Treatments for dysarthria in Parkinson's disease

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Dysarthria in Parkinson's disease can be characterised by monotony of pitch and loudness, reduced stress, variable rate, imprecise consonants, and a breathy and harsh voice. Use of levodopa to replenish dopamine concentrations in the striatum seems to improve articulation, voice quality, and pitch variation, although some studies show no change in phonatory parameters. Traditional speech therapy can lead to improvement of dysarthria, and intensive programmes have had substantial beneficial effects on vocal loudness. Unilateral surgical lesions of subcortical structures are variably effective for the alleviation of dysarthria, whereas bilateral procedures typically lead to worsening of speech production. Among deep-brain stimulation procedures, only stimulation of the subthalamic nucleus improves some motor components of speech although intelligibility seems to decrease after surgery. Due to the variable treatment effects on parkinsonian speech, management of dysarthria is still challenging for the clinician and should be discussed with the patient.

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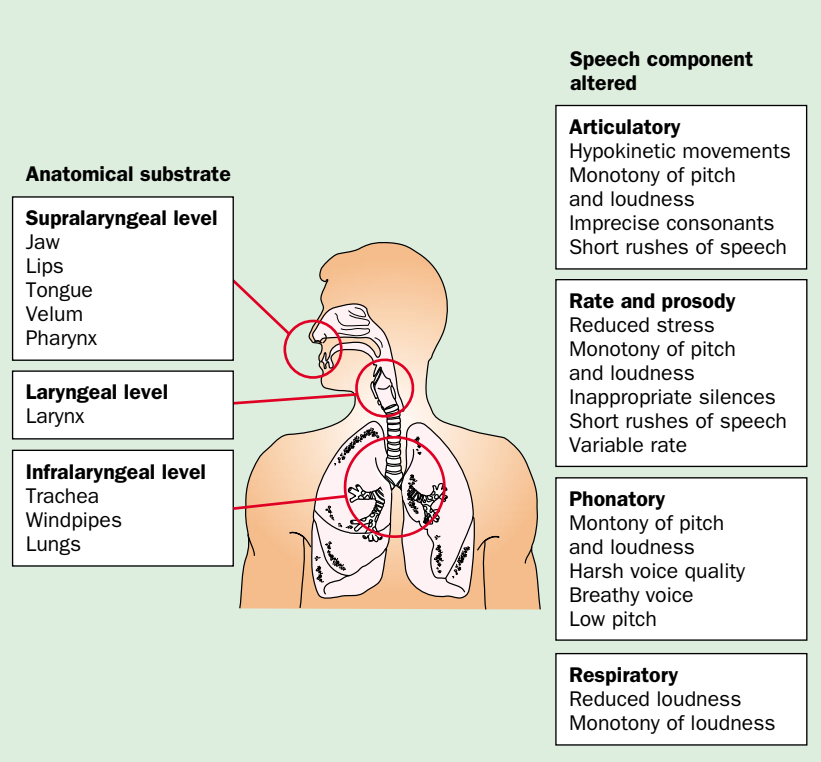

Figure 1. Anatomical substrate of speech components and parkinsonian dysarthria characteristics. PD speech has been defined by Darley, Aronson, and Brown[3,6] as a hypokinetic dysarthria, and can be associated with specific changes to three of the main components of speech production: respiration, phonation, and articulation. The prosodic component can also be taken into account because several speech components are involved in the dysfunction.[9]

Parkinson's disease (PD) affects 1–2% of people age over 60 years; the mean age of symptom onset is about 58 years,[1] but patients can develop the symptoms before age 40 years. In a survey, 70% of patients with PD indicated that their speech was impaired during the disease process.[2] Dysarthria is a collective name for impairments of speech-organ motor control resulting from a lesion of the peripheral nervous system or CNS.[3] Dysarthria can appear at any stage of PD and worsens in the later stages of the disease[4,5] to cause a progressive loss of communication and social isolation. After 10 years of disease progression, the classic symptom triad—akinesia, rigidity, and tremor—is usually still improved by dopamine therapy, whereas axial signs, such as dysarthria, worsen for most patients.[4] PD dysarthria is characterised by a monotony of pitch and loudness, reduced stress, variable rate, short rushes of speech, imprecise consonants, and a breathy and harsh voice.[3,6–8] These characteristics have been attributed to a weakness (hypokinesia) and slowness (bradykinesia) of speech-organ movements, rigidity, and rest tremor (figure 1),[9] which lead to bradykinetic and hypokinetic articulatory movements[10] associated with orofacial hypomimy.

