An Examination between Laryngeal Physiology and Parkinson's Disease: Severity and Treatment

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Abstract

The purpose of this in-depth literature review is to examine the relationship between laryngeal physiology and Parkinson's disease in terms of the severity and possible treatment. This research aims to determine the distinct characteristics of Parkinsonian speech and possible causes of these speech deficits. In addition, a specific type of Parkinson's disease treatment, deep brain stimulation, was explored to determine effectiveness on laryngeal physiological deficits found in Parkinson's disease patients. Through synthesizing peer reviewed journals and various studies, data was examined in order to take an in-depth look at the unique relationship between laryngeal physiology and Parkinson's disease. Findings indicated that Parkinsonian speech characteristics include vocal tremors, breathiness, hoarseness, and decreased vocal projection possibly due to bowed vocal folds or incomplete glottal closure. Low frequency deep brain stimulation treatment may serve as a potential resource for mitigating speech and voice deficits, however results are inconclusive.

Key Words: Parkinson's disease, laryngeal physiology, phonation, deep brain stimulation, Parkinsonian speech

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In the communication sciences and disorders field, a quintessential anatomical structure is the larynx. Speech language pathologists are interested in studying the larynx because it serves an important role in the human body due to its many biological functions. For example, it protects the lower respiratory tract, creates a temporary air flow dam during a cough or sneeze, plays an important role during the glottal effortful closure reflex, and provides an open passage way into the esophagus for swallowing. The larynx also has an important non-biological function; it serves as the primary sound generator. Phonation consists of vibratory cycles in which the vocal folds move from their resting positions into an interactional position with the exhaled airstream to produce laryngeal tone. Through this process, the larynx utilizes air pressure and airflow in order to create laryngeal tone or a raw sound, which is the product of the exhaled airstream from the lungs interacting with the vocal folds in the larynx to produce a sound in the audible frequency range. In order to expand upon laryngeal physiology and its contribution to phonation, it is first necessary to identify and define the anatomical structures within the larynx.

In *Essentials of Anatomy and Physiology for Communication Disorders*, Seikel describes the larynx as an unpaired "musculo-cartilaginous structure located at the upper end of the trachea, which is comprised of the cricoid, thyroid, and epiglottis cartilages, as well as the paired arytenoid, corniculate, and cuneiform cartilages" (p. 128). The thyroid cartilage is the largest laryngeal cartilage shaped like a shield and articulates with the hyoid bone as well as the cricoid cartilage via the crico-thyroid synovial joint. The cricoid cartilage is shaped like a ring, rising up high in the back and rests on top of the trachea making it the most inferior cartilage out of all the laryngeal cartilages. The paired arytenoid cartilages are pyramid shaped and rest on the posterior,

superior, convex portion of the cricoid cartilage articulating with the cricoid cartilage on each side via the crico-arytenoid joint (p. 143). The most superior portion of the arytenoid cartilage is the apex, the most lateral portion is the muscular process, and the most anterior portion is the vocal process. The paired corniculate cartilages rest superior to the arytenoid cartilages and the cuneiform cartilages are located in the aryepiglottic folds, superior and anterior to the corniculate cartilage. Finally, the epiglottis attaches to the inside angle of the thyroid and bends over the opening to the larynx.

Understanding the anatomy of the larynx is necessary in order to understand how these cartilages function together in order to produce laryngeal tone. Seikel states, “The cricothyroid and cricoarytenoid joints are the only functionally mobile points of the larynx” (p. 146). The movement of the cartilages, specifically the thyroid and arytenoid cartilages, via the cricothyroid and cricoarytenoid joints is important to understand because it demonstrates the foundational mechanical processes of producing laryngeal tone, which will run through vocal mechanisms, refining and sophisticating this raw sound into voice. These joints perform two major movements with the goal of approximating the vocal folds in order to produce laryngeal tone. This goal is accomplished through rocking the thyroid and arytenoid cartilages down in the front and gliding the arytenoid cartilages medially along the facet for the arytenoid cartilage located on the cricoid cartilage. The vocal folds originate from the inside angle of the thyroid cartilage and insert into either the vocal process of the arytenoid cartilage or the muscular process of the arytenoid cartilage. By rocking the arytenoid cartilages forward and gliding them medially so that the tips of the vocal processes are approximated, the vocal folds are brought closer to the middle of the airway where they can interact with the exhaled airstream from the lungs resulting in phonation and producing the desired laryngeal tone.

