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Comparing Deep Brain Stimulation and Levodopa as Treatment Methods for Parkinson's Disease

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Comparing Deep Brain Stimulation and Levodopa as Treatment Methods for Parkinson's Disease

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Abstract

Comparing Deep Brain Stimulation and Levodopa as Treatment Methods for Parkinson's Disease

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INTRODUCTION

Parkinson's disease is a progressive and degenerative disease of the central nervous system that affects over one million people in North America (Goberman & Coelho, 2002). Age is the most consistent risk factor for PD (with family history being the second most consistent risk factor) and approximately 10% of patients reported symptoms of PD before age 40 (Goberman & Coelho, 2002). Symptoms of PD, reduced gross motor movement, rigidity, and tremors, result when the production of dopamine is decreased and the supply to the basal ganglia circuit is reduced (Duffy, 2005). Diminished supply of dopamine is a crucial factor in the development of Parkinson's disease and the consequential symptoms.

Parkinson's disease can be treated surgically and pharmacologically. Deep brain stimulation (surgical) and levodopa (pharmacological) currently are the most effective methods of treatment. Research has proven that both forms of treatment are effective in improving motor function in patients with Parkinson's disease. However, speech does not seem to improve significantly with either form of treatment.

Purpose

The purpose of this report is to review critically the available research on deep brain stimulation and levodopa as a means of treatment for Parkinson's disease in an attempt to determine why neither of these treatments improves speech. The etiology and the motor and speech characteristics of Parkinson's disease will be described. Deep brain stimulation and levodopa will be reviewed in detail to determine the effects of these treatments on both motor and speech function. Finally, evidence to suggest reasons why neither deep brain stimulation nor levodopa improve speech will be reviewed.

Etiology

A basic understanding of neuroanatomy is necessary for considering the etiology of Parkinson's disease (Duffy, 2005). The basal ganglia are located deep within the cerebral hemispheres and are closely related (both anatomically and functionally) to the substantia nigra. Both of these structures are interconnected and function together as a part of the basal ganglia control circuit. The basal ganglia control circuit is responsible for several functions: regulation of muscle tone and movements that support goal-directed activities, postural adjustments, and adjustment of movements to the environment (Duffy, 2005). Damage to the basal ganglia circuit results in a movement disorder in which an individual is unable to initiate voluntary movement and overall range of movement is reduced.

The substantia nigra is responsible for the production of dopamine. Dopamine is a chemical important for transmitting signals from one group of brain cells to another to facilitate movement ("Parkinson's disease," 2009). Therefore, when the neurons (nerve cells) of the substantia nigra are destroyed, the production of dopamine is reduced and its supply to the basal ganglia circuit is lessened (Duffy, 2005). Diminished dopamine is a crucial factor in the development of Parkinson's disease and the consequential symptoms. A loss of dopamine results in symptoms of Parkinson's disease such as reduced gross motor movement, tremors, and rigidity. Although the specific etiology of PD is unknown, degenerative diseases tend to be the most common causal factors. Any time that the circuitry of the basal ganglia is damaged, whether due to degenerative, vascular, neoplastic, toxic, traumatic, inflammatory, or metabolic diseases, a motor speech disorder can result (Duffy, 2005). An example of a toxic disease is environmental exposure to harmful chemicals. Although many hypothesized toxins may be linked to PD, no specific

toxins have been found in the brains of individuals with PD (Olanow & Tatton, 1999). Genetic factors may play a role in causing PD. According to Olanow and Tatton (1999), approximately 5–10% of PD patients have an genetically inherited form of Parkinsonism. The incidence of PD is greater in family members than in age-matched controls. Parkinsonism is a generic term that refers mainly to the symptoms of PD rather than its cause (Duffy, 2005). While the specific etiology (reason for reduced production of dopamine in the brain) of PD continues to be unknown, research has helped to explain the neurological condition which produces these PD symptoms.

