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Post-Operative Management of Parkinson Patients with Deep Brain Stimulation

Niels Allert[1] and Volker Arnd Coenen[2]
[1]*Neurological Rehabilitation Center Godeshoehe, Bonn*
[2]*Stereotaxy and MR-based OR-techniques/Department of Neurosurgery, University of Bonn, Germany*

1. Introduction

Oral medication still remains the mainstay for treating Parkinson's disease (PD). Since the introduction in the late 60ies levodopa is regarded as the gold standard because of the best efficacy on akinesia and rigidity compared to other drugs like dopamine agonists or anticholinergics (Poewe et al., 2010). However, oral dopaminergic treatment of motor symptoms is complicated by the development of motor fluctuations and dyskinesias. These complications can be observed in 40 % after 5 years and 80 % after 10 years PD duration. Moreover, tremor can be refractory to oral medication. During the past 15 years deep brain stimulation (DBS) has become an important therapeutic option for patients with these motor complications. DBS has been approved in Europe for the treatment of tremor in 1995 and for advanced PD in 1998. Approval by the Food and Drug Administration (FDA) was given for tremor in 1997 and for advanced PD in 2002. The main targets for DBS are the subthalamic nucleus (STN), the internal globus pallidus (GPi) and the ventral intermediate (Vim) thalamic nucleus. DBS is a symptomatic treatment and does not influence the progression of the disease (Hilker et al., 2005). Therefore, the number of patients eligible for DBS is limited. Experts estimate that some 10 to 15 % of PD patients are suitable candidates. The implantation of stimulation electrodes is not therapeutic in itself. Apart from the precise electrode localization the subsequent programming of optimal stimulation parameters and adjustment of medication is mandatory (Deuschl et al., 2006). An increasing number of PD patients are treated by DBS so that more and more doctors and care givers involved in the treatment of PD patients are regularly concerned with the special demands of these patients. The following chapter gives an overview on specific issues in the post-operative management of PD patients with DBS.

2. Post-operative management of PD patients with DBS

One of the reasons for the resurgence of surgical therapies in the treatment of PD is that DBS surgery is associated with a limited risk of permanent morbidity of 1 % (Voges et al., 2007). Furthermore, DBS is adjustable and reversible concerning efficacy as well as side effects. This is in contrast to earlier neurosurgical techniques which used permanent

lesions of the GPi (pallidotomy) or Vim (thalamotomy). The implantation of electrodes alone does not result in a permanent clinical benefit but requires optimal programming of stimulation parameters and adjustment of medication. However, the surgical procedure often results in a transient microlesion effect already mimicking some of the benefits of chronic DBS (Mann et al., 2009). This microlesion effect implies that motor symptoms like tremor or akinesia are improved immediately after surgery although DBS has not been started. A microlesion effect is usually observed for a couple of days but can last even longer up to weeks. A significant microlesion effect can make the evaluation of stimulation effects on motor symptoms difficult. Therefore, some DBS centers routinely release patients after electrode implantation and admit them a couple of weeks later to start DBS programming. In other centers, DBS programming is started shortly after electrode implantation.

2.1 Surgical issues – Follow-up of wound healing and proper technical function

Proper technical function of the DBS system should be ascertained after surgery. Most notably, the impedances of all stimulation contacts of the commonly used quadripolar stimulation electrodes should be checked. A disconnection of a single or more electrode contacts is characterized by high impedances (depending on the impulse generator model measurements of >2.000 Ohm, >4.000 Ohm or >40.000 Ohm) when measured in a monopolar stimulation mode with the respective contact as cathode and the impulse generator case as anode. Moreover, in this case increasing the voltage will not result in stimulation effects of the central nervous system and will not increase the baseline current of the impulse generator. A short circuit on the other hand is characterized by very low impedances (< 100 Ohm) of the respective contacts when measured in a bipolar stimulation mode with one contact as cathode and the other contact as anode. Detecting a disconnection of single contacts or a short circuit does not necessarily imply a surgical revision. If stimulation of alternative contacts is effective and satisfying the dysfunction should only be regularly controlled in the course of time to exclude extension of technical problems to other leads. In contrast, complete fractures of electrodes or extension leads necessitate surgical revision. Lead fractures have been reported in up to 5 % of the patients in earlier reports (Hamani & Lozano, 2006). In most of these cases the connection between stimulation electrode and extension lead was located below the mastoid. This risk can be significantly reduced if the connection is located at the calvarium (Blomstedt & Hariz, 2005).