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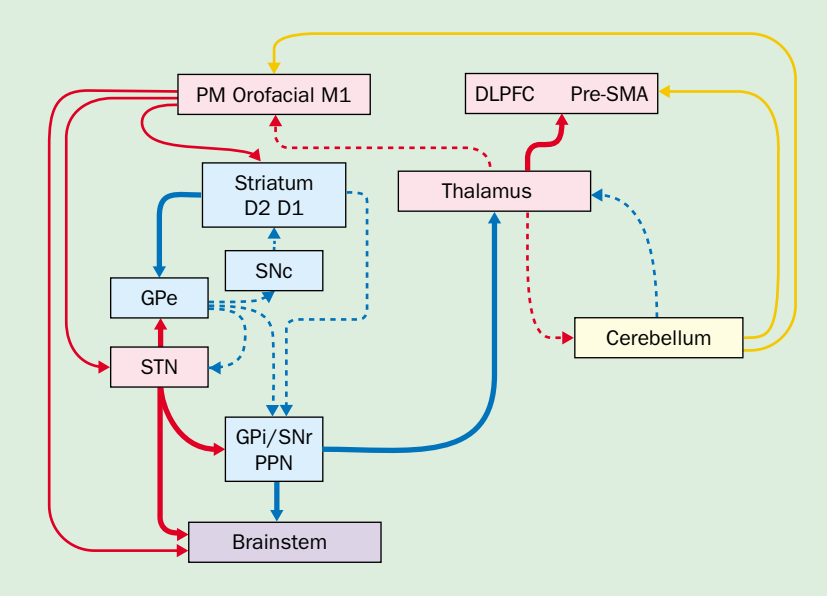

Figure 2. Cerebral activation abnormalities during speech in PD. The three main kinds of cerebrocortical dysfunction during PD speech[28] are: an overactivation of the rostral part of the pre-SMA and the bilateral dorsolateral prefrontal cortex (DLPFC); an underactivation of the orofacial M1; and an underactivation of the cerebellum. These results reinforce the fact that two parallel neural pathways are implicated in motor control of speech production: the basal ganglia are involved in the first and the cerebellum in the second. In PD, dysfunction of the first pathway seems to affect the second. D1, D2=D1 and D2 striatal dopamine receptors; GPe=external globus palildus; GPi=internal globus pallidus; orofacial M1=primary motor cortex corresponding to the orofacial somatotopy; PM=premotor cortex; PPN=pediculopontine nucleus; SNc=substantia nigra pars compacta; SNr=substantia nigra pars reticulata; STN=subthalamic nucleus; red lines=excitatory glutamatergic projections; blue lines=inhibitory GABAergic projections; dotted lines=underactivity of the projection, and thickness of the continued lines represent the range from supposed normal (thin) and hyperactive (thick) activities; yellow lines=corticocerebellar connections.

Reviews on the effects of treatment on PD dysarthria have recently been published.[11–14] However, they do not include comments on new surgical treatments such as deep brain stimulation. In this review, we provide a current outline of the effect of PD treatments on parkinsonian speech; particular attention will be paid to surgical lesions and deep brain stimulation.

Physiopathology of parkinsonian dysarthria

Progressive dopaminergic neurodegeneration of the nigrostriatal pathway, projecting from the substantia nigra to the striatum, is the main pathogenetic process in PD[15] and the origin of basal ganglia motor loop dysfunction.[16–19] Thus, nigrostriatal denervation leads to the appearance of parkinsonian motor symptoms,[20] including changes to speech. Impaired neuromuscular function is the basis of dysarthria in PD. Progression of PD with time concomitant with aggravation of dysarthria suggests a link to the increasing severity of cerebral non-dopaminergic lesions.[21] Owing to its complexity, speech production cannot be restricted to the motor-cortex–basal-ganglia–cortex loop. In terms of motor speech function, it should be emphasised that this neural pathway[22] is tightly connected with the cortex–basal-ganglia–cerebellum–cortex circuit.[23] Speech production involves particularly the supplementary motor area (SMA) or anterior cingular cortex for the generation of speech,[24] and the left insula for planning of speech articulatory movements.[22,25] Speech movements are initiated in the primary motor cortex corresponding to the trunk and orofacial somatotopic areas.[22] The language component of speech involves participation of prefrontal, frontal, and temporal cortices,[26] such as Broca's and Wernicke's areas, which increase the complexity of the function.

