Review Article

Effect of Deep Brain Stimulation on Speech Performance in Parkinson’s Disease

Sabine Skodda

Department of Neurology, Knappschaftskrankenhaus, Ruhr University Bochum, In der Schornau 23-25, 44892 Bochum, Germany

Correspondence should be addressed to Sabine Skodda, sabine@skodda.de

Received 14 July 2012; Accepted 17 October 2012

Academic Editor: Jan O. Aasly

Copyright © 2012 Sabine Skodda. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Deep brain stimulation (DBS) has been reported to be successful in relieving the core motor symptoms of Parkinson’s disease (PD) and motor fluctuations in the more advanced stages of the disease. However, data on the effects of DBS on speech performance are inconsistent. While there are some series of patients documenting that speech function was relatively unaffected by DBS of the nucleus subthalamicus (STN), other investigators reported on improvements of distinct parameters of oral control and voice. Though, these ameliorations of single speech modalities were not always accompanied by an improvement of overall speech intelligibility. On the other hand, there are also indications for an induction of dysarthria as an adverse effect of STN-DBS occurring at least in some patients with PD. Since a deterioration of speech function has more often been observed under high stimulation amplitudes, this phenomenon has been ascribed to a spread of current-to-adjacent pathways which might also be the reason for the sporadic observation of an onset of dysarthria under DBS of other basal ganglia targets (e.g., globus pallidus internus/GPi or thalamus/Vim). The aim of this paper is to review and evaluate reports in the literature on the effects of DBS on speech function in PD.

1. Introduction

1.1. Dysarthria in Parkinson’s Disease (PD). Nearly 90% of individuals with Parkinson’s disease (PD) develop voice and speech disorders (dysarthria) in the course of their disease [1]. Affected patients may complain about a quiet or weak voice and about difficulties to get speech started. Further, they often report that they are asked to repeat their words because listeners have difficulties to understand although patients themselves may self-estimate their speech as loud and sufficiently articulated [2]. Dysarthria can emerge at any stage of the disease and worsen in the later stages [3] causing a progressive loss of communication and leading to social isolation. Parkinsonian dysarthria has traditionally been interpreted as manifestation of rigor and hypokinesia on the speech effector organs [4] inducing to a multidimensional motor speech impairment including alterations of speech respiration, phonation, articulation, and prosody. Thus, based upon global clinical impression, hypokinetic dysarthria is characterized by a breathy and harsh voice, monotony of pitch and loudness, reduced stress, variable speech rate with short rushes of speech, and imprecise articulation resulting in a reduction of overall speech intelligibility [5–8]. From the therapeutic point of view, the effect of dopaminergic medication on different speech parameters and overall speech intelligibility in particular remains somewhat inconclusive. There are some reports of positive levodopa effects on tongue strength and endurance and of an improvement of speech intelligibility assessed by perceptual analysis in PD patients [9–11]. However, the majority of studies found no relevant effect of dopaminergic therapy on speech rate [12, 13], prosodic and phonatory parameters [14, 15], and overall intelligibility [16–18]. Nonpharmacological treatment strategies such as repetitive transcranial magnetic stimulation and laryngeal collagen augmentation injections seem to offer some positive, albeit transient, effects on voice and speech impairment in PD; however, the interpretation of data is limited by the very small number of so far treated patients [19, 20]. Up till now, behavioral speech therapy with special emphasis on rescaling
the reduced amplitude of speech motor output is considered as the most effective therapeutic approach but is often found to be unsatisfying in a subgroup of patients [21, 22].

1.2. Effects of Lesional Surgical Treatment on Speech in PD. Before the rise of dopamine therapy, functional neurosurgery procedures, such as thalamotomy and pallidotomy, were used to treat symptoms of PD. Thalamotomy was generally performed in the ventrolateral and ventrointermediate nuclei of the thalamus to improve Parkinsonian tremor. Unilateral thalamotomy had been found to worsen speech independent if the lesion was in the dominant or nondominant hemisphere [23, 24]. However, there are also reports of neutral outcomes for speech following thalamotomy [25]. Bilateral thalamotomy had been associated with word blocking, slow speech and hypophonia, and a persistent worsening of dysarthria, some of the patients developed palilalia [26–28]. Because of these serious adverse events on Parkinsonian speech, bilateral thalamotomy has been abandoned for the treatment of PD.

Pallidotomy usually involved lesions of the posteroverentral portion of the internal part of the globus pallidus and was used to alleviate Parkinsonian symptoms and reduce contralateral dyskinesias [29]. Concerning speech function, the majority of studies found no effect of pallidotomy on hypokinetic dysarthria [30–33]. Though, some studies describe positive changes of labial force production and stability in a subgroup of patients [34] and an improvement of phonatory and articulatory measurements in PD speakers after unilateral and bilateral pallidotomy [35, 36]. On the other hand, others report on a worsening of speech function with development of transient dysarthria, facial weakness, swallowing problems, and alterations in verbal fluency [37]. In summary, the current literature about the effects of ablative surgical procedures on motor speech function in PD remains equivocal; investigations conducted in the early stereotactic era at least suggested that least bilateral thalamotomy was most likely to result in negative speech outcomes [38].

1.3. Effects of Deep Brain Stimulation on Speech in PD. In the last years, numerous studies have proven the beneficial effects of high-frequency deep brain stimulation (DBS) of the subthalamic nucleus (STN), globus pallidus internus (GPI), and the ventral intermediate nucleus of the thalamus (Vim) on motor symptoms in PD [39–43]. However, the effects of DBS on voice and speech have been found to be variable or even adverse. According to the speech item of the Unified Parkinson’s Disease Rating Scale (UPDRS), the prevalence of dysarthria under STN-DBS has been reported to vary between 1% after 6 months up to 70% at three-year followup with an average of 9.3% [44–46]. On the other hand, there are also reports of an amelioration of distinctive parameters of voice and nonspeech articulatory measures in individual PD patients under STN-DBS (e.g., [47]). In a similar vein, studies on speech performance under DBS of targets different from STN have produced heterogeneous results. Under GPI-DBS, overall speech performance based upon perceptual rating showed an improvement in a small series of seven patients [48], whereas worsening of speech intelligibility has been observed in other studies [49–51]. Similarly, Vim-DBS has been reported to have a worsening effect on perceptual assessment and electrophysiological outcome parameters of speech in patients with tremor-dominant PD as well as in patients treated with Vim-DBS for essential tremor [52–56].

Based upon these observations, growing interest has been focused on the impact of DBS on speech in PD, and numerous subsequent investigations with more subtle analyses of overall speech performance and of distinctive speech parameters have been conducted to gain a better understanding of the mechanisms why and how DBS can induce alterations of voice and speech in some PD patients.

The aim of the current paper is to review and discuss the existing studies on voice and speech performance in PD as a basis for a better information and management of patients.