Wound healing should be carefully controlled during the first weeks after surgery but also at control visits thereafter. A sterile seroma can occur around the impulse generator but resolves spontaneously in most cases. However, bacterial infections can develop requiring surgical and antibiotic treatment. Bacterial infections are most often found at the impulse generator site within 4 to 6 weeks after implantation but can also occur at other parts of the DBS system (extension lead, electrode). They are exceptionally rare intracranially (Sillay et al., 2008). In most cases of a bacterial infection at the DBS system mere antibiotic treatment does not eradicate bacteria completely so that the system or part of the system needs to be explanted and replaced after a period of consolidation (Bhatia et al., 2010). Apart from infections, skin erosions can be a problem (Sixel-Döring et al., 2010). Such skin erosions can even occur after months and need surgical revision to prevent secondary bacterial infection (Lanotte et al., 2009).

2.2 Determination of optimal stimulation parameters

DBS for PD is a chronic high frequency stimulation of central nervous tissue without feedback mechanisms like the sensing of intrinsic electric activity by cardiac pacemakers. Telemetric programming allows adjusting the stimulation frequency (in Hz), the impulse width (in μs) and the stimulation amplitude (in V in case of constant voltage stimulation and in μA in case of constant current stimulation). Furthermore, quadripolar stimulation electrodes allow variable activation of single or more electrode contacts for either monopolar DBS with the impulse generator serving as anode or bipolar DBS with anode and cathode both being located at the electrode. The principle of DBS programming in PD is to systematically evaluate the clinical responses to stimulation in order to obtain maximal benefit on motor symptoms without or with minimized side effects. This process has to encounter variable latencies of stimulation effects at the different DBS targets which range from seconds to weeks.

2.2.1 Subthalamic nucleus - STN

The subthalamic nucleus has become the primary DBS target in PD during the last 10 to 15 years (Benabid et al., 2009). The sub-target for PD motor symptoms is the dorsolateral hence sensorimotor STN better referred to as the anterior lateral and superior STN (Coenen et al., 2008). The main reasons for this preference are that all PD motor symptoms including tremor, akinesia and rigidity are improved allowing a reduction of dopaminergic medication by some 60 %. The latter also results in a significant reduction of dyskinesias. Recent studies, however, have questioned whether GPi DBS may be equally effective or even superior in individual patients (Follett et al., 2010).

STN DBS directly improves PD off symptoms. Therefore, stimulation effects are best evaluated in the medication off state. The influence of the different stimulation parameters on motor symptoms has been evaluated more systematically in several studies (e.g. Moro et al., 2002). Stimulation frequency has to be above 60 Hz. An increase of the frequency to more than 100 Hz improves DBS efficacy whereas the further benefit of frequencies above 130 Hz is usually limited. To prevent unnecessary current drainage a frequency of 130 Hz can be recommended as the preferred frequency for STN DBS. The impulse width is commonly set at the lowest technical option of 60 μs. Increasing the impulse width does usually not result in specific advantages but results in a negative relationship of DBS efficacy and current drainage. Activation of different contacts of the commonly used quadripolar stimulation electrodes allows to further modify the volume of tissue activated. Initially, efficacy of DBS as well as side effects is evaluated for all contacts separately to determine the contact(s) with the best short term efficacy within seconds to minutes.

Rigidity has been found to be a particularly valuable symptom because of a short latency response. Moreover, rigidity can be reliably evaluated when using reinforcing manoeuvres like voluntary movements of the contralateral hand (Froment's manoeuvre). Akinesia often responds within short time as well but full anti-akinetic effect of DBS may take hours to days. Whereas tremor in DBS of the Vim usually responds within seconds, variable latencies can be observed in STN DBS which range from seconds to weeks. Stimulation-induced dyskinesias are another important motor phenomenon to evaluate DBS efficacy. Stimulation-induced dyskinesias have been demonstrated to be a predictor of good long-term outcome of DBS (Gago et al., 2008). Most importantly, dyskinesias often develop or increase in severity with a latency of minutes to hours so that careful

observation of patients after initiation of DBS is recommended. In case of persistent stimulation induced dyskinesias or if the therapeutic window of sufficient symptom control and the induction of dyskinesias is narrow a more dorsal stimulation contact often offers a beneficial alternative.