Motor characteristics

The effects of PD on motor behaviors start on one side of the body and then progress to the opposite side until the motor behaviors of the entire body are affected. The major motor behaviors exhibited by patients with PD are bradykinesia, akinesia, hypokinesia, resting tremors, rigidity, and reduced postural reflexes (Schulz & Peterson, 1999). Bradykinesia is characterized by slow movement or difficulty beginning movements, while akinesia is characterized by immobility as if the person is 'frozen' and unable to move. Both bradykinesia and akinesia are key characteristics of PD 'off states,' when the dopamine levels in the brain are particularly low. Rigidity in persons with PD appears as stiffness or tightness in the body. [The terms 'hypokinesia' (reduced movement) and 'akinesia' (no movement) tend to be used interchangeably with 'bradykinesia.'] (Goberman & Coelhoe, 2002).

Hypokinesia is characterized by specific motor behaviors in patients with PD: festination, micrographia (small writing), and masked facies. Festination is a behavior

characterized as an acceleration in gait with small, jerky movements. Patients with PD exhibit festination by small movements during speaking, or by walking in rapid shuffling steps. In many patients with PD, there is difficulty initiating and terminating movement (Marquardt, 2010). Masked facies, another motor characteristic that may accompany PD, is defined as a masked facial expression in which the person does not smile or make any emotional expression (Duffy, 2005). PD also is accompanied by resting tremors. These tremors tend to take effect while the body is in a resting or still position, and decreases during voluntary movement. Although the head and limbs are most affected by resting tremors, the jaw, lips and tongue also can be affected.

PD is also characterized by reduced postural reflexes and irregular gait. According to Duffy (2005), "because postural reflexes are impaired, the patient may be unable to make adjustments to tilting or falling and have difficulty turning in bed or moving from a sitting to a standing position" (p. 188). Also, patients with PD tend to not swing their arms as they walk, which reduces their postural stability.

Speech characteristics

Speech problems develop when Parkinson's disease has progressed to the point where the entire body is affected by these movement-reducing behaviors. Approximately 60-90% of patients with PD are affected by speech deficits, and these deficits generally increase in severity with disease duration and severity (Plowman-Prine, Okun, Sapienza, Shrivastav, Fernandez, & Foote, 2009). The most common speech characteristics exhibited by patients with PD dysarthria are reduced articulatory gestures, vowel prolongation, voice disorders, and abnormalities in speech rate (Pinto, Ozsancak,

Tripoliti, Thobois, Limousin-Dowsey, & Auzou, 2006). Also, they may not use limb gestures that accompany speech. Consequently, individuals with PD are less likely to participate in conversations or have confidence in communication as compared to healthy aging adults (Trail, Fox, Ramig, Sapir, Howard, & Lai, 2005). These voice and speech deficits traditionally are attributed to the motor behaviors of PD (rigidity, bradykinesia, hypokinesia, and tremor). Reduced amplitude is a major speech difficulty for many patients with PD, as exhibited by the fact that 45% out of every 200 patients exhibit this difficulty.

CONSONANT ERRORS

Articulatory gestures tend to be imprecise. For stops (/p, b, t, d, k, g/), individuals with PD produce a continual emission of air causing them to be produced more like fricatives /f, h, s, v, sh, th, z/. Frication of phonemes is likely because they have a reduced ability to control articulators for speech, and thus are unable to stop airflow during speech production. This particular articulatory problem is referred to as 'spirantization.' In a study by Logemann and Fisher (1981), articulatory deficits demonstrated by patients with PD may have been a result of inadequate tongue elevation (Logemann & Fisher, 1981). When the patient is not able to elevate the tongue sufficiently, inadequate constriction for stops and fricatives occurs causing the sounds to be articulated imprecisely.

VOWEL ERRORS

Another speech difficulty often experienced by patients with PD is vowel prolongation. The articulators of patients with PD were determined to have difficulty attempting to make controlled, methodical movements for speech and to be less controlled and more immobile during vowels. Respiratory support for speech can be

measured by vowel prolongation time. Metter and Hanson (1986) reported that speakers with PD produced the shortest vowel prolongation times compared to normal speakers (Goberman & Coelho, 2002). They also found that vowel prolongation time decreases as PD progresses. Canter (1963) found that vowel prolongation time was decreased by an average of 50 percent compared to normal speakers (Goberman & Coelho, 2002).

