agonists and levodopa. Supportive care should be provided by a multidisciplinary team. For a cognitively intact patient with a loss of effect from GPi stimulation, surgery with bilateral STN DBS may be considered.

In Vim DBS patients, in the case of reoccurrence of tremor, adjustment in stimulation parameters including a change in electrode configuration, amplitude, frequency, and pulse width should be attempted. However, at some point, adverse effects such as dysarthria, dystonia, paresthesia, and disequilibrium may occur as a result of

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increasing amplitudes that may limit the capacity to control the tremor. To address the issue of tolerance, one option may be to provide a stimulation holiday, possibly resetting the threshold of the tremor. If a modification of stimulation parameters is ineffective, or if functional disability from other symptoms of PD emerges, STN DBS should be considered.

Points To Be Addressed

Prospective studies addressing patients' characteristics, symptom control, as well as medication regimen are needed. Prospective studies are needed to determine if STN DBS has a modifying influence on the course of the disease and specifically on symptoms resistant to DBS or dopaminergic medication. It is unknown to what extent quality of life is compromised by such resistant symptoms. Studies should aim at defining predictive factors for patients at risk for developing secondary resistant symptoms.

3. WHAT ARE LONG-TERM COMPLICATIONS THAT NEED NEUROSURGICAL ATTENTION?

3.1. Infections and Erosions

Available Data and Conclusions

DBS-related infections present variably in terms of time and location. Rates of infections vary from 1% to 15%[20,22,29,39,42,54,65,67,148–157] and may occur even months or years following surgery. They may be related to systemic infections (sepsis), cellulitis, or skin erosion exposing the implant. No adequate studies are available concerning this issue.

Practical Recommendations

Delayed infections and erosions may occur following DBS surgery. Routine inspection of the skin over the implant should occur at follow-up visits. Any sign of infection such as swelling and erythema or skin erosion should be referred for immediate attention to the neurosurgeon.

3.2. Sudden Loss of Efficacy or Intermittent Stimulation Effects

Available Data

Sudden loss of stimulation efficacy following previous symptom control suggests a hardware-related complication. The risks of these complications varies from 2.7% to 50% of patients.[20,22,29,39,42,54,65,67,148–157] This wide range may be related to differences in reporting of duration of follow-up, surgical technique, patient selection, the neurosurgeon's experience, and changes in the hardware available for implantation over time.

The following list summarizes the possible causes of a sudden failure of stimulation: IPG-related problems, electromagnetic interference, IPG malfunction, IPG end of battery, internal IPG wire breakage, wiring- and lead-related problems, wire or lead fracture, disconnections, and lead dislocation.

Conclusions and Practical Recommendations

There is limited literature regarding how to determine hardware-related complications, prevention, and optimal management if they occur. The following discussion is based on expert opinion.

If a sudden loss of stimulation efficacy occurs, the following steps may be taken to determine the etiology.

One, the IPG status should be assessed. The most frequent cause of a sudden decrease in stimulation efficacy is an accidental turning OFF of one or both neurostimulators. Unless the patient used a magnet or the patient Access Review Therapy Controller to turn OFF the neurostimulator, electromagnetic interference (EMI) is likely the cause. Unusually high numbers of ON–OFF cycles, in a range of >25 events in the neurostimulator log, are further evidence of this. Resetting the magnet activation counter to zero at the end of each follow-up visit permits frequent inadvertent activation cycles to be correlated with specific time intervals. Household devices used in close proximity to the IPG may cause EMI. Examples are electric shavers, electric toothbrushes, microwaves, and mixers, as well as electric drills and other power tools. EMI has occasionally been reported due to antitheft devices in department stores or security gates with metal detectors. A detailed patient history, including the time an increase in PD symptoms was first noted, usually helps to narrow down the source of EMI. Patient training and education is required to avoid this. If patients with Kinetra neurostimulators experience problems related to EMI, the neurostimulator's magnetic switch may be disabled.