Apart from motor symptoms DBS can induce psychiatric and behavioural changes. Most of these changes are transient but can be observed during periods of days to weeks and even a couple of months (Temel et al., 2006). The origin of such changes still needs further exploration. Both, direct stimulation of nervous tissue involved in the processing of emotions as well as changes in dopaminergic medication seem to play a role. Although direct stimulation-induced depression has been observed in PD patients with STN DBS (Bejjani et al., 1999), depression seems to be more often due to the reduction of dopaminergic medication and can be improved by increasing this medication (Thobois et al., 2010). In contrast, hypomania seems to be more directly related to stimulation effects. Stimulation of the inferior and medial STN corresponding to the limbic subnucleus of the STN has been proposed as a likely structure. Our own studies also suggest the medial forebrain bundle as a candidate (Coenen et al., 2009). Clinically, stimulation induced hypomania can usually be improved by either reducing the volume of tissue activated or by stimulating a more dorsal contact.

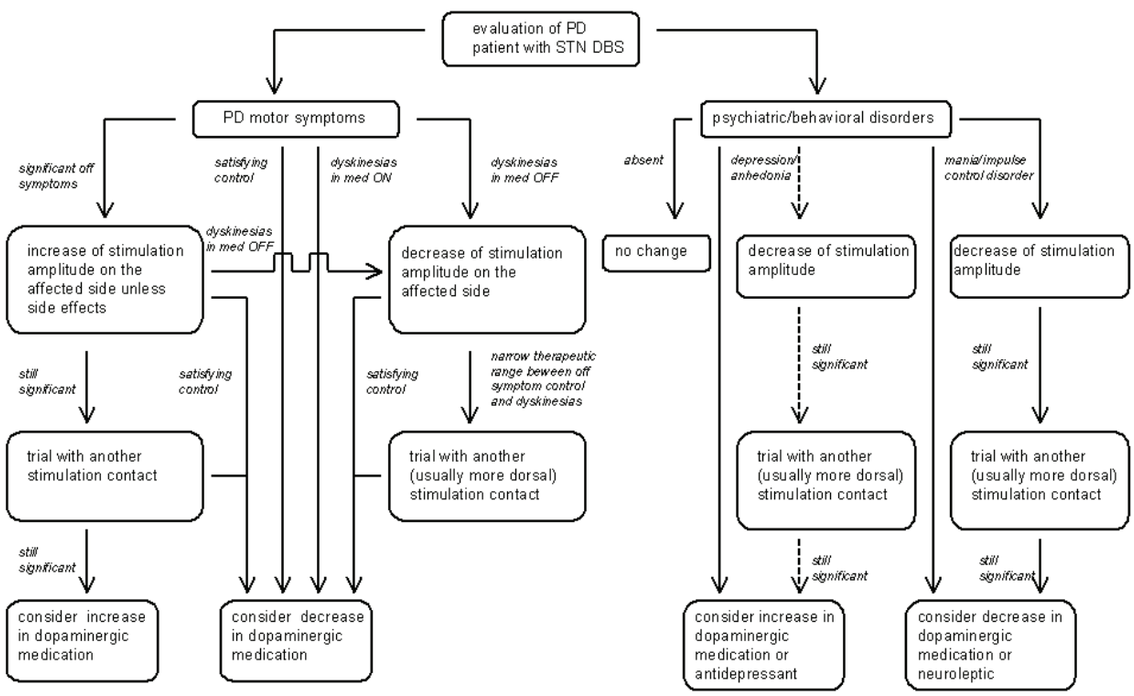


Fig. 1. Algorithm for stimulator and medication adjustments in PD patients with STN DBS (dotted arrows: rare) (adapted from Allert et al., 2011)

Due to the different latencies of stimulation effects as well as adaptive changes with chronic stimulation patients need to be observed and stimulation parameters as well as medication continuously adapted during the first days to weeks. Figure 1 depicts a clinical algorithm for this process of stimulator and medication adjustments.