VOICE DISORDERS

Voice disorders also are a frequent speech problem in individuals with PD. In fact, they tend to be the first speech-related problem to occur during the progression of PD and appear very early on in the disease process. In a study of 200 people with PD, 89 percent had voice disorders characterized by hoarseness, roughness, tremulousness, and breathiness (Logemann & Fisher, 1981). Duffy (2005) also noted that individuals with PD with articulation problems had voice problems as well. Duffy (2005) noted that "dysphonia can be the presenting and most prominent and debilitating speech feature" in individuals with PD (p. 197). Dysphonia can be described as an impaired voice that sounds harsh, breathy, and reduced in loudness. To most listeners, a patient with PD will sound strained and strangled, and will most likely vocalize with a whisper quality. Although few patients with PD have a true voice tremor, their voice may have an unsteady-like quality caused by the head and upper limb tremors. Tremulous lips, tongue, and jaw also contribute to a tremor-like voice. Logemann et al. (1981) found that vocal tremulousness was present in 14% of their 200 parkinsonian patients. Although motor movement may be severely impaired in individuals with PD, speech impairment is a serious concern as well.

TREATMENT FOR PARKINSON'S DISEASE

There are several surgical and pharmacological treatment methods for Parkinson's disease. The route of treatment will vary depending on the type and severity of the disease. However, since PD often is treated by a combination of surgical and pharmacological methods simultaneously, both forms of treatment will be considered.

Surgical treatments

Treatments for PD include ablative surgeries (i.e. thalamotomy, pallidotomy, subthalamotomy), transcranial magnetic stimulation (TMS), fetal cell transplantation, and deep brain stimulation (DBS). Ablative surgeries (thalamototomy, pallidotomy, subthalamotomy) involve the placement of a thermolesion on one or both sides of a specified area of the brain in order to improve the motor symptoms of PD. Ablative surgeries are considered to be destructive in the sense that they are producing a lesion in the brain. These procedures have been shown to improve motor function significantly in patients with PD when they are done unilaterally. However, when done bilaterally, there is a significantly higher risk of serious complications (Samra, Riklan, Levita, Zimmerman, Waltz, Bergmann, & Cooper, 1969). Some of these complications include dysfunction of speech, swallowing, balance, and memory in as many as 50% of patients (Follett, 2000).

DEEP BRAIN STIMULATION: THE GOLD STANDARD OF SURGICAL TREATMENT

While ablative procedures can provide substantial clinical benefits, the current surgical trend is moving toward deep brain stimulation (DBS) because it can provide

similar relief of symptoms without a destructive lesion. DBS has proved to be the most promising form of surgical treatment because it produces the most benefits and least number of risks and side effects compared to other surgical treatments. DBS is defined as a neurosurgery which "involves the placement of a four-channel depth electrode in the region of the subthalmic nucleus (STN) or globus pallidus interna (GPi) for delivery of therapeutic electric field stimulation by an implanted pulse generator (pacemaker)" (Barlow & Hammer, 2009, p. 362). Since DBS is providing electrical stimulation rather than creating a lesion in the brain, it produces significant improvement in PD symptoms without the adverse effects often caused by ablative procedures. DBS of the subthalmic nucleus has rapidly emerged as the routine method for treatment of idiopathic PD producing significant improvements in both motor function and quality of life (Barlow & Hammer, 2009). Since DBS has quickly become the gold standard of surgical treatment for PD, it will be discussed in greater detail as the major surgical method of treatment for PD symptoms. The effect of DBS on both motor and speech behaviors will be considered.

Deep Brain Stimulation: Effects of Motor Characteristics

The most promising targets for DBS include the pallidum (GPi) and the subthalmic nucleus (STN). According to Follett (2000), DBS targeting either the GPi or STN showed significant clinical improvement without serious side effects. Some of these improvements in motor movement include improvements in bradykinesia, rigidity, tremor, and gait. These improvements were so significant that many patients were able to reduce their concurrent levodopa intake by 30-56%.

The Unified Parkinson's Disease Rating Scale (UPDRS) is a scale used to evaluate the longitudinal effects of the disease progression of PD. The UPDRS is frequently used to evaluate the symptoms in patients with PD following DBS. UPDRS scores for patients following DBS-STN showed significant improvements. Motor scores on the UPDRS for patients who were off their medication saw a 43-71% improvement; and, activities of daily living scores improved by 30-60%. Overall, the research reflects the fact that DBS of the STN or the GPi results in significantly consistent improvements in motor movements for the patient with PD.