2. Methods

A Medline literature search were undertaken including articles published until September 2012 using the search terms “Parkinson’s disease/PD” and “deep brain stimulation/DBS” and “dysarthria” and/or “speech” and/or “voice”. The search results were narrowed down to investigations focused on voice and speech performance under DBS based upon qualitative description or perceptual, acoustic or electrophysiological analyses, since it has been noticed that the UPDRS speech item alone shows poor sensitivity to detect speech problems [57]. Furthermore, the reference lists of the chosen articles were checked for additional publications fulfilling these criteria.

3. Results

A number of 35 publications were identified with numbers of participants ranging from one (case reports) up to 57 ([56, 58–91], see Table 1). The great majority of data (n = 34) were derived from patients under STN-DBS; three studies compared speech performance under STN-DBS with DBS of the caudal zona incerta (cZi), and there were three further investigations on STN-GPi and STN-Vim. Concerning methods, studies differed considerably with respect to the participants’ characteristics (disease duration, dosage of concomitant medication, time period after DBS surgery, etc.), underlying speech tasks (sustained phonation, syllable production, word or sentence reading, free monologue, and performance of nonspeech movements of the articulatory muscles), and the kind of analysis (extensive perceptual assessments, acoustic analysis of different sets of speech parameters, and electrophysiological measurements of articulatory and phonatory function) which limits the direct comparability.

Therefore, the main findings of the studies are described and discussed in the following sections.

3.1. Impact on STN-DBS on Voice and Speech. Since over the last decade, the favoured DBS target for the treatment
| Sample size | Target (all studies with stimulation on versus stimulation off) | Outcome measure | Results | R |
|------------|---------------------------------------------------------------|----------------|---------|---|
| 16 | STN ($n=8$), cZi ($n=8$); pre-op and 12 months post-op | Phonatory control: alternations between voicing and voiceless states in a reading task | Progressive deterioration of phonatory control but unaffected by DBS | [58] |
| 6 | STN | Perceptual ratings and acoustic analysis | Deterioration of overall speech performance (perceptual ratings); mixed results concerning single speech parameters (acoustic analysis) | [59] |
| 16 | STN ($n=8$), cZi ($n=8$); pre-op and 12 months post-op | Mean intensity during reading, intensity decay during syllable repetition | STN: increase of intensity; cZi: slight reduction of intensity during reading; no significant change of intensity decay | [60] |
| 14 | STN ($n=7$), cZi ($n=7$) | Articulatory capacity, accuracy of plosive consonants | STN: increased articulatory rate; cZi: deterioration of articulatory rate and quality | [61] |
| 10 | STN | Acoustic analysis of different speech parameters | Mixed results with positive and negative effects on single speech parameters | [62] |
| 14 | STN | Acoustic analysis of syllable repetition precision | Deterioration of syllable repetition capacity | [63] |
| 57 | (I) $n=36$ with both electrodes within STN, (II) $n=16$ with only one electrode within STN, (III) $n=5$ with no electrode within STN | Perceptual rating according to UPDRS speech item | (I) 50% improvement, 36% deterioration of speech; (II) 69% improvement, 25% deterioration; (III) 100% deterioration | [64] |
| 27 | STN and GPi; pre-op and 6 months post-op | Peak velocities of jaw movements | STN: deterioration of jaw movement velocity; GPi: no deterioration | [65] |
| 11 | STN; stimulation frequencies with 60 Hz and 130 Hz | Perceptual rating, acoustic analysis, and aerodynamic measurements | Amelioration of outcome measures under 60 Hz stimulation | [66] |
| 2 | STN | Case reports: description of speech performance | Reoccurrence of severe stuttering | [67] |
| 11 | STN | Acoustic analysis of different speech tasks | No changes in speech performance | [68] |
| 2 | STN | Articulatory accuracy measured by electropalatography | Deterioration in one patient, amelioration in the other patient | [69] |
| 32 | STN; pre-op, 1 month, 6 months, and 12 months post-op | Perceptual analysis based upon validated rating scale | Amelioration of speech in $n=7$; deterioration in the other patients by an average of 14.2% ± 20.15% off-medication; deterioration more often with high voltage and medially located electrodes within the left STN | [70] |
| 17 | STN | Aerodynamic measures | Increased intraoral pressure in $n=7$; increased velopharyngeal closure in $n=5$ | [71] |
| 9 | STN | Perceptual and acoustic analysis | Amelioration of voice, no influence on fluency | [72] |
| 18 | STN | Aerodynamic measures | Increased inspiratory driving pressure ($n=9$); increased vocal fold closure ($n=9$); more benefit from low-frequency stimulation | [73] |
| 10 | STN; 4 V (high) and 2 V (low) voltage | Perceptual analysis based upon validated rating scale | Deterioration during high-amplitude stimulation in $n=6$, in patients with electrodes in medial and/or posterior position | [74] |
| 14 | within and above STN; 4 V (high) and 2 V (low) voltage | Perceptual analysis based upon validated rating scale | Deterioration of speech during high-amplitude stimulation independently from side of stimulation | [75] |
| 19 | STN | Acoustic analysis of sustained vowel phonation | Improvement of single measures of voice | [76] |
| 19 | STN | Perceptual ratings by patient, physician, and professional speech therapist, additional acoustic analysis | Deterioration of overall speech performance (perceptual ratings); amelioration of single speech/voice parameters (acoustic analysis) | [77] |
Table 1: Continued.

| Sample size | Target (all studies with stimulation on versus stimulation off) | Outcome measure | Results | R |
|-------------|---------------------------------------------------------------|-----------------|----------|---|
| n = 12 STN  | Perceptual rating and acoustic analysis                      | No deterioration; amelioration of glottal stability and vocal tremor | [78]    |
| n = 9 STN   | Acoustic analysis of articulatory and phonatory function      | Mixed results with improvement or deterioration of articulation and phonation in different patients | [79]    |
| n = 10 STN, different parameter settings | Perceptual analysis of different speech tasks | Deterioration in n = 4 patients under “normal” stimulation parameters; further deterioration during high-amplitude and/or frequency stimulation | [80]    |
| n = 4 STN   | Qualitative description and acoustic analysis                 | Mixed results with improvement and deterioration of speech in the different patients | [81]    |
| n = 10 STN  | Perceptual rating according to UPDRS speech item, PET study  | Improvement of overall speech performance | [82]    |
| n = 7 STN; pre-op and 3 months post-op | Perceptual dysarthria assessment and rating according to UPDRS speech item | Modest improvement of lip movements, loudness, and pitch; slight reduction of intelligibility | [83]    |
| n = 16 STN  | Acoustic analysis and force measurements of articulatory muscles | Decrease of reaction and movement time of articulatory organs; increase of maximal strength and precision; improvement of respiratory and phonatory function | [84]    |
| n = 26 STN; follow up at several years post-op | Perceptual ratings, measurement of articulatory force (lip and tongue force) | Improvement of articulatory force; deterioration of overall speech performance (perceptually rated) in a subgroup of patients | [85]    |
| n = 1 STN   | Case report: descriptive                                     | Emergence of dysfluencies under STN-DBS | [86]    |
| n = 26 STN  | Acoustic analysis                                           | Improvement of distinctive speech parameters | [87]    |
| n = 7 STN; pre-op and 3 months and 6 months post-op | Acoustic analysis | Mild improvement of sound pressure level and pitch variability | [88]    |
| n = 14 STN (n = 7), Vim (n = 7) | Measurement of articulatory force (lip and tongue force) | Vim: deterioration of static and dynamic control of articulatory organs; STN: improvement | [89]    |
| n = 1 STN, pre-op and 2 years post-op | Descriptive | Improvement of oral control and intelligibility | [90]    |
| n = 10 STN  | Perceptual rating according to UPDRS speech item, measurement of articulatory force (lip and tongue force) | Improvement of static and dynamic control of articulatory organs; improvement of reaction time; improvement of overall speech performance | [91]    |
| n = 23 Vim (patients with tremor) | Perceptual rating | Development of dysarthria in n = 7 | [52]    |