It is necessary for speech language pathologists to have a strong foundational knowledge of both the larynx and laryngeal physiology in order to treat patients with communication disorders and diseases. Parkinson's disease (PD) is a neurodegenerative disease that affects the central nervous system, specifically motor movements resulting in tremors. While this disease affects many individuals, the etiology is still not quite clear. By further investigating Parkinson's disease such as its etiology, a possible genetic component, and common factors among individuals who are affected by this disease, the knowledge obtained will provide greater insight establishing a strong foundation to build upon and relate to the communication sciences and disorders field. In addition, there is a wide variety of treatments created and tested to mitigate symptoms. Deep brain stimulation (DBS) is a fairly recently developed treatment for Parkinson's disease. Its relevance to the communication sciences and disorders field is of high importance because Parkinson's disease affects motor movement including the movement of the cartilages in the larynx, which leads to problems in phonation and overall a person's ability to produce voice. Specifically, Parkinsonian speech contains speech characteristics such as mono-loudness, mono-pitch, and hypophonia. Speech language pathologists would benefit from specifically knowing the characteristics of Parkinsonian speech as well as determining what is not functioning properly in the larynx contributing to abnormal phonation in patients with PD. What is occurring at the level of the vocal folds in Patient's with Parkinson's disease, and how does it differ from individuals who are not affected by this disease? In addition, deep brain stimulation is important because speech language pathologists are interested in knowing specifically what areas of the brain should be stimulated in order to initiate the neural impulses that are sent to the laryngeal muscles aiding in vocal fold adduction and abduction to produce normal phonation during verbal discourse. What other areas contribute to laryngeal physiology and the production of laryngeal

tone allowing for the production of voice? These gaps are the purpose for an in-depth literature review of laryngeal physiology, Parkinson's disease, and deep brain stimulation as well as the factors that connect them in the communication sciences and disorders field.

First, it is important to understand the etiology of Parkinson's disease because with greater knowledge of the cause of this disease, it could potentially provide greater insight into preventing this disease from developing in an individual if it can be prevented. Another puzzling aspect specifically related to the communication sciences and disorders field is the relationship between Parkinson's disease and voice disorders. Parkinson's disease is known as primarily affecting motor movement resulting in tremors, the puzzling aspect in the communication sciences and disorders field comes into play when analyzing how Parkinson's disease specifically affects one's ability to produce laryngeal tone leading to voice. This vexing topic, however, goes beyond the comprehension of how Parkinson's disease affects one's speech. It involves identifying and comprehending what is exactly happening in individuals who have Parkinson's disease at the level of the vocal folds, identifying what is not functioning properly, and utilizing problem solving skills to test possible treatments. The primary focus involves figuring out how advanced treatment, such as deep brain stimulation, can target the specific areas of the brain responsible for speech production in order to improve one's voice and discourse abilities. Through an in depth literature review, it is necessary to examine how Parkinson's disease affects normal laryngeal physiology and the production of laryngeal tone.

The purpose of this in-depth literature review of laryngeal physiology is to identify and understand Parkinson's disease, which will involve exploring the etiology, how it affects the human body and whom this disease primarily affects. The goal is to examine the relationship between Parkinson's disease and the larynx, specifically by researching how this disease affects

the physiology of the larynx. With an understanding of treatments for Parkinson's disease, such as deep brain stimulation, it is necessary to assess how it interferes with brain functioning in an individual to assist the muscles of the larynx contributing to phonation. Finally, the goal is to explore how this deep brain stimulation treatment mimics the neural impulses to the brain communicating to laryngeal muscles to utilize air pressure and air flow to produce normal voicing in individuals with Parkinson's disease.