Two recent functional imaging studies with PET and oxygen-15-labelled H_2O have answered some questions about cerebral dysfunction in PD dysarthria. Liotti and colleagues[27] have analysed the brain-activation profile during a speech production task and showed that abnormalities of cerebral activation were essentially represented by an overactivation of orofacial primary motor cortex, inferior lateral premotor cortex, and SMA. Liotti and colleagues[27] interpreted "the motor-premotor cortical effects in the group with PD pre-voice treatment as pre-treatment abnormalities". However, they pointed out that the small group size (five patients) and the absence of control individuals made the findings preliminary. Another study showed that in PD, there is a lack of activation in the right orofacial motor cortex and the bilateral cerebellar hemispheres, an abnormal increase in regional cerebral blood flow in the right superior premotor cortex and the bilateral dorsolateral prefrontal cortex, and an overactivation of the SMA.[28] The researchers concluded that parkinsonian dysarthria is associated with an altered recruitment of the main motor cerebral regions (orofacial motor cortex, cerebellum), and an increased involvement of premotor and prefrontal cortices (bilateral dorsolateral prefrontal cortex, SMA, superior premotor cortex). These abnormal activations are different from those reported during hand motor tasks, and could result as a compensatory mechanism, but might also arise directly as part of the pathophysiology of PD. The common result of these two studies is the overactivation of the SMA, interpreted by both groups as a consequence of doing a complex task. However, the discrepancy regarding activation of the primary motor cortex reflects the contradictory functional imaging results also obtained in terms of hand-movement tasks. So far, there have only been two PET studies of parkinsonian dysarthria, and it is still difficult to make conclusion on the cerebral activation changes related to this specific symptom (figure 2).

Effect of drugs on PD dysarthria

Levodopa, a dopamine precursor, replenishes the dopaminergic system in the striatum.[29] Patients with PD

Table 1. Variability in the effects of levodopa on dysarthria in PD

	Improvement	Steady-state	Worsening
Intelligibility	Rigrodsky and Morrison, 1970 (21 patients, perceptual rating of speech components)[33] Fetoni et al, 1997 (nine patients, UPDRS)[35] Wolfe et al, 1975 (17 patients, perceptual rating of speech components)[40]	Quaglieri and Celesia, 1977 (30 patients; 14 without any surgery, qualitative rating of global speech)[36] Wolfe et al, 1975 (17 patients; perceptual rating of speech components)[40]	Marsden and Parkes, 1976 (case reports; clinical observations)[32] Critchley, 1976 (case study; perceptual evaluation)[37] Anderson et al, 1999 (case study; perceptual count of speech dysfluencies)[38] Benke et al, 2000 (24 advanced patients; psycholinguistical tests for perceptual speech assessment)[39] Goberman et al, 2003 (nine patients; perceptual count of speech dysfluencies)[41]
Laryngeal level	Mawdsley and Gamsu, 1971 (20 patients, two with previous thalamotomy, acoustical recordings for phonation duration)[34] Jiang et al, 1999 (15 patients, electroglottographic recording)[42] Sanabria et al, 2001 (20 patients, acoustics for phonatory parameter analysis)[43] Gallena et al, 2001 (six early stage patients, laryngeal electromyography)[44] Goberman et al, 2002 (nine patients, acoustic recording for phonatory parameter analysis) [45]	Jiang et al, 1999 (15 patients; acoustics, airflow and electroglottographic recording)[42] Poluha et al, 1998 (ten patients; vowel duration measurements)[46]	
Supralaryngeal level	Vercueil et al, 1999 (11 patients, airflow, rib cage and abdomen movements)[47] Leanderson et al, 1971 (seven patients, five with previous thalamotomy, labial EMG)[48] Leanderson et al, 1972 (12 patients, five studied before and after levodopa, labial EMG)[49] Nakano et al, 1973 (18 patients, early introduction of levodopa, orofacial EMG)[50] Cahill et al, 1998 (16 patients, lip pressure measurements)[51] Svensson et al, 1993 (nine patients, jaw movement kinematics)[52]	Solomon and Hixon, 1993 (14 patients; 14 healthy people, chest wall kinematics and oral pressure)[53] De Letter et al, 2003 (ten patients; tongue force measurements)[54]	Vercueil et al, 1999 (one patient; airflow, rib cage and abdomen movements)[47] Gentil et al, 1998 (14 patients; lips and tongue force measurements)[55] Gentil et al, 1999 (case study; PD for 13 years, lips and tongue force measurements)[56] De Letter et al, 2003 (ten patients; tongue force measurements)[54]