STN: Subthalamic nucleus, GPi: Globus pallidus internus, cZi: Caudal zona incerta, R: Reference.

of motor symptoms in PD was the STN, most data about a possible deterioration of speech performance are derived from patients under STN-DBS. According to UPDRS speech item ratings alone, a meta-analysis of 37 cohorts comprised of 921 patients reported an incidence of dysarthria as adverse event under STN-DBS of 9.3% [46] which is in general confirmed by other small studies (e.g., [64]). On the other hand, beneficial effects on speech performance are documented at least in individual patients although the improvement was much less pronounced than that on limb movements and tended to decrease in the long term (e.g., [82, 92, 93]). There are further studies which combine perceptual assessment of overall speech function with acoustic analysis and electrophysiological measurements which suggested that STN-DBS can improve articulatory and phonatory components such as loudness in Parkinsonian speech [77, 78, 85, 87, 88, 90, 91, 94]. For example, in one investigation, the authors found an improvement of articulatory force and overall speech function in the majority of 26 PD patients with STN-DBS using perceptual analysis and electrophysiological measurements [85]. In another study, no negative effects of STN-DBS were seen in 12 PD patients; on the contrary, some aspects of speech as vocal tremor tended to improve but without effects on global speech intelligibility [78]. Worsened overall speech performance according to perceptual ratings was seen in another study on 19 patients under STN-DBS; however, technical measures showed stimulation-induced improvements of single speech dimensions affected by the PD-specific motor disorder [77]. The authors concluded that STN-DBS could reduce designated disease-inherent dysarthrophonic symptoms, such as reduced loudness or glottic tremor, however, that these actions on speech could
be outweighed by a general dysarthrogenic effects of STN-DBS, probably based on a decline of complex (e.g., prosodic) functions [77]. Similarly, other investigators proposed that STN-DBS has a differential impact on different modalities of Parkinson's speech with the potential to ameliorate phonation, however, at the cost of a deterioration of articulatory capacities leading to a reduced overall speech intelligibility [69, 79, 94]. Furthermore, STN-DBS was reported to induce abnormalities in speed and regularity of nonspeech syllable repetition as a possible hint for a negative effect on basal motor speech performance [63].

3.2. Influence of Stimulation Parameters and Side of STN Stimulation. Other investigators intended to find correlations between parameter settings such as amplitude, frequencies, and polarity between the stimulation electrode contacts and speech performance [64, 70, 74, 75, 80, 95, 96]. Consistently, the authors reported a deterioration of dysarthria rated by perceptual assessments under high-amplitude or high-frequency stimulation settings which, however, were required for the optimization of motor performance at least in some individual patients. Likewise, the exact contact position within the STN was found to be of importance since stimulation right within the STN and especially in the medial and/or posterior portion of the nucleus was linked with poorer speech intelligibility [70, 74, 75]. In another investigation on 37 PD patients the exact positions of the STN electrodes were correlated to clinical outcomes with the result that better symptom relief with reduced need for post-op medication was expected in patients whose electrodes were accurately positioned in both STN [64]. However, even in the subgroup of 36 patients with exact electrode position in both STNs, 36% showed a deterioration of speech under stimulation compared to 50% featuring an improvement [64]. Two other studies surveyed a possible differential impact of left- and right-sided STN stimulation on different aspects of speech performance and found that selective left-sided stimulation had a profoundly negative effect on prosody, articulation, and hence, intelligibility [95, 96].

3.3. Impact of Presurgery Speech Performance and Microlesion Effects. Up till now, there are only few investigations with speech testings before and at certain time intervals after DBS surgery which would be a reasonable approach to define subgroups of PD patients who are particularly on risk to experience deterioration of speech under STN-DBS or to identify a possible microlesion effect of electrode placement [58, 60, 65, 70, 83, 88, 90]. In the largest of these studies, 32 PD patients under STN-DBS were tested pre- and postsurgically with several follow-up examinations and compared with a group of medically treated PD patients [70]. Dysarthria was rated perceptually according to the widely used assessment for the Intelligibility of Dysarthric Speech battery. As a main result, speech intelligibility deteriorated on average by 14% after 1 year of STN-DBS when the patients were off-medication/on-stimulation and by 13% in the off-medication/off-stimulation state compared to off-medication state preoperatively. Similar results were found in the on-medication/ on-stimulation state when compared to the on-medication state preoperatively (average deterioration of 17%). However, there was a substantial variability between individual patients, even with an improvement of dysarthria in 7 patients. In the medical treatment group, the decline of speech intelligibility after 1 year lays within a comparable range. The authors found a correlation of poorer speech outcome after 1 year and a higher presurgical general motor impairment in the on-medication condition, probably explained by the presence of nondopaminergic pathology. Furthermore, high voltage stimulation of locally located electrodes on the left STN was found to be associated with a significantly higher risk of speech deterioration [70]. Another study on 7 patients found no consistent effects of DBS surgery alone (i.e., no hint for microlesion effect) and no consistent stimulation effect on speech under STN-DBS after three months but a slight improvement of pitch variability and sound pressure levels under stimulation six month post-op [88]. Another two studies with PD patients tested before and 12 months after DBS surgery in the stimulation off-condition provided evidence for a progressive reduction of phonatory control, but not of speech intensity, which was interpreted as either progression of the disease, an effect of reduced post-op levodopa dosage, or a microlesion effect [58, 60].