2.2.2 Globus pallidus internus - GPi

Stimulation of the GPi also improves the major PD motor symptoms, i. e. tremor, akinesia and rigidity. However, initial studies suggested a less profound effect on akinesia corresponding to only little changes in dopaminergic medication. Moreover, failure of GPi DBS has been reported within the first years as well as after longer periods up to 10 years (Durif et al., 2002; Allert et al., 2010). More recent studies, in contrast, have demonstrated similar motor outcome after STN DBS and GPi DBS with more favourable effects on non-motor symptoms like depression in patients with GPi DBS (Follett et al., 2010). Further studies are certainly needed to evaluate the differential effects of GPi DBS and STN DBS on motor and non-motor symptoms in PD.

DBS of the GPi differs in some aspects from DBS of the STN. Anatomically, the sensorimotor area of the GPi is larger than the sensorimotor area of the subthalamic nucleus. To increase the volume of tissue activated higher amplitudes and impulse widths are used. Correspondingly, the higher energy consumption results in a shorter battery longevity of impulse generators. A differential effect of variable impulse widths has not been demonstrated. Historically, longer impulse widths were often used for pallidal stimulation in the 90ies, because the most common impulse generators at that time doubled energy consumption above amplitudes of 3.6 V so that the combination of an amplitude below 3.7 V and a longer impulse width resulted in improved battery longevity. Newer generations of impulse generators have a linear relationship of stimulation amplitude and current consumption so that the initial use of short impulse widths (60 μs) is justified and probably preferable.

Another important difference is that GPi DBS reduces levodopa-induced dyskinesias directly. Hence, stimulation effects have to be evaluated in the medication on and off state separately. This is even more important because DBS of different parts of the GPi can have opposite motor effects. Thus, a more ventral target in the GPi may result in suppression of levodopa induced dyskinesias but increase in akinesia whereas a more dorsal target may even elicit dyskinesias while improving OFF akinesia (Krack et al., 1998).

As mentioned above, changes of dopaminergic medication are less profound in GPi DBS than in STN DBS (Follett et al., 2010). Adjustment of medication after GPi DBS is, therefore, less likely to induce dopaminergic withdrawal symptoms like apathy and anhedonia and probably contributes to the more favorable outcome on non-motor symptoms like depression (Follett et al., 2010).

2.2.3 Ventral intermediate thalamic nucleus - Vim

Whereas in the late 80ies the ventral intermediate thalamic nucleus has been the first target for DBS in patients with medically refractory tremor it's role in PD has diminished with the demonstration that DBS of the GPi and STN not only improves tremor but also reduces akinesia and rigidity. Vim DBS is very effective in reducing PD tremor but has no impact on akinesia and rigidity. Stimulation effects can be observed within seconds making DBS programming easier than that in STN and GPi DBS. Stimulation parameters are comparable to

those for GPi and STN DBS, i. e. a frequency of 130 Hz and short impulse widths of 60 μs can be recommended for initial programming. In case of unsatisfactory tremor control higher stimulation frequencies can be helpful although a significant benefit of higher stimulation frequencies for tremor suppression has not been systematically demonstrated. The most important side effects of Vim DBS, particularly in the case of bilateral DBS are impairment of speech and gait/balance. These side effects are reversible and depend on the volume of tissue activated. If tremor is incompletely suppressed the aim of DBS programming is to find an optimal compromise between tremor control and acceptable side effects.

2.3 Adjustment of medication

Adjustment of dopaminergic medication is primarily necessary in PD patients with STN DBS. A reduction of some 60 % of the levodopa equivalent dosage has been reported in a number of studies (Benabid et al., 2009) whereas little changes are observed in PD patients with GPi DBS (Follett et al., 2010; Deep brain stimulation for Parkinson's disease study group, 2001). Since Vim DBS only improves tremor but not akinesia and rigidity, the possibility to reduce dopaminergic medication depends on the tremor control by DBS and prevalence of akinesia and rigidity in the individual patient.