The studies have been classified in terms of speech components, investigation to assess intelligibility, laryngeal, and supralaryngeal changes. For each study, the number of people studied and the primary outcome are included in brackets. The small number of patients and the variability of the population (early stage, late stage, early introduction of levodopa, levodopa associated with fluctuations) result in a weak level of evidence in terms of reliability and reproducibility of the results.

have a favourable response to levodopa therapy at the start of treatment.[30] However, this response decreases as dopaminergic and non-dopaminergic systems degrade.[4] Hence, dysarthria develops as a consequence of dopaminergic and non-dopaminergic progressive degradation.[31] Motor fluctuations appear with an alternate state of severe parkinsonian disability ("off" period) and a state of improved mobility ("on" period), often impaired by dyskinesias.[32] These fluctuations can contribute to the aggravation of dysarthria (table 1[33–56]).

Dopaminergic treatments

Perceptual assessment

A trend towards improvement on both spontaneous speech and oral reading was first reported in the 1970s.[33] Increase of speech volume after levodopa treatment also improves speech intelligibility in patients with PD,[34] and qualitative rating by a neurologist showed some improvement in speech and facial expression.[35] However, some patients do not improve.[36] Common complications can appear after some years of levodopa treatment; these include orofacial or respiratory dyskinesias, oromandibular dystonias,[32] "peak-dose dysphonia",[37] and neurogenic stuttering.[38,39]

Dopamine agonists can be used in monotherapy early in the disease, or with levodopa. A perceptual improvement of dysarthria has been described when piribedil is used in association with amantadine or levodopa.[57] Pergolide, with or without levodopa, did not achieve significant improvement for dysarthria,[58] but perceptual assessment of dysarthria showed a trend towards improvement during coadministration of bromocriptine and levodopa.[59,60]

Electrophysiological studies of respiration

No systematic differences in rest breathing were found between the end and the middle of the levodopa cycle. Significant differences were measured for 11 of the

14 patients in three of 15 variables in speech breathing throughout the levodopa drug cycle.[53] Another study indicated some improvement in the diaphragm component of breathing at rest, although some worsening seemed to be indicated for intercostal muscles after drug administration.[47]

Electrophysiological studies of phonation

Fundamental frequency and intensity are the two main outcome measures of phonatory analysis. On the one hand, voice quality improves with levodopa;[40] for example, coupled acoustic and glottographic measurements showed an increase in sound pressure level but no change for other phonatory factors.[42] A recent acoustic analysis showed a significant increase of fundamental frequency after levodopa administration, which seems to be directly related to the decrease of laryngeal hypokinesia and rigidity induced by dopaminergic stimulation.[43] The same kind of results have been observed during electromyography of laryngeal muscles.[44] On the other hand, some studies show no change in phonatory factors after levodopa administration.[46] Hence, the effect of dopaminergic treatment seems to be limited and variable among patients—even if some group differences are small, phonatory improvements owing to dopaminergic treatment can be seen in individual patients.[45] Schulz and Grant[13,14] emphasised that such variability in the findings may result from drug variation among participants or dysarthria severity.[61]

Electrophysiological studies of rate and prosody

Pitch variation improves with levodopa therapy, although the speech rate was unchanged.[40] Dysfluency changes have been noted in some patients, supporting the hypothesis that speech dysfluencies may be related to an increase or decrease in the concentration of dopamine in the brain.[41] A recent acoustic analysis showed a significant reduction of intensity variation and the tremor index.[43]

Electrophysiological studies of articulation

Levodopa restores orofacial muscle activity assessed with electromyography,[62] leading to a decrease of tonic hyperactivity of labial muscles[48] and the restoration of labial motor control involved in speech.[49] This beneficial effect results also in the reduction of reaction and movement times measured during lip kinematical analysis after levodopa is given,[50] and the improvement of lip contractions.[51] Lip rigidity is also reduced immediately after levodopa ingestion.[63] Moreover, mandibular movements during syllable-repetition tasks are improved.[52] However, speech organ forces are reduced and less accurate when taking levodopa than when not taking medication.[54–56] Another study showed that maximum force and contraction do not differ between the "on" and "off" states.[44] A recent report also showed no significant improvement in laryngeal and articulatory speech components after administration of apomorphine, a dopaminergic receptor agonist.[64] To summarise, the main effect of levodopa is an improvement of lip activity; however, apomorphine had no effect.