3.4. Summary

3.4.1. Impact on STN-DBS on Speech and Possible Mechanisms. As a first recapitulation of these data, the impact of STN-DBS on speech performance can be variable, and the available data still do not allow predicting the risk of the onset or deterioration of dysarthria in the individual patient. STN-DBS seems to have some potentials to ameliorate at least phonatory dysfunctions as voice tremor and reduced loudness; however, these beneficial effects might be counterbalanced by a prodysarthrogenic actions whose mechanisms are not yet fully understood. Since several studies document an association of dysarthria with higher voltage/frequency STN stimulation, one might assume a spread of current to the corticobulbar pathways for laryngeal motor control with an induction of a spastic/pseudobulbar dysarthria [93]. However, this proposed mechanism should not only induce a deterioration of connected speech performance, but also of other vocalizations as sustained phonation which has not been found in the previous investigations (e.g., [63, 75]). Current spread into other pathways, namely, the pallid fugal and cerebellothalamic fibers seem to more adequately account for speech impairment, especially in patients with electrodes placed within the medial portion of the STN [97]. Furthermore, the optimal implantation position of the electrodes is typically chosen on the basis of limb motor effects of stimulation disregarding the possibility that STN could have a different role or somatotopy for speech and body motor control. This assumption is corroborated by a positron emission study on PD patients.
which could demonstrate different patterns of activation with speech production and hand movements which were differentially modulated by STN-DBS [82]. Additionally, one might assume a further microlesional effect induced by the electrode insertion itself which could induce an earlier decompensation of the already dysfunctional speech system in the course of PD [58, 70]. This hypothesis would at least account for the finding that PD patients with higher presurgical global motor impairment are on higher risk to develop speech problems within the first year under STN-DBS, even in the off-stimulation condition [70]. Another possible factor could be the reduction of dopaminergic medication under STN-DBS since one could assume that a certain amount of medication could still be required to ensure a satisfying speech performance. However, although the available data are somewhat inconsistent, dopaminergic medication has been estimated to have at best limited effects on speech performance, and deterioration of dysarthria in the course of PD rather seems to reflect nondopaminergic dysfunction [98]. Therefore, it is not likely that a deficit of dopaminergic medication relevantly accounts for the observed speech abnormalities under STN-DBS, the more so, since indeed for some patients, speech was reported to be worse on-medication/on-stimulation [70, 93, 99].

Summarized, since there are no established algorithms for the prediction of the impact of STN-DBS on speech for the individual patient uptill now, neurologists have to carefully keep in mind the possibility of speech deterioration and to inform the patient accordingly, when the indication of STN-DBS is discussed. Patients who are suffering from symptoms unresponsive to dopaminergic therapy (with higher motor impairment in the best medication on state) should be aware of the possibility of a detrimental effect of STN-DBS on speech, and a preexisting severe Parkinsonian dysarthria cannot be the main indication for STN-DBS.

In the postsurgical management of PD patients under STN-DBS, patients should be carefully monitored concerning speech function. If speech performance shows a relevant deterioration in the on-stimulation condition, a meticulous testing and adjustment of electrode contact sides and stimulation parameters can be helpful to achieve a clinically optimal balance between satisfactory motor function and intelligibility of speech in the individual patient. In some cases, however, it can be necessary that the patient himself can vary the stimulation parameters within a certain preset range, for example, reduce the stimulation amplitude for a better speech performance during longer conversations. If these strategies remain disappointing, speech therapy should be provided betimes, wherein best evidence has been documented for Lee Silverman Voice Treatment (LSVT) which has shown to be effective at least in a subgroup of patients with impaired speech intelligibility under STN-DBS [100, 101].

3.4.2. Impact of DBS of GPi and cZi on Speech. In contrast, effects of DBS of the GPi on speech performance have only scarcely investigated so far. In one study including 27 PD patients velocity of externally scaled jaw movements was found to be significantly reduced under STN-DBS, but not under GPi-DBS, leading to the authors’ recommendation to consider the GPi at the preferable target for PD patients with preexisting oromandibular dysfunction [65]. In large controlled trials, speech performance has mostly been assessed by item 18 of the UPDRS Motor Scale, which shows poor sensitivity to detecting speech problems and indeed identified only 38% of patients with speech deterioration in one study [57, 70]. At least according to the UPDRS speech item, the rate of dysarthria as an adverse event seems to occur less often under GPi-DBS than under STN-DBS [42, 102, 103].

Recently, DBS of the caudal zona incerta (cZi) has been compared concerning its effect on speech performance in comparison to STN-DBS in small groups consisting of 7 to 8 patients [58, 60, 61]. Results showed a differential impact of cZi-STN on different measures of speech, whereas phonatory control remained unaffected by cZi-STN (and STN-DBS), patients showed a small but significant reduction of speech intensity and a decrease in articulation rate and quality [58, 60, 61].

The ventral intermediate nucleus of the thalamus (Vim) is an established target for the treatment of medically intractable tremor syndromes of different etiologies. Since Vim-DBS cannot alleviate the other motor manifestations of PD, thalamic stimulation is only used as an individual option in exceptional tremor-dominant PD cases. Therefore, no systematic studies on the impact of Vim-DBS on Parkinsonian speech are available. However, there are reports on an induction of dysarthria under Vim-STN interpreted as being induced by the spread of current-into-adjacent pathways [52, 104, 105].

4. Conclusion

DBS has been proven to be an effective treatment for PD patients with refractory tremor or motor fluctuations, but its impact on speech can be variable, and deterioration of speech intelligibility can counterbalance the motor benefits of the procedure. Uptill now, the mechanisms responsible for a worsening of Parkinsonian dysarthria under STN-DBS are not fully understood, but it is plausible to assume a combination of preexisting hypokinetic dysarthria as a manifestation of progressive and nondopaminergic dysfunction with microlesion- and stimulation-induced effects as spreading of current-into-adjacent pathways. According to very few and preliminary data, speech function seems to be less compromised under GPi-DBS than under STN-DBS, but this first impression demands further corroboration. Consecutive studies with large numbers of patients are warranted which refine and further develop the previous investigations (e.g., [70, 85, 106]) including patients at presurgical and defined follow-up intervals under DBS with subtle speech investigations (ideally consisting of perceptual ratings of overall speech intelligibility in combination with objective acoustic analysis and/or electrophysiological testings) in on- and off-stimulation conditions. The aim of these studies should be to gain a better understanding of the underlying
pathophysiology to identify patients who are at risk to develop speech deterioration under STN-DBS. Furthermore, investigations on the effects of GPi- and cZi-STN on speech performance are necessary to decide which target is most appropriate in the individual PD patient for best motor and speech performance.