Two, if the programming device does not connect telemetrically with the IPG, it suggests battery exhaustion. When the battery is close to running out, then the Access Review Therapy Controller indicates "end of life" (EOL). If parameter settings are back to default (factory settings) with 0 V output device switched OFF and serial number unavailable, the program controller indicates "power on reset" (POR). This has been found to be associated with hardware failure[158] and referral to the neurosurgeon is indicated for possible IPG replacement.

Three, if after interrogation, it is found that the IPG is ON with appropriate settings, a connection problem may

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be suspected. The integrity of the electrical circuit can be assessed through the measurement of impedance and battery load. Impedances greater than 2,000 Ohm (Soletra or Itrel II) or 4,000 Ohm (Kinetra) suggest a connection problem. If the open-circuit current of the neurostimulator battery (Itrel II neurostimulator 7 μA and Kinetra neurostimulator 15 μA) does not increase while carefully raising the amplitude to a maximum of 3 to 5 V, an interrupted electrical circuit is very likely. Impedances below 50 Ohm and high battery drain > 2,000 μA indicate a short circuit in the system. Plane radiographs may show disconnection or a fracture in the DBS electrode or extension wires.[156] In rare occasions, connection problems are not evident on X-ray and may require surgical externalization of the system. Regardless, referral to the neurosurgeon should be made for further evaluation and treatment.

Four, if a connection problem has been ruled out, lead migration should be considered. An imaging study is mandatory. Significant migration may be visible on skull X-rays, though minor displacements may require CT or MRI. It is important to note that with MRI there is a theoretical risk of injury from electric current induced by the magnetic field.[159] Referral to the neurosurgeon is indicated for further evaluation, including the comparison of the new imaging study with the postoperative scans.

3.3. Pain and Discomfort Following Implantation

Available Data

Neck pain or restrictions of head movements may occur in relation to the extension wires, although this has not been specifically reported in the literature.

Practical Recommendations

Complaints of neck discomfort or pain during head movements should be referred to the neurosurgeon for further evaluation of treatment options.

Points To Be Addressed

Device-related complications and infections can lead to significant morbidity and have an important economic impact. There is a clear necessity for prospective controlled trials to assess the incidence of these complications and techniques to prevent them.

4. LONG-TERM PROBLEMS

4.1. Are There Specific Postoperative Social or Job-Related Problems?

Available Data

There are two studies on short-term social outcomes in patients with STN DBS.[98,160] There are no studies on long-term social outcomes or on job-related problems. In a retrospective short-term study utilizing the Social Adjustment Scale, late age of PD onset was associated with poor global social adjustment. Poor adjustments in social life, leisure, and social adaptation were associated with the persistence of levodopa-induced motor symptoms. Despite motor improvements, 6 (25%) of 24 patients had a deterioration in marital relations, explained as related to changes in autonomy and roles.[98] A qualitative study at 6 months following surgery documented descriptive psychological and relationship difficulties.[160] Other studies have also reported in passing behavioral changes sufficiently noticeable to involve disturbance of family or social interactions.[123]

Conclusions

There are insufficient data to comment on social outcomes adequately.

Pragmatic Recommendations

Clinicians should be aware of the potential for impairments in social outcomes following the procedure. This should be addressed during preoperative counseling and postoperative assessments. The extent of baseline social impairment in this group with advanced PD presenting for surgery may be a contributory factor. Social rehabilitation programs, individual/couple counseling, or support groups may be useful for some patients.

Points To Be Addressed

As social outcomes are of significant clinical relevance to patients, studies focusing on such outcomes in the long-term should be performed. Prospective controlled studies to determine prognostic or contributory factors for social outcome would be indicated. Similar to epilepsy surgery, where social rehabilitation programs are part of the multidisciplinary treatment plan, DBS surgery is also a major life event and, although insufficient data exist, rehabilitation programs are considered important for the future development of DBS therapy.

4.2. Long-Term Development of Dementia

Available Data

Dementia is a late and likely an age-dependent complication of PD in approximately 30% of patients. The diagnosis frequently coexists with or is preceded by medication-induced psychiatric side effects.[161] The pathophysiology may be associated with the neurodegenerative process of PD affecting cortical regions and nondopaminergic brainstem or basal frontal nuclei.[162] De-

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mentia is not improved by dopaminergic medications and DBS does not reverse cognitive decline.