Non-dopaminergic treatments

Catabolic dopaminergic enzyme inhibitors

Dopamine can be inactivated in the presynaptic terminal by monoamine-oxidase B (MAO-B) and in the synaptic cleft by catechol-O-methyl-transferase (COMT). Hence, inhibition of MAO-B and COMT can inhibit degradation of dopamine. Subjective and objective improvement of the rate and range of oral motor diadochokinesis and respiratory function (measures of vital capacity and words per exhalation during speech reading) has been reported with use of selegiline, a MAO-B inhibitor.[65] However, no significant improvement has been observed on acoustic assessments in patients with untreated early-onset PD untreated with levodopa.[66]

Other drugs

Brumlik and colleagues[67] observed a tendency towards improvement in maximum intensity speech range and speaking rate in patients taking trihexyphenidyl compared with those taking placebo. Little improvement in articulation after administration of anticholinergic drugs has been described.[68] Clonazepam has been studied in a double-blind trial in 11 patients, ten patients had an improvement in nine of 14 features of speech.[69] Amantadine might be implicated in the eventual generation of "vocal" myoclony, as described in a case report of myoclonus involving laryngeal and pharyngeal muscles triggered by speech.[70] This uncommon drug-induced impairment resulted in speech arrests and involuntary vocalisations.

The effects of levodopa, dopaminergic agonists, and other drugs on dysarthria in PD are variable. All the studies reviewed were done in small and diverse groups of patients, leading to an inherently low level of evidence and reliability. The results of early levodopa administration seem more positive than the more recent "on"/"off" medication studies. Nevertheless, there is no evidence of systematic improvement in dysarthria owing to dopamine-replacement therapy. There is still insufficient information for clinical practice to allow any viable prediction regarding speech response to levodopa.

Speech therapy and PD dysarthria

Speech therapy is based either on behavioural treatment—conscious training to strengthen muscles involved with coordination of respiration, phonation, or articulation—or on the use of devices and biofeedback. The treatment of dysarthria in PD is challenging for the clinician because of the neurodegenerative features of the disease, the variable effect of pharmacological therapy, and the common cognitive and emotional symptoms of patients. Issues of sensorimotor perception and integration,[71] as well as motor coordination,[72] were not considered in traditional speech therapy leading to inconsistent treatment effects.[73] However, recent development of an intensive voice treatment known as the Lee Silverman voice treatment (LSVT®) has renewed interest in effective speech therapy.[74]

Traditional speech therapy in PD

Studies over the past two decades focused on different components of speech, such as respiration, pitch, and

articulation with increased vocal loudness as the main objective. Breathing and prosodic[75] exercises lead to some improvement in speech production. Less intensive speech therapy causes some improvement in vocal loudness and pitch,[76] but follow-up data are lacking. In a group study, increase in maximum phonation times, vocal intensity, and improvement in voice monotony, speech intelligibility, and swallowing occurred after rehabilitation.[77]

Delayed auditory feedback increased speech intelligibility in two of 11 patients.[78] Increase in vocal loudness and fundamental frequency and decrease of fast speech rate after delayed auditory feedback were seen in a 3 month follow-up of two patients.[79] However, these positive effects have been showed in only a small number of patients. In order to compensate for speech hypophonia, voice amplifiers can also increase vocal loudness artificially.[80] In a case study of a microcomputer-based wearable biofeedback mechanism, patients themselves could consciously modulate speech loudness.[81] This mechanism led to an improvement of perceptual and acoustic assessments. Masking noise has also been used to force patients to increase their vocal loudness. Significant improvement has been reported in ten patients,[82] but it seems difficult for the patients to voluntarily control this increase in the absence of masking noise.[83] Unfortunately, there have been no studies of the use of devices in conversation.

LSVT®

LSVT® is an intensive, high effort speech treatment designed to rescale the amplitude of motor output of speakers with PD dysarthria.[84] The effects of LSVT® have been documented with the widest range of outcome measures,[85] can be interpreted with confidence, and are for the most part positive. LSVT® is based on the learning of intensive respiratory and phonatory training during 16 sessions a month, with emphasis on recalibration of speech effort so that speakers appreciate the level of effort needed to speak.[86] Treatment has been developed over the past decade, and the focus of this treatment is vocal loudness.[74,87–90]