Overall, the larynx is an essential structure in the communication sciences and disorders field because of its non-biological function as the primary sound generator. Speech language pathologists understand the importance of this musculo-cartilaginous structure because of its contribution to the process of phonation and production of laryngeal tone. Phonation is comprised of vibratory cycles in which the cartilages of the larynx work together to approximate the vocal folds so that they can interact with the exhaled airstream yield the raw sound that will be refined by articulators contributing to one's voice. With a strong foundation of normal laryngeal physiology, speech language pathologists are interested in how normal laryngeal physiology is affected by different communication disorders and diseases. Specifically, Parkinson's disease affects motor movement including the movement involved in laryngeal physiology. Speech language pathologists are interested in how this disease affects phonation as well as exploring treatment that can help mitigate these issues affecting one's voice in patients with Parkinson's. Investigating Parkinson's disease and its etiology will help provide insight into what is not functioning properly at the level of the vocal folds. With that understanding, researching advanced treatment, such as deep brain stimulation, can provide solutions to treat the voicing problems associated with Parkinson's disease. The purpose of this research is to examine the relationship between Parkinson's disease and voicing disorders as well as examine deep brain

stimulation as treatment to target the areas of the brain responsible for sending neural impulses to the muscles of the larynx that engage in phonation in order to mimic the process of normal phonation resulting in laryngeal tone.

Background of Parkinson's Disease

With a strong understanding of the anatomy and physiology of the larynx, speech language pathologists utilize this strong foundation of knowledge to help individuals who are experiencing problems resulting in deficits of their speech or language abilities. One population of patients speech language pathologists work with include Parkinson's disease (PD) patients. Parkinson's disease is a common neurodegenerative disorder that affects approximately one million Americans. It occurs when certain neurons become impaired or die resulting in a lower production of the neurotransmitter, dopamine (*Parkinson's Disease*, 2017). This disease primarily impairs motor movement in individuals around the age of sixty, however signs and symptoms may manifest through the body as early as the age of forty.

Signs and Symptoms of Parkinson's Disease

According to the Mayo Clinic, early signs and symptoms of this nervous system disorder are difficult to recognize at first and may even be hardly recognizable in the early stages of this disease (*Parkinson's Disease*, 2020). These signs and symptoms mainly include "tremors, bradykinesia, rigid muscles, impaired posture and balance, loss of automatic movements, soft, slurred, or monotone speech, and impaired writing abilities" (*Parkinson's Disease*, 2020). Among these general symptoms, "PD manifests itself through speech disorders, which can be observed as early as 5 years before the diagnosis" (Vaiciukynas et al., 2017). Since speech disorders can be observed years prior to the diagnosis of PD, it is important to be able to recognize the speech characteristics of PD. These perceptive speech and voice symptoms include

"soft, breathy, monotone voices perceived by the patient to be normal loudness" (Merati et al., 2005). Parkinsonian speech affects one's ability to speak at a normal volume because while their voice displays vocal deficits such as a soft and breathy, the PD patient is not aware of their speech deficits because they perceive it as normal loudness. This discrepancy is problematic when exchanging communicative messages because the PD patient may not speak loud enough for his/her communicative partner to hear their message, which results in ineffective communication.

In addition, there is less variability in pitch and loudness, the phonation is difficult to sustain, and "Parkinsonian speech is characterized by hypophonia, dysphonia, hypokinetic articulation, dysprosodia, rush, hesitant and/or dysfluent speech" (Perju-Dumbraca et al., 2017). Parkinsonian speech contains problems with volume such as mono-loudness, intensity, vocal quality such as hypophonia, reduced range of articulation, and pitch such as monotone affecting their ability to communicate or to be understood leading to expressive language problems. As these symptoms worsen, everyday functioning can become difficult and frustrating for the individual, especially communication, which negatively influences their life.