Adjustment of dopaminergic medication is very important in patients undergoing STN surgery. In most DBS centers test stimulation is used during electrode implantation to evaluate neurophysiologically the localization of the electrode. Patients are awake and examined for characteristic side effects like muscle contractions from stimulation of the pyramidal tract, dysaesthesias from stimulation of the medial lemniscus, oculomotor disorders from stimulation of the oculomotor nerve or the pyramidal tract (gaze palsy). Moreover, test stimulation is used to evaluate the effect on PD motor symptoms. The latter can best be evaluated in the medication off state so that dopaminergic medication is not given on the day of surgery. To prevent unforeseen long-term effects of dopamine agonists with a long half-life some DBS centers routinely reduce or even discontinue dopamine agonists for a couple of days before surgery. A reduction of dopaminergic medication may also be necessary immediately after electrode implantation because of a microlesion effect with an increase in dyskinesias. Many DBS centers favour a levodopa monotherapy during the first weeks of DBS programming because of the short half-life which allows better adjustments in response to the DBS effect. A mean reduction of 60 % of the levodopa equivalent dosage can be observed in STN DBS with complete withdrawal in 10 to 30 %. If the DBS effects consolidate and the need for further dopaminergic medication can be better evaluated dopamine agonists can be initiated and adjusted following the same principles as in PD patients without DBS. Apart from the effects on motor symptoms the effects of dopaminergic medication on non-motor symptoms have to be evaluated. Most notably, apathy and anhedonia/depression have been associated with a treatable dopaminergic deficit (Thobois et al., 2010; Czernecki et al., 2008). The goal of DBS and medication adjustment, therefore, should be optimal control of both, motor as well as non-motor symptoms but not a drastic reduction or even withdrawal of dopaminergic medication.

2.4 Management of symptom deterioration

DBS of neither target prevents further disease progression (Hilker et al., 2005). All patients with successful DBS therapy will experience worsening of symptoms and development of new symptoms during the following years.

In case of acute onset of symptoms a technical dysfunction of the DBS system should be excluded. This check should include determination of battery function and a control of the impedances to detect a possible lead fracture/disconnection or a short circuit. Battery failure or a disconnection of the active stimulation contact(s) can result in a sudden increase in PD motor symptoms. A short circuit, in contrast, can result in sudden spread of electrical current to adjacent nervous tissue like the pyramidal tract or the oculomotor nerve with consequent tetanic muscle contraction or eye deviation. A secondary dislocation of stimulation electrodes has been reported in early studies of hardware complications (Blomstedt & Hariz, 2005; Hamani & Lozano, 2006). However, with improved surgical techniques it has become very uncommon in our own experience. In the first step, test stimulation can be helpful to detect a dislocation since either no centrally elicited stimulation effects will be observed or the thresholds for stimulation effects and side-effects will have changed significantly compared to previous stimulation protocols. As a second step, X-ray and computertomography can be used to further evaluate a probable dislocation. In case of disconnections of only single contacts of an electrode or short circuits between contacts the first step should be to evaluate DBS efficacy after reprogramming. In our experience this approach often results in satisfactory symptom control, thereby avoiding surgical revision. Moreover, it is of note, that the individual leads for the 4 contacts of a quadripolar stimulation electrode cannot be sufficiently visualized radiologically so that in case of a disconnection or a short circuit of single contacts X-ray can only demonstrate abnormal loops or sharp bends as possible loci minoris resistentiae.

Battery failure requires immediate impulse generator replacement particularly in patients with STN DBS because of the often dramatic increase in PD motor symptoms and the increased demand of dopaminergic medication. In fact, sudden battery failure should be avoided by regularly checking the remaining battery capacity to anticipate the appropriate time for impulse generator replacement. It is noteworthy that a reduction of symptom control after replacement of impulse generators can be observed (Allert et al., 2009). The origin of such deterioration can be an erroneous change of stimulation parameters or an erroneous connection of the stimulation electrodes to the 2 channels of a double channel impulse generator resulting in a change of the laterality. However, even if such iatrogenic errors are avoided, some patients may need DBS reprogramming for optimal symptom control (Allert et al., 2009).

In case of a rather slow symptom worsening three possibilities have to be encountered. Firstly, DBS efficacy has deteriorated but can be improved by reprogramming (Okun et al., 2005; Moro et al., 2006). Secondly, PD medication needs to be adjusted to obtain the full therapeutic potential. Thirdly, symptom worsening is due to the natural course of the disease. Optimization of stimulation parameters is particularly important during the first months of DBS. In many patients a slow reduction of DBS efficacy after the initial programming is observed which can be compensated by increasing the stimulation amplitude. In more complex clinical situations reprogramming with changes of the active stimulation contacts can be helpful. If stimulation parameters have been optimized but symptoms continue to be worsened the response to dopaminergic medication needs to be evaluated. Particularly in the case of STN DBS dopaminergic medication may have been decreased too much.