Unlike classical respiratory speech-component therapy, LSVT® is supported by evidence of improved vocal loudness for 24 months after treatment.[74] Beneficial effects of this treatment on speech intelligibility, pitch, rate, facial expression, and swallowing have also been observed.[91,92] In addition, it seems that LSVT® is able to reverse the abnormal cerebral activations during sustained vowel and paragraph reading.[26]

Efficacy of speech therapy

Evidence-based appraisal of the effect of speech therapy on parkinsonian dysarthria has been the subject of a recent review.[93] This critical review of five clinical studies[72,73,85,89,94] concluded that there is insufficient evidence for improvement after speech therapy. Deane and colleagues[95,96] reached the same conclusions when comparing randomised controlled trials of speech and language therapy with placebo or no intervention and different kinds of speech therapy. They mentioned that a comparison between a placebo and a large randomised controlled trial is still needed to show clearly the efficacy of speech therapy on PD dysarthria.[96] "Outcome measures with particular relevance to patients, their carers, physicians and speech and language therapists should be chosen and the patients followed for at least 6 months to determine the duration of any improvement".[95] Most studies reported improvement after speech therapy with a range of treatment from 2 weeks to 4 weeks, especially when using an intensive voice treatment.[88] Ramig and co-workers[74] provided evidence for speech improvement lasting over 2 years, as well as some preliminary results in the efficacy of LSVT® on swallowing function and facial expression.[97]

Together, these findings set the benchmarks for speech therapy. Behavioural therapy is potentially able to control motor complications and change the anatomofunctional basis of speech impairment in PD. Even if speech therapy has beneficial effects on PD dysarthria, other medical or surgical interventions given to the patients must be considered. Speech therapy effectiveness is dependent on an efficient treatment of all PD symptoms. Most of the studies of speech therapy involved a few patients and this fact raises again the significant sources of potential bias. However, systematic reviews[95,96] that address this issue are available.

Effects of surgical procedures on PD dysarthria

Before the rise of dopamine therapy, functional neurosurgery procedures, such as thalamotomy and pallidotomy, were used to treat parkinsonian symptoms. Some significant improvement after surgical treatment has been observed for motor impairment of limbs; however, the effect on PD dysarthria is more commonly deleterious than it is beneficial. Deep brain stimulation has been used to improve some PD symptoms; but due to its lesion-like effects, there is still a risk of speech worsening.

Subcortical lesions of the basal ganglia

Lesion of the thalamus

Thalamotomy is mainly used to improve tremor; other symptoms improve inconsistently after the surgery.[98] The lesion is generally made in the ventrolateral and the ventral intermediate nuclei of the thalamus. Unilateral thalamotomy worsens speech[99] when the lesion is in either the dominant or the non-dominant hemispheres.[100] Bilateral thalamotomy has been associated with word blocking, slow speech and hypophonia, and a persistent worsening of dysarthria.[101,102] Patients with PD who were given unilateral or bilateral thalamotomy were more dysarthric after surgery,[36] and some of them developed palilalia.[103] Because of its deleterious effect on speech, bilateral thalamotomy has been abandoned for the treatment of PD symptoms.

Lesion of the globus pallidus

Pallidotomy is used to alleviate PD signs and reduce contralateral dyskinesias,[104] and involves lesion of the posteroventral portion of the internal part of the globus pallidus.[105] Neither early[106] nor more recent[107–112] reports

Table 2. The effects of deep brain stimulation on perceptual assessment and electrophysiological measurement of speech subcomponents of dysarthria in PD

	Thalamic stimulation	Pallidal stimulation	Subthalamic nucleus stimulation
Perceptual assessment			
Improvement		Gross et al, 1997 (seven patients; UPDRS)[123]	Limousin et al, 1998 (24 patients; UPDRS)[124] Rousseaux et al, 2004 (nine patients; UPDRS, Lille dysarthria test)[125]
Worsening	Tasker, 1998 (19 patients, 16 with PD, retrospective analysis of qualitative rating scale)[126] Taha et al, 1999 (33 patients, six with PD, qualitative rating scale)[127] Obwegeser et al, 2001 (41 patients, 10 with PD, qualitative evaluation)[128] Putzke et al, 2003 (19 patients, qualitative rating scale)[129]	Ghika et al, 1998 (six patients; UPDRS)[130] Krause et al, 2001 (six patients; UPDRS)[131] Lyons et al, 2002 (nine patients; UPDRS)[132]	Krack et al, 2003 (49 patients; UPDRS)[133] Romito et al, 2003 (33 patients; UPDRS)[134] Moretti et al, 2003 (case study; neurolinguistic evaluation)[135] Hariz et al, 2000 (case report; qualitative rating scale)[136]
Electrophysiological measurement			
Improvement			Pinto et al, 2003 (26 patients; UPDRS, lips and tongue force measurements)[137] Gentil et al, 1999 (ten patients; UPDRS, lips and tongue force measurements)[138] Dromey et al, 2000 (seven patients; UPDRS, acoustic analysis)[139] Gentil et al, 2001 (26 patients; UPDRS, acoustic analysis)[140] Gentil et al, 2003 (16 patients; UPDRS, acoustic analysis, lips and tongue force measurements)[141]
Worsening	Gentil et al, 2000 (14 patients, four patients with VIM stimulation, lips and tongue force measurements)[142]		Santens et al, 2003 (seven patients; acoustic analysis)[143] Wang et al, 2003 (six patients; UPDRS, acoustic analysis)[144]