Specifically, at the level of the vocal folds, "videolaryngostroboscopy studies have shown that as Parkinson's disease progresses, glottis competence and vocal fold vibration are compromised, with a bowed closure configuration, phase asymmetry, aperiodicity, voice tremor, and mucosal wave abnormalities" (Perju-Dumbraca et al., 2017). PD interferes with one's ability to properly phonate because of issues involved in vocal fold vibration. The bowed closure, asymmetry, and aperiodicity of the vocal folds all contribute to the impaired process of producing laryngeal tone since the vocal folds are not able to approximate close enough to vibrate, resulting in the speech deficits seen in Parkinsonian speech. Parkinson's disease is a

concern for speech language pathologists because they help their clients express and communicate their wants, needs, thoughts, and feelings through speech and language. Parkinson's disease interferes with the patient's ability to communicate with others around them, which leads to ineffective communication, negatively affecting their life.

Parkinson's Disease Etiology

In terms of the etiology of Parkinson's disease, there are scientific suggestions stating that this disease could result from genetic/hereditary factors as well as neurological components. Scientists believe that the cause of this disease could be due to specific genetic mutations. Genetic research has shown that, "11 genes have been mapped by genetic linkage with six genes identified: SNCA, UCH-L1, PRKN, LRRK 2, PINK 1, and DJ-1 genes" (Davie, 2008). With these six specific genes identified as contributors to the onset of Parkinson's disease when mutated, it strongly supports the hypothesis that etiology is due to genetic components as well as mutations to these genes. By further analyzing these genes, it seems like "the proteins that they encode for are providing significant insight into the disease mechanisms that may be responsible for PD and other neurodegenerative disease" (Davie, 2008). Davie explains that an abnormal aggregation of proteins, including the ones encoded for by the six specific genes, is a major component of lewy bodies. Lewy bodies add a neurological component to the etiology of Parkinson's disease because they are a "pathological hallmark of PD" (Davie, 2008). Essentially, these are clumps of substances in brain cells that contain proteins from mutated genes that cannot be broken down and serve as an indicator of Parkinson's disease. Other pathological hallmarks of Parkinson's disease can be attributed to neurological factors. This includes a loss of cells within the substantia nigra portion of the brain and as this disease progresses, there is a loss of cells in other areas of the brain including areas of the midbrain, basal forebrain, and neocortex

(Davie, 2008). Specifically, the loss of cells within the substantia nigra portion of the brain is the most concerning because cells in that area of the brain are responsible for producing dopamine, a neurotransmitter that sends signals to the brain regarding motor control (Henderson, 2017). As the brain slowly starts losing these cells, there is not enough dopamine to maintain normal motor functions affecting one's ability to walk, talk, and engage in other motor movements. This results in noticeable signs and symptoms such as tremors, bradykinesia, rigid muscles, impaired balance and posture, decreased ability to engage in unconscious movements, and soft or slurred speech (*Parkinson's Disease*, 2020). While there seems to be a genetic and neurologic component contributing to the etiology of this disease manifesting itself through various signs and symptoms negatively affecting everyday life, there are also potential environmental factors that contribute to the development of this disease. Davie states that an urban environment is linked to an increased risk of PD due to the exposure to pesticide usage and wood preservations (Davie, 2008). In addition, "the only consistent environmental factor is a strong negative correlation between cigarette smoking and the development of the disease" (Davie, 2008). These environmental factors suggest that harmful environmental chemicals and toxins such as one that come from pesticides, wood preservations, and cigarettes are correlated with the development of PD. With scientific evidence supporting both nature and nurture with regards to the cause of PD, it appears that both genetics and environmental factors contribute to the development of this disease. Ultimately, each case in an individual is unique and therefore the exact cause of this disease is still unknown to this day, but science has provided greater insight into the nature and nurture factors that increase one's risk of developing PD.