Deep Brain Stimulation: Effects on Speech Characteristics

While improvements in motor movement are a rather reliable outcome of DBS, the same point cannot be made about the impact on speech characteristics. In a review of seven studies assessing the speech outcomes of DBS-STN, the conclusion was that the procedure was not generally associated with a positive outcome on speech production in patients with PD (Iulianella, Adams, & Gow, 2008). The impact on speech appeared to vary from subject to subject. For example, one study investigated changes in acoustic measures of speech for patients who had received DBS-STN (Dromey, Kumar, Lang, & Lozano, 2000). Speech effects were reported to be "modest and inconsistent." While four of the seven patients experienced a small decrease in speech intensity, the remaining three patients experienced a slight increase in intensity during DBS. A study analyzed the effects of left and right DBS-STN separately in seven patients who had been implanted with a bilateral DBS device (Santens, DeLetter, Borsel, DeReuck, & Caemaert, 2003). The speech tasks of a 200 word reading passage and sustained "ah" were evaluated during varying STN stimulation conditions. No significant improvements in

speech were found. Although different aspects of speech are important for evaluation purposes, the most functional aspect is speech intelligibility. The ability to be easily understood is the most important factor in speech production. Overall, studies have shown that relative to baseline, speech intelligibility does not improve following DBS-STN (Tripoliti, Dowsey-Limousin, Tisch, Borrell, and Hariz, 2006). In fact, Tripoliti et al. (2006) reported that two patients in their study showed a 40% decrease in intelligibility following DBS-STN.

Speech effects in patients with DBS in general were insignificant and inconsistent. While most patients saw no changes in speech, there were a limited number that either received a modest benefit or even a substantial worsening of speech characteristics. Based on the research, it is uncertain as to why speech effects can vary so much between individual patients. Professionals and patients alike can safely assume that while DBS may significantly improve motor symptoms of DBS, the neurosurgery cannot be relied upon to improve speech symptoms. Based on the present research, the effect of DBS on speech has proved to be highly variable across facilities, measures, and patients.

Pharmacological treatments

Pharmacological treatments for PD include levodopa, dopamine agonists, and COMT and MAO-B inhibitors. Each of these drugs works in slightly different ways to alleviate symptoms of PD. Levodopa, which works to restore the brain's dopamine levels, is currently considered the most effective type of pharmacological treatment because it produces the greatest effects on limb function. Levodopa can provide benefit to the motor qualities of patients with PD. However, long-term use of the drug often can

result in complications that may occur later in the disease. Some of these complications may include wearing-off, dyskinesias, freezing episodes, and unpredictable 'on-off' fluctuations (Korczyn & Nussbaum, 2002). Alternative drugs (such as dopamine agonists and COMT and MAO-B inhibitors) have been considered to help either delay the use of levodopa or to reduce its administrative level. While dopamine agonists and COMT and MAO-B inhibitors complement the effects of levodopa, neither dopamine agonists, COMT inhibitors, nor MAO-B inhibitors alone can have the positive impact on motor movement that levodopa can (Schapira, Emre, Jenner, & Poewe, 2009). Given that levodopa is considered to be the most effective pharmacological treatment for symptoms of PD, it will be discussed further by examining the effects of levodopa on both motor and speech movements.

Levodopa: Effects on Motor Characteristics

Levodopa is the most effective method of pharmacological treatment for PD because it produces the greatest effect on motor function. Administration of levodopa serves to significantly reduce the progression of PD by reducing bradykinesia, tremor, rigidity, postural instability, and loss of motor skills (Freire & Santos, 2010). In fact, a significantly positive motor response to levodopa (exceeding 25%-30% reduction in the motor section of the UPDRS) is used as a diagnostic criterion for PD (Poewe, Antonini, Zijlmans, Burkhard, Vingerhoets, 2010). Levodopa is known to be so effective in the early stage of the disease that many symptoms of PD are significantly reduced and many people who had previously been diagnosed with PD seem almost asymptomatic. However, administration of levodopa cannot replace lost nerve cells.