References

[1] A. K. Ho, R. Iansek, C. Marigliani, J. L. Bradshaw, and S. Gates, “Speech impairment in a large sample of patients with Parkinson’s disease,” Behavioural Neurology, vol. 11, no. 3, pp. 131–137, 1998.
[2] J. R. Duffy, “Hypokinetic dysarthria,” in Motor Speech Disorders, J. R. Duffy, Ed., pp. 187–215, Elsevier, St. Louis, Mo, USA, 1995.
[3] W. J. Mutch, A. Strudwick, S. K. Roy, and A. W. Downie, “Parkinson’s disease: disability, review, and management,” British Medical Journal, vol. 293, no. 6548, pp. 675–677, 1986.
[4] K. K. Baker, L. O. Ramig, E. S. Luschei, and M. E. Smith, “Thyroarytenoid muscle activity associated with hypophonia in Parkinson’s disease and aging,” Neurology, vol. 51, no. 6, pp. 1592–1598, 1998.
[5] F. L. Darley, A. E. Aronson, and J. R. Brown, “Clusters of deviant speech dimensions in the dysarthrias,” Journal of Speech and Hearing Research, vol. 12, no. 3, pp. 462–496, 1969.
[6] F. L. Darley, A. E. Aronson, and J. R. Brown, “Differential diagnostic patterns of dysarthria,” Journal of Speech and Hearing Research, vol. 12, no. 2, pp. 246–269, 1969.
[7] G. J. Canter, “Speech characteristics of patients with Parkinson’s disease. I: Intensity, pitch, and duration,” The Journal of Speech and Hearing Disorders, vol. 28, pp. 221–229, 1963.
[8] G. J. Canter, “Speech characteristics of patients with Parkinson’s disease: II. Physiological support for speech,” The Journal of Speech and Hearing Disorders, vol. 30, pp. 44–49, 1965.
[9] M. De Letter, P. Santens, and J. Van Borsel, “The effects of levodopa on tongue strength and endurance in patients with Parkinson’s disease,” Acta Neurologica Belgica, vol. 103, no. 1, pp. 35–38, 2003.
[10] M. De Letter, P. Santens, and J. Van Borsel, “The effects of levodopa on word intelligibility in Parkinson’s disease,” Journal of Communication Disorders, vol. 38, no. 3, pp. 187–196, 2005.
[11] M. De Letter, P. Santens, M. De Bodt, G. Van Maele, J. Van Borsel, and P. Boon, “The effect of levodopa on respiration and word intelligibility in people with advanced Parkinson’s disease,” Clinical Neurology and Neurosurgery, vol. 109, no. 6, pp. 495–500, 2007.
[12] M. De Letter, P. Santens, M. De Bodt, P. Boon, and J. Van Borsel, “Levodopa-induced alterations in speech rate in advanced Parkinson’s disease,” Acta Neurologica Belgica, vol. 106, no. 1, pp. 19–22, 2006.
[13] A. K. Ho, J. L. Bradshaw, and R. Iansek, “For better or worse: the effect of Levodopa on speech in Parkinson’s disease,” Movement Disorders, vol. 23, no. 4, pp. 574–580, 2008.
[14] J. Gamboa, F. J. Jiménez-Jiménez, A. Nieto et al., “Acoustic voice analysis in patients with Parkinson’s disease treated with dopaminergic drugs,” Journal of Voice, vol. 11, no. 3, pp. 314–320, 1997.
[15] A. M. Goberman and C. Coelho, “Acoustic analysis of Parkinsonian speech I: speech characteristics and L-Dopa therapy,” NeuroRehabilitation, vol. 17, no. 3, pp. 237–246, 2002.
[16] K. K. Baker, L. O. Ramig, A. B. Johnson, and C. R. Freed, “Preliminary voice and speech analysis following fetal dopamine transplants in 5 individuals with Parkinson disease,” Journal of Speech, Language, and Hearing Research, vol. 40, no. 3, pp. 613–626, 1997.
[17] K. Kompoliti, Q. E. Wang, C. G. Goetz, S. Leurgans, and R. Raman, “Effects of central dopaminergic stimulation by apomorphine on speech in Parkinson’s disease,” Neurology, vol. 54, no. 2, pp. 458–462, 2000.
[18] S. Skodda, W. Visser, and U. Schlegel, “Short- and long-term dopaminergic effects on dysarthria in early Parkinson’s disease,” Journal of Neural Transmission, vol. 117, no. 2, pp. 197–205, 2010.
[19] A. E. Dias, E. R. Barbosa, K. Coracini, F. Maia, M. A. Marcolin, and F. Fregni, “Effects of repetitive transcranial magnetic stimulation on voice and speech in Parkinson’s disease,” Acta Neuroligica Scandinavica, vol. 113, no. 2, pp. 92–99, 2006.
[20] G. S. Berke, B. Gerratt, J. Kreiman, and K. Jackson, “Treatment of Parkinson hypophonia with percutaneous collagen augmentation,” Laryngoscope, vol. 109, no. 8, pp. 1295–1299, 1999.
[21] S. Sapir, L. O. Ramig, and C. M. Fox, “Intensive voice treatment in Parkinson’s disease: lee silverman voice treatment,” Expert Review of Neurotherapeutics, vol. 11, no. 6, pp. 815–830, 2011.
[22] G. Ramsay, “Physical, occupational, speech and swallowing therapies and physical exercise in Parkinson’s disease,” Journal of Neural Transmission, vol. 118, no. 5, pp. 773–781, 2011.
[23] D. S. Bell, “Speech functions of the thalamus inferred from the effects of thalamotomy,” Brain, vol. 91, no. 4, pp. 619–638, 1968.
[24] J. N. Petrovici, “Speech disturbances following stereotaxic surgery in ventrolateral thalamus,” Neurosurgical Review, vol. 3, no. 3, pp. 189–195, 1980.
[25] M. N. Linhares and R. R. Tasker, “Microelectrode-guided thalamotomy for Parkinson’s disease,” Neurosurgery, vol. 46, no. 2, pp. 390–398, 2000.
[26] G. J. Canter and D. R. Van Lancker, “Disturbances of the temporal organization of speech following bilateral thalamic surgery in a patient with Parkinson’s disease,” Journal of Communication Disorders, vol. 18, no. 5, pp. 329–349, 1985.
[27] A. Stracciari, M. Guarino, F. Cirignotta, and P. Pazzaglia, “Development of palilalia after stereotaxic thalamotomy in Parkinson’s disease,” European Neurology, vol. 33, no. 3, pp. 275–276, 1993.
[28] R. R. Tasker, J. Siqueira, P. Hawrylyshyn, and L. W. Organ, “What happened to VIM thalamotomy for Parkinson’s disease?” Applied Neurophysiology, vol. 46, no. 1–4, pp. 68–83, 1983.
[29] L. V. Laitinen, A. T. Bergenheim, and M. I. Hariz, “Ventroneuroseptolateral pallidotomy can abolish all parkinsonian symptoms,” Stereotactic and Functional Neurosurgery, vol. 58, no. 1–4, pp. 14–21, 1992.
[30] J. F. Buck and L. S. Capper, “Speech problems in parkinsonian patients undergoing anterior choroidal artery occlusion or chemopallidotomy,” Journal of the American Geriatrics Society, vol. 4, no. 12, pp. 1285–1290, 1956.
[31] J. Ghika, F. Ghika-Schmid, H. Fankhauser et al., “Bilateral contemporaneous posteroventral pallidotomy for the treatment of Parkinson’s disease: neuropsychological and neurological side effects: report of four cases and review of Parkinson’s Disease
the literature,” *Journal of Neurosurgery*, vol. 91, no. 2, pp. 313–321, 1999.