The Effect of Parkinson's Disease on Phonation and Speech

Parkinson's disease seems to have a negative effect on the vocal folds affecting phonation. In the Blumin et al. (2004) study, he sought out to discover common laryngeal findings in patients who are contemplating deep brain stimulation treatment in order to mitigate the voice and speech problems associated with their case of Parkinson's disease. Some common voice problems associated with this disease include "vocal tremor, decreased volume, hoarseness, low volume, monotone quality, various degrees of breathiness, and decreased vocal projection" (p. 253). These voice problems are significant because they have a negative impact on the quality of a person's voice, affecting the PD patient's expressive language skills as well as the communicative partner's ability to hear and understand what the patient with PD is communicating. Speech language pathologists are interested in discovering what exactly causes these voice problems since it leads to deficits in speech intelligibility, which is within their scope of practice.

Bowed Vocal Folds, Aperiodic Voicing, and Micropertubations

It is important to understand the main characteristics of Parkinsonian speech as well as explore possible causes to these distinct speech characteristics. By studying patients with PD for two years, researchers found that their voices became more aperiodic with increased micropertubations of frequency and amplitude (Galaz et al., 2018). Confirming these phonatory abnormalities, Perju-Dumbrava et al. (2017) claims that "videolaryngostroboscopy studies have shown that as PD progresses, glottis competence and vocal fold vibration are compromised, with a bowed closure configuration, phase asymmetry, aperiodicity, voice tremor, and mucosal wave abnormalities" (p. 2). With multiple studies exploring the characteristics of Parkinsonian speech, the main characteristic seems to be bowed vocal folds, aperiodic voicing, and micropertubations. A possible explanation for the aperiodic voicing and increased micropertubations of frequency

and amplitude that Galaz found could be due to vocal fold bowing. Blumin et al. (2004) states that “vocal fold bowing is a consistent laryngeal finding in patients with PD” (p. 257). In normal phonation, the vocal folds flutter and come together through adduction. When approximated close enough, they are able to interact with the exhaled airstream creating laryngeal tone. However, when vocal folds are bowed, the vocal folds are not able to approximate close enough to produce that laryngeal tone, therefore the resulting sound is breathy and contributes to hypophonia. The laryngeal muscles are rigid as well because when vocal folds are bowed, the muscles are not able to fully relax. As a result, bowed vocal folds affect frequency (pitch) and amplitude because the laryngeal muscles are rigid in a contracted state and therefore are not relaxing enough, which creates extra tension in these laryngeal muscles contributing to that muscular rigidity.

Another study agrees with Blumin et al. because they found vocal tremors as the main acoustic difference in individuals with PD in comparison to the control group. This study utilized acoustic analysis software to analyze the vocal acoustics of individuals with PD and individuals who were not affected by this disease and their findings showed perturbations during a sustained production of the low, front vowel /a/ (Zarzur et al., 2010). These studies present a strong and homogenous notion that bowed vocal folds are not only a characteristic present in PD patients, but also contribute to the phonatory characteristics of Parkinsonian speech such as hypophonia; however, there are other factors to consider in addition to bowed vocal folds that could account for the vocal deficits associated with Parkinsonian speech.

Thyroarytenoid Muscles

Another factor, in addition to bowed vocal folds, that might contribute to the speech characteristics of Parkinsonian speech is the laryngeal musculature. By using electromyography

(EMG), amplitudes of the thyroarytenoid (TA) muscles of the larynx were compared individuals with PD and individuals without PD. The findings suggest that "reduced levels of TA muscle activity may contribute to the characteristic hypophonic voice disorders that frequently accompany PD" (Baker et al., 1998). This study found that there was less activation of TA muscles in PD patients signifying that PD is associated with bradykinesia (slow movement) of the TA muscles of the larynx since this study focused on the variability or range of the TA activation. The TA muscles are paired muscles that comprise the muscle mass of the vocal folds. When the TA muscles contract, the arytenoid cartilages move in a way that allows the vocal folds to become longer and thinner, resulting in a higher frequency/pitch. When these muscles relax, the vocal folds become shorter and thicker, resulting in a lower frequency/pitch. TA muscle activation is essential for laryngeal tone to be produced. When researchers found that there was less activation of the TA muscles in the individuals with PD, it is possible that this lower amount of TA activation is correlated with PD and the characteristics of Parkinsonian speech, such as bradykinesia, because the TA muscles are not activated adequately enough to produce proper laryngeal tone.