2.5 Counseling of patients and care givers

The selection of appropriate candidates for DBS surgery is a work-up of the individual risk and benefit relationship. It is important to identify those symptoms that will most probably

respond to DBS, but also those symptoms that are likely to persist or even take a risk of deterioration. Comorbidities have to be evaluated in the same way as psychosocial risk factors. Moreover, the patient has to be informed on the mere symptomatic nature of the therapy which cannot prevent disease progression (Hilker et al., 2005).

The success of DBS as perceived by the patient strongly depends on the own expectations and it is an important goal of the pre-operative counseling to ensure that these expectations are realistic. Apart from the limitations of the prognostic benefit, complications of DBS surgery (most notably infection rate and risk of haemorrhage) should be discussed. Furthermore, the time course of stabilization should be outlined. Although many patients experience significant improvements within a short time after surgery, the stabilization period for motor control and side effects can take 3 to 6 months. Particularly after STN DBS the possibility of behavioural and psychiatric changes has to be discussed with patients and care givers. Hypomania is often rather perceived and complained about by family and friends than by the patient. Important decisions on social and financial issues, e. g. divorce, selling or buying expensive goods etc. should not be planned shortly after DBS surgery.

Another issue to be discussed before surgery are limitations to drive a motor vehicle. Depending on the country's legal regulations driving may be restricted for a certain time period because of the neurosurgical intervention itself. Apart from that, the ability to drive a motor vehicle should be evaluated during the stabilization period after DBS surgery by examining both significant motor impairments as well as cognitive/behavioural limitations.

After surgery patients with DBS and their care givers require counseling on how to behave in daily life. Issues of concern are limitations for medical diagnostics and therapies as described in 2.6. Furthermore, the risk of interference of DBS with environmental electromagnetic waves has to be discussed. The main models of impulse generators in the 90ies had a magnetic reed switch that could not be switched off. Strong electromagnetic waves for example from an electric drill or other sources could result in sudden discontinuation of DBS with reoccurrence of motor symptoms. In newer generations of impulse generator a magnetic reed switch has been omitted or can be telemetrically disabled by the clinician programmer. The risk of interference with environmental electromagnetic waves in these impulse generators appears to be very low.

A handheld patient programmer is an optional device to have limited control of impulse generator functions. In our experience many but not all DBS centers provide patients with such a patient programmer to enable them on the one hand to make sure that DBS is on in case of symptom worsening and on the other hand to allow them checking the impulse generator battery. In some patients the patient programmer may even be helpful to further adjust and optimize stimulation parameters within predefined limits. In any case, the patient and/or patient care giver requires sufficient and often repeated education on the use of such a patient programmer. In fact, if the patient programmer is not used in daily life there is a significant risk of erroneous use which can for example result in accidentally switching off DBS.

Another area of concern for patients is limitations for physical activities particularly sports. Damage of either the stimulation electrode or the extension lead seems feasible if they are hit by high energy so that activities with a likelihood of such events should be avoided. Falls also seem to bear a risk. However, in our own experience technical damage related to physical activity or falls is exceptionally rare although patients may ask for a confirmation of normal function after such events. Heat, exposure to sunlight or the use of a sauna do not

bear a particular risk for patients with DBS. In summary, physical activities are not substantially restricted in patients with DBS.

2.6 Implications of DBS on medical diagnostics/therapies

Chronic DBS has implications for several medical diagnostics and therapies. A surface electrocardiogram (ECG) can show electrical artefacts particularly in the monopolar stimulation mode when the impulse generator serves as the anode (Martin et al., 2003). The artefact can be reduced or prevented by either switching off DBS during the ECG or by programming a bipolar stimulation mode when both cathode and anode are located intracranially (Frysinger et al., 2006). The latter also allows for longer ECG monitoring like ambulatory 24 hours Holter ECG.