32. A. E. Lang, A. M. Lozano, E. Montgomery, J. Duff, R. Tasker, and W. Hutchins, “Posteroventral medial pallidotomy in advanced Parkinson’s disease,” *The New England Journal of Medicine*, vol. 337, no. 15, pp. 1036–1042, 1997.

33. R. Scott, R. Gregory, N. Hines et al., “Neuropsychological, neurological and functional outcome following pallidotomy for Parkinson’s disease. A consecutive series of eight simultaneous bilateral and twelve unilateral procedures,” *Brain*, vol. 121, no. 4, pp. 659–675, 1998.

34. S. M. Barlow, R. P. Iacono, L. A. Paseman, A. Biswas, and L. D’Antonio, “The effects of posteroventral pallidotomy on force and speech aerodynamics in Parkinson’s disease,” in *Neuromotor Speech Disorders: Nature Assessment and Management*, M. Cannon, K. M. Vorston, and D. R. Beukelman, Eds., pp. 117–155, Brooks, Baltimore, Md, USA, 1998.

35. G. M. Schulz, T. Peterson, C. M. Sapienza, M. Greer, and W. Friedman, “Voice and speech characteristics of persons with Parkinson’s disease pre-and post-pallidotomy surgery: preliminary findings,” *Journal of Speech, Language, and Hearing Research*, vol. 42, no. 5, pp. 1176–1194, 1999.

36. G. M. Schulz, M. Greer, and W. Friedman, “Changes in vocal intensity in Parkinson’s disease following pallidotomy surgery,” *Journal of Voice*, vol. 14, no. 4, pp. 589–606, 2000.

37. A. I. Tröster, S. P. Woods, J. A. Fields, C. Hanisch, and W. W. Beatty, “Declines in switching underlie verbal fluency changes after unilateral pallidal surgery in Parkinson’s disease,” *Brain and Cognition*, vol. 50, no. 2, pp. 207–217, 2002.

38. B. E. Murdoch, “Surgical approaches to treatment of Parkinson’s disease: implications for speech function,” *International Journal of Speech-Language Pathology*, vol. 12, no. 5, pp. 375–384, 2010.

39. G. Deuschl, C. Schade-Brittinger, P. Krack et al. et al., “A randomized trial of deep-brain stimulation for Parkinson’s disease,” *The New England Journal of Medicine*, vol. 31, pp. 896–898, 2006.

40. F. M. Weaver, K. Follett, M. Stern et al., “Bilateral deep brain stimulation vs best medical therapy for patients with advanced parkinson disease: a randomized controlled trial,” *Journal of the American Medical Association*, vol. 301, no. 1, pp. 63–73, 2009.

41. J. Yoges, A. Koulousakis, and V. Sturm, “Deep brain stimulation for Parkinson’s disease,” *Acta Neurochirurgica, Supplementum*, vol. 97, no. 2, pp. 171–184, 2007.

42. F. M. Weaver, K. A. Follett, M. Stern et al., “Randomized trial of deep brain stimulation for Parkinson disease: thirty-six-month outcomes,” *Neurology*, vol. 79, pp. 55–65, 2012.

43. J. S. Perlmutter and J. W. Mink, “Deep brain stimulation,” *Annual Review of Neuroscience*, vol. 29, pp. 229–257, 2006.

44. J. A. Obeso, C. W. Olafson, M. C. Rodriguez-Oroz, P. Krack, R. Kumar, and A. E. Lang, “Deep-brain stimulation of the subthalamic nucleus or the pars interna of the globus pallidus in Parkinson’s disease,” *The New England Journal of Medicine*, vol. 345, no. 13, pp. 956–963, 2001.

45. P. Biboolnurak, A. E. Lang, A. M. Lozano et al., “Levodopa response in long-term bilateral subthalamic stimulation for Parkinson’s disease,” *Movement Disorders*, vol. 22, no. 7, pp. 990–997, 2007.

46. G. Kleiner-Fisman, J. Herzog, D. N. Fisman et al., “Subthalamic nucleus deep brain stimulation: summary and meta-analysis of outcomes,” *Movement Disorders*, vol. 21, supplement 14, pp. S290–S304, 2006.

47. F. Klostermann, F. Ehlen, J. Vesper et al., “Effects of subthalamic deep brain stimulation on dysarthrophonia in Parkinson’s disease,” *Journal of Neurology, Neurosurgery and Psychiatry*, vol. 79, no. 5, pp. 522–529, 2008.

48. C. Gross, A. Rougier, D. Guehl, T. Boraud, J. Julien, and B. Bioulac, “High-frequency stimulation of the globus pallidus internus in Parkinson’s disease: a study of seven cases,” *Journal of Neurosurgery*, vol. 87, no. 4, pp. 491–498, 1997.

49. J. Giha, J. G. Villemure, H. Fankhauser, J. Favre, G. Assal, and F. Giha-Schmid, “Efficiency and safety of bilateral contemporaneous pallidal stimulation (deep brain stimulation) in levodopa-responsive patients with Parkinson’s disease with severe motor fluctuations: a 2-year follow-up review,” *Journal of Neurosurgery*, vol. 89, no. 5, pp. 713–718, 1998.

50. M. Krause, W. Fogel, A. Heck et al., “Deep brain stimulation for the treatment of Parkinson’s disease: subthalamic nucleus versus globus pallidus internus,” *Journal of Neurology Neurosurgery and Psychiatry*, vol. 70, no. 4, pp. 464–470, 2001.

51. K. E. Lyons, S. B. Wilkinson, A. I. Tröster, and R. Pahwa, “Long-term efficacy of globus pallidus stimulation for the treatment of Parkinson’s disease,” *Stereotactic and Functional Neurosurgery*, vol. 79, no. 3-4, pp. 214–220, 2002.

52. J. M. Taha, M. A. Janssen, and J. Favre, “Thalamic deep brain stimulation for the treatment of head, voice, and bilateral limb tremor,” *Journal of Neurosurgery*, vol. 91, no. 1, pp. 68–72, 1999.

53. A. A. Obwegeser, R. J. Uitti, R. J. Witte, J. A. Lucas, M. F. Turk, and R. E. Wharen, “Quantitative and qualitative outcome measures after thalamic deep brain stimulation to treat disabling tremors,” *Neurosurgery*, vol. 48, no. 2, pp. 274–284, 2001.