The population involved in the study and the kind of investigation are included in brackets. The small number of patients and the variability of the population led to a weak level of evidence in terms of reliability and reproducibility of the results.

indicated significant improvement of dysarthria after unilateral and bilateral lesions of the globus pallidus; by contrast, in most cases a worsening of dysarthria was observed with the development of transient dysarthria, facial weakness, swallowing problems, and alteration of verbal fluency.[113] However, Barlow and colleagues[114] reported that half of the patients involved in a study of labial force production and stability experienced improvement on this motor component of speech after bilateral posteroventral pallidotomy. Positive changes in phonatory and articulatory measures in patients with PD who had unilateral posteroventral pallidotomy have also been reported.[115]

Subthalamotomy

In studies of unilateral lesioning of the subthalamic nucleus, few patients' speech improves,[116,117] although a series of 21 patients has shown improvement of dysarthria measured with the Unified Parkinson's Disease Rating Scale (UPDRS).[118] Postural asymmetries have been described after unilateral subthalamotomy,[119] hence axial symptoms may not respond well to surgery.[120] A report suggested that one of the major complications was speech disturbance.[121]

Deep brain stimulation

Stimulation of deep cerebral structures was used to guide lesioning procedures in the early years of surgical treatment of PD; now, advances in the knowledge of basal ganglia pathophysiology[16–19] and neurosurgical procedures[122] have led to a great interest in deep brain, high frequency stimulation as a treatment of choice for parkinsonian signs. Variable effects have been seen in the different targets (table 2[123–144])—ventral intermediate nucleus of the thalamus,[102] posteroventral part of the internal globus pallidus,[145] and dorsolateral part of the subthalamic nucleus.[146]

Stimulation of the ventral-intermediate nucleus

Thalamic stimulation is now preferred to thalamotomy, particularly because of the risk of side-effects, such as worsening of dysarthria and other axial symptoms after thalamotomy, especially in bilateral procedures.[126] Thalamic stimulation does not significantly improve speech, and development of dysarthria and disequilibrium was seen in some patients.[127] A worsening of articulatory organ forces in patients with PD has also been observed after stimulation of the ventral intermediate nucleus.[146] Recent reports reached the same conclusions, confirming that bilateral thalamic stimulation causes more dysarthria than a unilateral procedure.[128,129]

Stimulation of the internal globus pallidus

After stimulation of the internal globus pallidus, clinical dysarthria assessment by use of item 18 of the UPDRS

showed an improvement of speech relative to baseline.[123] Worsening of speech was observed in other studies.[130,131] Dysarthria is an adverse effect of stimulation that can be resolved with adjustments to the stimulation.[132] Assessment of globus pallidus stimulation effects has not benefited so far from electrophysiological studies focusing specifically on speech impairment.

Stimulation of the subthalamic nucleus

Clinical assessment of subthalamic nucleus stimulation for dysarthria by use of the UPDRS showed a beneficial effect. This effect is less pronounced than that on limb movements.[124,137] However, the use of the UPDRS subjective five-point scale seems to be insufficient to assess speech disorders comprehensively and to distinguish the observed impairments.[137] This initial perceptual improvement of intelligibility tends to decrease in the long-term.[133,137] Both improvement and worsening of intelligibility have been observed after surgery,[125] and residual dysarthria after surgery is one of the common side-effects.[124] Hesitation in speech initiation was also observed after stimulation of the subthalamic nucleus.[135] Recent reports highlighted that this kind of surgery is indicated for patients who did not present with axial signs or cognitive impairment.[136,147] One explanation for this worsening of speech intelligibility after surgery would probably be the inadequate recruitment of surrounding structures, such as the corticospinal tract, during chronic stimulation.[137] To summarise, a transient worsening of speech is commonly observed after surgery and is probably associated with a microlesioning effect due to the implantation of the electrodes; in some cases, when stimulation-induced dysarthria lasts longer, it may be necessary to adjust stimulation to a lesser improvement of limb motor function.