Similarly, DBS artefacts can interfere with other electrodiagnostics like electroencephalography (EEG), electromyography and evaluation of evoked potentials. If DBS cannot be switched off during the examination, a bipolar DBS mode is advised to reduce or eliminate these artefacts. The use of transcranial magnetic stimulation (TMS) has not been sufficiently investigated. In vitro experiments indicate that TMS can induce currents of charge densities that can induce tissue damage so that routine application should be avoided (Shimojima et al., 2010; Deng et al., 2010).

A cardiac pacemaker or defibrillator is not a contraindication for DBS. A major concern is that the high frequency stimuli of the impulse generator are sensed by the pacemaker and interfere with proper function. Therefore, a bipolar DBS mode is recommended and has been found safe in such patients (Capelle et al., 2005).

X-ray examinations including computertomography (CT) can be safely performed in patients with DBS. In contrast, magnetic resonance imaging (MRI) should only be performed within important limitations. A full body radiofrequency coil cannot be used in patients with DBS because of the risk of electrode heating and tissue damage (Henderson et al., 2005). The use of head transmit coils has been found to be safe and helpful by many groups (Chhabra et al., 2010; Nazarro et al., 2010, Tagliati et al., 2009) but is only recommended according to the manufacturer's guidelines in experienced centers. One study of the effects of 1.5 Tesla MRI in 570 patients did not report any local cutaneous nor neurological disorders during or after the MRI. Moreover, no change of the impulse generator settings occurred in impulse generators without magnetic reed switch or when the magnetic reed switch remained disabled during the procedure (Fraix et al., 2010).

Diathermy (e.g. shortwave diathermy, microwave diathermy or therapeutic ultrasound diathermy) is contraindicated because it can result in tissue damage or damage of parts of the neurostimulation system (Nutt et al., 2001).

3. Conclusion

DBS has become an important therapeutic option for PD patients. DBS is a symptomatic treatment of motor complications requiring not only expertise for the precise implantation of stimulation electrodes but also for the post-operative patient management. Optimal stimulation parameters are determined by clinical evaluation of short and long-term responses. Apart from motor symptoms, stimulation induced non motor symptoms have to be observed, most notably psychiatric and behavioural changes. Similarly, medication has to be adjusted in response to the DBS motor and non-motor effects. Impedance measurements

along with the clinical evaluation of stimulation effects are helpful to evaluate proper technical function. Regular follow-up visits are recommended to ensure optimal DBS efficacy. Impulse generators should be replaced before complete battery failure to prevent sudden recurrence of motor symptoms particularly in the case of STN DBS. DBS systems have implications on other medical therapies and diagnostics. Most notably, MRI with full body radiofrequency coils and diathermy are contraindicated because of possible tissue damage or damage of parts of the DBS system.

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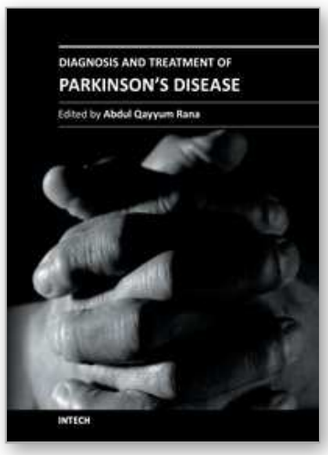
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Parkinson's disease is diagnosed by history and physical examination and there are no laboratory investigations available to aid the diagnosis of Parkinson's disease. Confirmation of diagnosis of Parkinson's disease thus remains a difficulty. This book brings forth an update of most recent developments made in terms of biomarkers and various imaging techniques with potential use for diagnosing Parkinson's disease. A detailed discussion about the differential diagnosis of Parkinson's disease also follows as Parkinson's disease may be difficult to differentiate from other mimicking conditions at times. As Parkinson's disease affects many systems of human body, a multimodality treatment of this condition is necessary to improve the quality of life of patients. This book provides detailed information on the currently available variety of treatments for Parkinson's disease including pharmacotherapy, physical therapy and surgical treatments of Parkinson's disease. Postoperative care of patients of Parkinson's disease has also been discussed in an organized manner in this text. Clinicians dealing with day to day problems caused by Parkinson's disease as well as other healthcare workers can use beneficial treatment outlines provided in this book.

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University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
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InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

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