54. M. I. Hariz, P. Krack, F. Alesch et al., “Multicentre European study of thalamic stimulation for parkinsonian tremor: a 6 year follow-up,” *Journal of Neurology, Neurosurgery and Psychiatry*, vol. 79, no. 6, pp. 694–699, 2008.

55. J. D. Putze, R. E. Wharen, Z. K. Wsolek, M. F. Turk, A. J. Strongosky, and R. J. Uitti, “Thalamic deep brain stimulation for tremor-predominant Parkinson’s disease,” *Parkinsonism and Related Disorders*, vol. 10, no. 2, pp. 81–88, 2003.

56. M. Gentil, P. Garcia-Ruiz, P. Pollak, and A. L. Benabid, “Effect of bilateral deep-brain stimulation on oral control of patients with Parkinsonism,” *European Neurology*, vol. 44, no. 3, pp. 147–152, 2000.

57. M. Richards, K. Marder, L. Cote, and R. Mayeux, “Interrater reliability of the Unified Parkinson’s Disease Rating Scale motor examination,” *Movement Disorders*, vol. 9, no. 1, pp. 89–91, 1994.

58. F. Karlsson, P. Blomstedt, K. Olofsson, J. Lindqvist, E. Nordh, and J. van Doorn, “Control of phonatory onset and offset in Parkinson patients following deep brain stimulation of the subthalamic nucleus and caudal zona incerta,” *Parkinsonism & Related Disorders*, vol. 18, pp. 824–827, 2012.

59. C. Dromey and S. Bjarnason, “A preliminary report on disordered speech with deep brain stimulation in individuals with Parkinson’s disease,” *Parkinsonism’s Disease*, vol. 2011, Article ID 796205, 2011.

60. S. Lundgren, T. Saes, F. Karlsson et al., “Deep brain stimulation of caudal zona incerta and subthalamic nucleus in patients with Parkinson’s disease: effects on voice intensity,” *Parkinson’s Disease*, vol. 2011, Article ID 658956, 8 pages, 2011.

61. F. Karlsson, E. Unger, S. Wahlgren et al., “Deep brain stimulation of caudal zona incerta and subthalamic nucleus in
patients with Parkinson’s disease: effects on diadochokinetic rate,” *Parkinson’s Disease*, vol. 2011, Article ID 605607, 10 pages, 2011.

[62] K. Chenausky, J. Macauslan, and R. Goldhor, “Acoustic analysis of PD speech,” *Parkinson’s Disease*, vol. 2011, Article ID 435232, 13 pages, 2011.

[63] S. Skodda, A. Flaschkamp, and U. Schlegel, “Instability of syllable repetition in Parkinson’s disease-Influence of levodopa and deep brain stimulation,” *Movement Disorders*, vol. 26, no. 4, pp. 728–730, 2011.

[64] J. H. Park, J. Y. Lee, H. J. Kim et al., “Electrode position and the clinical outcome after bilateral subthalamic nucleus stimulation,” *Journal of Korean Medical Science*, vol. 26, pp. 1344–1355, 2011.

[65] L. T. Robertson, R. J. St George, P. Carlson-Kuhta, P. Hogarth, K. J. Burchiel, and F. B. Horak, “Site of deep brain stimulation and jaw velocity in Parkinson disease,” *Journal of Neurosurgery*, vol. 115, pp. 985–994, 2011.

[66] C. Moreau, O. Pennel-Ployart, S. Pinto et al., “Modulation of dysarthropneumonia by low-frequency STN DBS in advanced Parkinson’s disease,” *Movement Disorders*, vol. 26, no. 4, pp. 659–663, 2011.

[67] M. J. Hammer, S. M. Barlow, K. E. Lyons, and R. Pahwa, “Subthalamic nucleus deep brain stimulation changes velopharyngeal control in Parkinson’s disease,” *Journal of Communication Disorders*, vol. 44, no. 1, pp. 37–48, 2011.

[68] D. Van Laancker Sidtis, T. Rogers, V. Godier, M. Tagliati, and J. J. Sidtis, “Voice and fluency changes as a function of speech task and deep brain stimulation,” *Journal of Speech, Language, and Hearing Research*, vol. 53, no. 5, pp. 1167–1177, 2010.

[69] M. J. Hammer, S. M. Barlow, K. E. Lyons, and R. Pahwa, “Subthalamic nucleus deep brain stimulation changes speech respiratory and laryngeal control in Parkinson’s disease,” *Journal of Neurology*, vol. 257, no. 10, pp. 1692–1702, 2010.

[70] M. Åström, E. Tripoliti, M. I. Hariz et al., “Patient-specific model-based investigation of speech intelligibility and movement during deep brain stimulation,” *Stereotactic and Functional Neurosurgery*, vol. 88, no. 4, pp. 224–233, 2010.

[71] E. Tripoliti, L. Zrinzo, I. Martinez-Torres et al., “Effects of contact location and voltage amplitude on speech and movement in bilateral subthalamic nucleus deep brain stimulation,” *Movement Disorders*, vol. 23, no. 16, pp. 2377–2383, 2008.

[72] V. S. Lee, X. P. Zhou, D. A. Rahn, E. Q. Wang, and J. J. Jiang, “Perturbation and nonlinear dynamic analysis of acoustic phonatory signal in Parkinsonian patients receiving deep brain stimulation,” *Journal of Communication Disorders*, vol. 41, no. 6, pp. 485–500, 2008.

[73] F. Klostermann, F. Ehlen, J. Vesper et al., “Effects of subthalamic deep brain stimulation on dysarthrophonia in Parkinson’s disease,” *Journal of Neurology, Neurosurgery and Psychiatry*, vol. 79, no. 5, pp. 522–529, 2008.

[74] L. D’Alatri, G. Paludetti, M. F. Contarino, S. Galia, M. R. Marchese, and A. R. Bentivoglio, “Effects of bilateral subthalamic nucleus stimulation and medication on parkinsonian speech impairment,” *Journal of Voice*, vol. 22, no. 3, pp. 365–372, 2008.

[75] M. Pützer, W. J. Barry, and J. R. Moringlane, “Effect of bilateral stimulation of the subthalamic nucleus on different speech subsystems in patients with Parkinson’s disease,” *Clinical Linguistics and Phonetics*, vol. 22, no. 12, pp. 957–973, 2008.

[76] A. L. Tornqvist, L. Schalen, and S. Rehncrona, “Effects of different electrical parameter settings on the intelligibility of speech in patients with Parkinson’s disease treated with subthalamic deep brain stimulation,” *Movement Disorders*, vol. 20, no. 4, pp. 416–423, 2005.

[77] S. Pinto, M. Gentil, P. Krack et al., “Changes induced by Levodopa and subthalamic nucleus stimulation on Parkinsonian speech,” *Movement Disorders*, vol. 20, no. 11, pp. 1507–1515, 2005.