In a 5-year prospective study of 49 patients, 6% of patients developed dementia, though notably there was no comparative control group.[29] Similar to that observed in the general PD population, the appearance of behavioral symptoms such as apathy and hallucinations appears to parallel this decline in the postoperative population.[29]

There are no reports of treatment for dementia in PD patients with DBS. Recently, a large randomized study on the effect of rivastigmine in PD dementia found a positive effect on cognitive function and overall status.[163]

Conclusions

Dementia occurs in PD patients with DBS, but the incidence seems to be similar to that of the general PD population. There are no specific studies on the treatment of patients with dementia with DBS.

Pragmatic Recommendations

Although insufficient data exist, as with clinical management of general PD patients, clinicians should be aware of the potential development of dementia in the PD DBS population. The underlying etiology is believed to represent the natural course of the disease process,[30] although further controlled studies are required to confirm this. Cognitive status should be screened during postoperative follow-up visits and appropriate therapy should be considered. A multidisciplinary approach should be utilized in the management of dementia. Significant cognitive deficits following intraoperative complications have a different underlying etiology and may not respond to standard dementia therapy such as cholinesterase inhibitors.

Points To Be Addressed

Controlled studies should address the frequency, predictive factors, and treatment of dementia in the DBS population. The limitations of DBS on nonmotor aspects of PD progression should be recognized and actively addressed.

4.3. General Medical Issues

Available Data

There are no formal studies on the interference of medical procedures with DBS. Pulse-modulated radiofrequency diathermy applied to the maxilla produced permanent diencephalic and brainstem lesions leading to a vegetative state in one STN DBS patient.[164] The electrical current induced by the magnetic field of MRI scanners carries a theoretical risk of injury.[159] We are aware of one case of a permanent brainstem lesion after spinal cord MRI (Ali Rezai, personal communication). Case reports indicate that cardiac pacemakers and a DBS system may be safely implanted in the same patient. Cardiac defibrillation carries a theoretical risk of injury; however, no neurological sequelae were reported in a patient following 10 cardioversions from an internal cardiac defibrillator.[165] Although not formally reported in the literature, EMI may result from a variety of medical devices (e.g., X-ray or vibrators used in physical therapy). Electrocoagulation during surgery may carry a theoretical risk by inducing currents within the DBS system, but thus far no adverse effects have been reported.

There are no formal studies on the interference of DBS with medical diagnostic procedures. DBS may cause artifacts in electrodiagnostic procedures such as ECG, EEG, and evoked potentials, which are not harmful to the patient unless they are misinterpreted as representing pathological abnormalities, particularly by automated computerized reading programs.

Conclusions

Diathermy can interfere with DBS and can cause life-threatening complications. There are no conclusive data on the safety of head coil MRI, cardiac defibrillation, or electrocoagulation. DBS may interfere with electrodiagnostic procedures but is not harmful to the patient.

Pragmatic Recommendations

Diathermy must be avoided in DBS patients. The use of MRI should be restricted to protocols that have been evaluated for safety. Safe protocols exist for MRI of the head but not for the spinal cord and trunk. Recommendations from the manufacturer, which may serve as a guideline, are provided in the Soletra and Kinetra packaging instructions. Cardiac devices can be implanted but the patient should be carefully followed for potential interactions between the two devices. Electrocoagulation during surgery should be restricted to a bipolar mode as suggested by the manufacturer. If artifacts on electrodiagnostic tests are a concern, the IPG can be switched OFF during testing.

4.4. Lifestyle Issues

Available Data and Conclusions

There is no literature regarding restrictions of DBS patients in professional or leisure activities.

Pragmatic Recommendations

Security gates with metal detectors may turn OFF the DBS device and should be avoided by DBS patients.

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Some professional or leisure activities may require the use of machinery that may result in EMI; solutions should be sought on an individual basis. Common sense suggests that any sport causing extreme mechanical stress to the device should be avoided (e.g., contact sports such as headers during soccer, parachuting). Legal regulations in some countries may forbid patients with DBS to operate machinery such as airplanes. DBS does not specifically limit driving a car, but, as with any general PD patient, this should be assessed on an individual basis.

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