There has been some controversy about when levodopa therapy should begin (Schapira et al., 2009). While levodopa initially shows satisfactory results in improving motor function, these results can begin to taper off with long-term levodopa use. Additionally, long-term use of levodopa may cause various motor complications (Schapira et al., 2009). Involuntary motor movements known as dyskinesias (which can manifest as twitching, twisting, and/or writhing) commonly develop in patients who take large doses of levodopa over the period of several years (Schapira et al., 2009). These motor complications eventually develop in the majority (approximately one-third) of patients after only two years of exposure to levodopa (Poewe et al., 2010). Once these drug-induced dyskinesias develop, the dosage of levodopa is usually reduced. However, the reduction of levodopa dosage is often accompanied by a return of the PD symptoms. Both doctors and patients must work to find a tolerable balance between the levodopa associated complications and the possible return of the PD symptoms. While levodopa can have serious side effects, the overall benefit of slowing the progression of PD continues to make levodopa one of the main options for treatment of motor disturbances in PD.

Levodopa: Effects on Speech Characteristics

Levodopa has proven to be the gold standard of treatment for PD. Studies examining the impact of levodopa treatment on speech have produced conflicting results. Sanabria, Ruiz, and Gutierrez (2001) obtained on- versus off-levodopa speech measures from individuals who had been taking levodopa continuously for several years (Adams & Jog, 2009). They showed improvements in several speech characteristics: vocal jitter, shimmer, single word intelligibility, and vocal tremor. Other studies have failed to show

improvements in vocal jitter and shimmer (Winkel & Adams, 1992), speech dysfluency (Goberman & Blomgren, 2003), maximum phonation time, sound pressure level, intelligibility, and rate of speech (Kompoliti, Wang, Goetz, Leurgans, & Raman, 2000). PD dysarthria, which is characterized by a "monotony of pitch and loudness, reduced stress, variable rate, short rushes of speech, imprecise consonants, and a breathy and harsh voice," is also a common symptom in PD (Pinto et al., 2004). As PD progresses, many patients develop PD dysarthria. According to Pinto et al. (2004), the effect of levodopa treatment on PD dysarthria have been shown to be variable. While some studies report improvements in intelligibility, others report no changes and a few studies even report worsening of the dysarthria (Marsden & Parkes, 1976; Critchley, 1976; Anderson et al., 1999). Long-term use of levodopa can have complications in motor function. The drug also can produce speech complications for patients who have taken it for several years. Complications include orofacial or respiratory dyskinesias, oromandibular dystonias, and neurogenic stuttering (Pinto et al., 2004). While levodopa has proven to be an effective form of pharmacological treatment for the motor symptoms of PD, the drug does not show promise in helping to improve speech problems associated with PD. Levodopa's effect on speech production in PD is apparently variable, and may have some negative effects on speech with long-term use.

An Important Question

While both deep brain stimulation (DBS) and levodopa improve the movement disorder aspect of the Parkinson's disease (tremors, bradykinesia, rigidity, etc.), neither form of treatment appears to have a significant positive impact on speech production.

Why do DBS and levodopa not help to significantly improve the speech quality of an individual with PD?

DISCUSSION

One theory about why neither deep brain stimulation nor levodopa improves speech in patients with Parkinson's disease is that speech activities and non-speech activities are organized differently neurologically. A treatment targeting the area of the brain that affects non-speech activities will not affect the area of the brain responsible for speech production. Consequently, speech and non-speech activities should be treated separately and would not be expected to improve simultaneously with the same form of treatment. Therefore, the reason why neither DBS nor levodopa help to improve speech is because these treatments target a 'non-speech' (i.e. motor function) area of the brain. Thus, speech functions will not improve and will have to be targeted separately and with another form of treatment. To assess this argument, studies of the neurological organization of speech and non-speech activities will be reviewed.

Nonspeech Oral Motor Therapy

Speech-language pathologists may use nonspeech oral motor therapy (NSOMT) to help facilitate speech production in children with speech development disorders or delay. NSOMT techniques and procedures include activities such as blowing, repetitive exercises of different muscle groups, resistance exercises (i.e. opening and closing the jaw under tension), clinician-assisted movement of the articulators, and sensory stimulation (i.e. applying vibration to the lips or tongue) (Ruscello, 2008). The rationale is that improving oral motor movements improves speech production. This rationale is based on a theory known as the "common effector perspective," which states that when the same effectors (structures) are used for different activities (i.e. speech versus

nonspeech) they are controlled by a common set of principles such as force of movement and timing (Bunton, 2009). While this theory may be plausible, supporting research is sparse.