[78] S. Pinto, S. Thobois, N. Costes et al., “Subthalamic nucleus stimulation and dysarthria in Parkinson’s disease: a PET study,” *Brain*, vol. 127, no. 3, pp. 602–615, 2004.

[79] M. Rousseaux, P. Krystko, O. Koziowska, C. Oszancak, S. Blond, and A. Destée, “Effects of subthalamic nucleus stimulation on parkinsonian dysarthria and speech intelligibility,” *Journal of Neurology*, vol. 251, no. 3, pp. 327–334, 2004.

[80] M. Gentil, S. Pinto, P. Pollak, and A. L. Benabid, “Effect of bilateral stimulation of the subthalamic nucleus on parkinsonian dysarthria,” *Brain and Language*, vol. 85, no. 2, pp. 190–196, 2003.

[81] S. Pinto, M. Gentil, V. Fraix, A. L. Benabid, and P. Pollak, “Bilateral subthalamic stimulation effects on oral force control in Parkinson’s disease,” *Journal of Neurology*, vol. 250, no. 2, pp. 179–187, 2003.

[82] R. Moretti, P. Torre, R. M. Antonello et al., “Speech initiation hesitation following subthalamic nucleus stimulation in a patient with Parkinson’s disease,” *European Neurology*, vol. 49, no. 4, pp. 251–253, 2003.

[83] M. Gentil, P. Chauvin, S. Pinto, P. Pollak, and A. L. Benabid, “Effect of bilateral stimulation of the subthalamic nucleus on parkinsonian voice,” *Brain and Language*, vol. 79, no. 2, pp. 233–240, 2001.

[84] C. Dromey, R. Kumar, A. E. Lang, and A. M. Lozano, “An investigation of the effects of subthalamic nucleus stimulation on acoustic measures of voice,” *Movement Disorders*, vol. 15, pp. 1132–1138, 2000.

[85] M. Gentil, P. García-Ruiz, P. Pollak, and A. L. Benabid, “Effect of bilateral deep-brain stimulation on oral control of patients with Parkinsonism,” *European Neurology*, vol. 44, no. 3, pp. 147–152, 2000.

[86] M. Gentil, C. L. Tournier, P. Pollak, and A. L. Benabid, “Effect of bilateral subthalamic nucleus stimulation and dopotherapy on oral control in Parkinson’s disease,” *European Neurology*, vol. 42, no. 3, pp. 136–140, 1999.

[87] M. Gentil, P. García-Ruiz, P. Pollak, and A. L. Benabid, “Effect of stimulation of the subthalamic nucleus on oral
control of patients with parkinsonism,” Journal of Neurology Neurosurgery and Psychiatry, vol. 67, no. 3, pp. 329–333, 1999.

[92] P. Limousin, P. Krack, P. Pollak et al., “Electrical stimulation of the subthalamic nucleus in advanced Parkinsonian’s disease,” The New England Journal of Medicine, vol. 339, no. 16, pp. 1105–1111, 1998.

[93] P. Krack, A. Batir, N. Van Blercom et al., “Five-year follow-up of bilateral stimulation of the subthalamic nucleus in advanced Parkinson’s disease,” The New England Journal of Medicine, vol. 349, no. 20, pp. 1925–1934, 2003.

[94] A. Farrell, D. Theodoros, E. Ward, B. Hall, and P. Silburn, “Effects of neurosurgical management of Parkinson’s disease on speech characteristics and oromotor function,” Journal of Speech, Language, and Hearing Research, vol. 48, no. 1, pp. 5–20, 2005.

[95] P. Santens, M. De Letter, J. Van Borsel, J. De Reuck, and J. Caemaert, “Lateralized effects of subthalamic nucleus stimulation on different aspects of speech in Parkinson’s disease,” Brain and Language, vol. 87, no. 2, pp. 253–258, 2003.

[96] E. Wang, L. Verhagen Metman, R. Bakay, J. Arzbaecher, and B. Bernard, “The effect of unilateral electrostimulation of the subthalamic nucleus on respiratory/phonatory subsystems of speech production in Parkinson’s disease—a preliminary report,” Clinical Linguistics and Phonetics, vol. 17, no. 4-5, pp. 283–289, 2003.

[97] C. C. McIntyre, M. Savasta, B. L. Walter, and J. L. Vitek, “How does deep brain stimulation work? Present understanding and future questions,” Journal of Clinical Neurophysiology, vol. 21, no. 1, pp. 40–50, 2004.

[98] E. M. Critchley, “Speech disorders of Parkinsonism: a review,” Journal of Neurology, Neurosurgery & Psychiatry, vol. 44, pp. 751–758, 1981.

[99] J. Volkmann, N. Allert, J. Voges, P. H. Weiss, H. J. Freund, and V. Sturm, “Safety and efficacy of pallidal or subthalamic nucleus stimulation in advanced PD,” Neurology, vol. 56, no. 4, pp. 548–551, 2001.

[100] J. Spielman, L. Mahler, A. Halpern, P. Gilley, O. Klepitskaya, and L. Ramig, “Intensive voice treatment (LSVT LOUD) for Parkinson’s disease following deep brain stimulation of the subthalamic nucleus,” Journal of Communication Disorders, vol. 44, pp. 688–700, 2011.

[101] T. Boertien, L. Zrinzo, J. Kahan et al., “Functional imaging of subthalamic nucleus deep brain stimulation in Parkinson’s disease,” Movement Disorders, vol. 26, pp. 2434–2436, 2011.

[102] T. Rouaud, T. Dondaine, S. Drapier et al., “Pallidal stimulation in advanced Parkinson’s patients with contraindications for subthalamic stimulation,” Movement Disorders, vol. 25, no. 12, pp. 1839–1846, 2010.

[103] M. I. Hariz, S. Rehncrona, N. P. Quinn, J. D. Speelman, and C. Wensing, “Multicenter study on deep brain stimulation in Parkinson’s disease: an independent assessment of reported adverse events at 4 years,” Movement Disorders, vol. 23, no. 3, pp. 416–421, 2008.

[104] R. Pahwa, K. E. Lyons, S. B. Wilkinson et al., “Long-term evaluation of deep brain stimulation of the thalamus,” Journal of Neurosurgery, vol. 104, no. 4, pp. 506–512, 2006.

[105] P. Dowsey-Limousin, “Postoperative management of vin DBS for tremor,” Movement Disorders, vol. 17, supplement 3, pp. S208–S211, 2002.

[106] E. Frost, E. Tripoliti, M. I. Hariz, T. Pring, and P. Limousin, “Self-perception of speech changes in patients with Parkinson’s disease following deep brain stimulation of the subthalamic nucleus,” International Journal of Speech-Language Pathology, vol. 12, no. 5, pp. 399–404, 2010.