In the electrophysiological assessment of ten patients with bilateral stimulation of the subthalamic nucleus, initial results suggested that parkinsonian speech impairment can be improved by this therapy.[138] Further studies showed that phonatory[139–141] and articulatory[137,141] components of speech are improved by subthalamic nucleus stimulation. Two recent studies tried to assess some findings regarding the effects of unilateral stimulation on speech subcomponents. Santens and colleagues[143] reported that "selective left-sided stimulation has a profoundly negative effect on prosody, articulation, and, hence, intelligibility". Similar observations were made by Wang and colleagues,[144] but further assessments are needed because only a few patients were investigated. In addition, subthalamic nucleus stimulation reverses most of the abnormal cerebral activations during speech production and silent articulation.[28]

Most of the studies of effects of surgery on dysarthria are uncontrolled, with selected patients that may not be representative of all patients that may be operated on. The fact that presurgery selection of patients is needed to ensure the minimum risks for those patients in terms of the surgery itself also add a bias that affects the patient populations studied. Particularly, this must be taken into account in view of the typical deterioration of dysarthria after subthalamic-nucleus stimulation, which can be controlled by modulation of stimulation in some cases. Dysarthria is less responsive to deep brain stimulation than global motor limb dysfunction. Stimulation of the target, and probably of surrounding structures, can induce specific speech impairment. Furthermore, pre-existing dysarthria is a disease-related impairment that can be worsened by a stimulation-related change in severity and quality of speech disorder. Speech intelligibility, which reflects the overall speech production understood by a listener, has a poor response to subthalamic nucleus stimulation whereas motor subcomponents of speech, such as lip or laryngeal movements assessed separately, could show some improvement induced by surgery.

Other treatments

Further evidence on the effects of other treatment strategies is needed. A perceptual, acoustic, and electroglottographic study in five patients with PD after fetal-cell transplant of dopaminergic cells showed no or slight effect on phonatory measures; systematic improvement has not been found.[148]

Collagen injections to reduce the space between the vocal cords are given in the hope of improving hypophonia resulting from diminished laryngeal activity.[149] Results are preliminary and seem to be satisfactory,[150,151] but further postsurgery investigation is needed because patients were mostly assessed by means of questionnaires and subjective self assessment. One patient in a case report showed improvement of dysarthria after weekly transcranial magnetic stimulation associated with serotoninergic treatment.[152] Improvement of speech intelligibility and vocal loudness in a few patients with PD has been seen after a music-therapy voice protocol.[153]

Conclusions

The effect of most PD treatments on dysarthria remains unsatisfactory. The effects of pharmacological interventions are highly variable, and no study has shown a predictable response to this kind of therapy. LSVT® seems to be one of the most effective behavioural therapies and improves vocal loudness, speech intelligibility, pitch, rate, facial expression, and swallowing. Among surgical treatments, only subthalamic nucleus stimulation has had some efficacy in the improvement of speech subcomponents, despite a common worsening of speech intelligibility when assessed clinically.

The pathophysiology of PD dysarthria is complex and, at least in part, different from that of limb dysfunction. Thus, the best medical and surgical treatment for limb motor disability should be different from that for dysarthria. An improvement of limb bradykinesia is thus not always associated with an improvement of dysarthria. Owing to the large range of treatment effects on parkinsonian speech, management of dysarthria is still challenging for the clinician and often disappointing for patients. Thus, speech treatment and management in PD should be part of a multidisciplinary approach and needs active involvement of patients.

Search strategy and selection criteria

References for this review were identified by searches of MEDLINE and Current Contents using the search of associated terms "speech, Parkinson", "speech, levodopa", "speech, deep brain stimulation", "speech, STN", "dysarthria, Parkinson", "dysarthria, levodopa", "dysarthria, deep brain stimulation" and "dysarthria, STN". References were also identified from relevant articles and through searches of the authors' files. Only articles published in English were reviewed.

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Authors' contributions

SP completed the reference searches and produced a draft to be completed and revised by CO, ET, ST, PLD, and PA. All the authors shared responsibility for the final version of the review.

Conflict of interest

The authors have no conflict of interest to declare.

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