Guisti-Braislin and Cascella (2005) studied four first-grade students who received 15 half-hour sessions of NSOMT to improve their articulation disorders. After the 15 sessions, there were minimal differences in their speech production when compared to baseline measures. Abrahamsen and Flack (2002) also investigated the efficacy of oral motor exercises on speech production. In a single-subject design study, a four-year-old with suspected developmental apraxia of speech received 10 hours of treatment based on non-speech oral motor exercises. Pre- and post- measures indicated no improvement in speech production. Finally, a study by Colone and Forrest (2000) studied two children who were identical twins with similar speech error patterns. While one twin received direct treatment of speech sound errors, the other twin received NSOMT. Each participant received the designated form of treatment over a period of seven sessions. The twin who received direct speech treatment demonstrated a positive change in sound production skills; however, the twin who received NSOMT did not demonstrate any improvement in speech production. Following the initial NSOMT, this twin received direct speech therapy and was then able to demonstrate a positive change in speech. Overall, the findings of the research on this topic indicate that oral motor exercises do not help to improve speech and therefore should not be used in clinical practice as a basis for improvement of speech function.

How does the ineffectiveness of NSOMT relate to why neither DBS nor levodopa help to improve speech in patients with PD? The fact that NSOMT does not improve speech production suggests that speech and nonspeech activities are organized differently

neurologically. NSOMT works to target motor function of the oral and facial structures. Because NSOMT is specifically targeting motor function, speech (which is neurologically separate from motor function) does not improve.

Functional Magnetic Resonance Imaging

Functional magnetic resonance imaging (or fMRI) is a relatively new form of magnetic resonance imaging (MRI). fMRI measures changes in blood flow as related to neural activity, and has become one of the leading methods for brain mapping. Recent studies have used fMRI to map speech and motor function in the brain, and many of these studies infer that speech and non-speech activities are organized differently.

Wildgruber, Ackermann, Klose, Kardatzki, and Grodd (1996), used fMRI to compare covert speaking (a task directly related to speech) to isolated vertical tongue movement (a non-speech task). Wildgruber et al. reported that (at the level of the primary motor cortex), speech movements were related to increased activation in the left motor strip, while non-speech activities resulted in increased bilateral symmetric activation. A similar study conducted by Riecker, Ackermann, Wildgruber, Dogil, and Gross (2000) compared neuronal activation of speech and non-speech tasks via fMRI. Riecker et al. (2000) found that lateral tongue movements were associated with bilateral cerebellum activation; however, speaking was accompanied by unilateral right-sided neuronal activation.

Apraxia of speech in adults is a disorder that is most commonly associated with stroke, and is associated with an inability to perform speech motor movements, while non-speech oral motor movements are typically intact. A study by Bonilha, Moser,

Rorden, Baylis, and Fridriksson (2006), compared speech and non-speech movements using fMRI to help determine where brain damage typically occurs to cause apraxia of speech. Controversy has long existed regarding apraxia of speech because it is uncertain whether the speech disorder is caused by damage to the insula or the inferior frontal gyrus. Bonilha et al. (2006) found that speech movements activated the left inferior frontal gyrus and non-speech tasks resulted in activation of the insula. Overall, these studies infer that fMRI demonstrates different neuronal activation patterns for speech versus non-speech activities. This difference in neuronal activation further implies that speech and non-speech activities are organized differently neurologically.

CONCLUSIONS

Although it is difficult to prove with certainty that speech and non-speech tasks are organized differently neurologically, the current research points to this concept being supportable. Speech-language pathologists and medical professionals alike must carefully examine the compelling evidence. Both clinical evidence in speech therapy as well as neuroimaging studies point to the theory that speech tasks and motor tasks are in different areas in the brain. While the research is limited, the findings are relatively consistent. This concept of different neurological organization provides a straightforward explanation as to why DBS and levodopa, which are both successful in treating motor function deficits in PD, do not result in any significant improvement in speech problems associated with PD. In order to attempt to improve speech function in patients with PD, a completely separate approach or form of treatment would be necessary. For example, while a patient with PD may get DBS or continue to use levodopa to treat motor dysfunction, they may need to use behavioral speech therapy as a means to improve their speech. Although DBS and levodopa are both excellent forms of treatment for patients with PD, it is essential that clinicians and medical professionals alike help to educate patients as to what they can expect (in regard to both motor and speech function) from either treatment.

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