Zarzur et al. (2010) also examined the laryngeal muscles believing that there are more factors than bowed vocal folds that contribute to the characteristics of Parkinsonian speech. Possible contributing factors could involve insufficient breath support or respiration, improper resonance of the velum, articulation deficits, and the cricothyroid (CT) and thyroarytenoid (TA) muscles rhythmically contracting (p. 42). This study agrees with Blumin et al. (2004) because it supports the homogenous notion that bowed vocal folds are abducted too far apart, even when the CT and TA muscles are activated and therefore the vocal folds cannot adduct or approximate close enough to interact with the exhaled airstream to produce normal laryngeal tone. In addition,

these laryngeal muscles are contracting when they should be relaxing, contributing to vocal fold rigidity, supporting the Blumin et al. (2004) findings. Essentially, these findings indicate that bowed vocal folds as well as laryngeal muscles are two major components that contribute to the speech deficits seen in PD patients.

Laryngeal Somatosensory Deficits

Also supporting the notions from Zazur et al. (2010), Hammer and Barlow (2010) express the idea that "respiratory and phonatory control are influenced by laryngeal somatosensory function and that speech-related deficits in PD are related to abnormal laryngeal somatosensory function" (p. 401). Somatosensory deficits are common symptoms due to impairment of the peripheral and central nervous systems and especially in PD patients, the emphasis revolves around the motor fibers. This study found that these "laryngeal somatosensory deficits were associated with greater reductions in respiratory driving pressure" (Hammer & Barlow, 2010). These laryngeal somatosensory deficits associated with other factors contributing to Parkinsonian speech, specifically with reduced respiratory driving pressure. Individuals need sufficient air pressure and airflow from the lungs to supply adequate subglottic air pressure to abduct the vocal fold or engage them in the process of phonation, creating laryngeal tone within the audible frequency range.

By analyzing all these particular studies, it is evident that bowed vocal folds are a consistent factor contributing to the laryngeal deficits in the normal production of laryngeal tone affecting speech, contributing to the characteristics of Parkinsonian speech. While bowed vocal folds are a consistent factor in these studies, it would be remiss to neglect the other variables such as laryngeal muscle activity, specifically the movements of the cricoarytenoid and thyroarytenoid muscles, and somatosensory deficits that also contribute and interfere with the

production of laryngeal tone in individuals with PD. By examining the relationship between PD and laryngeal physiology, these studies indicate that PD affects phonation and the production of laryngeal tone due to bowed vocal folds, inadequate laryngeal muscle activation, and somatosensory deficits, all contributing to the Parkinsonian speech characteristics such as vocal tremors, hoarseness, monotone quality, low volume, breathiness, decreased vocal projection, and micropertubations.

Deep Brain Stimulation Treatment

Although there is still no cure for Parkinson's disease, there are medications and a variety of treatments available for PD patients in order to mitigate the symptoms of this disease. A specific type of treatment used to accomplish this task is deep brain stimulation. In *Deep Brain Stimulation for the Treatment of Parkinson's Disease,* Volkmann explains that this type of treatment for PD utilizes high-frequency electrical stimulation in order to target areas of the brain, mimicking a lesion, but avoiding the destruction of essential brain tissue (p. 6). Deep brain stimulation treatment "is accomplished by implanting an electrode with four contacts into the target area within the brain and connecting it to an internal pulse generator usually located in the chest area" (p. 6). This treatment is unique in the fact that it is able to utilize electrical stimulation to directly affect areas of the brain without the need of brain surgery or concern of damaging tissues in the brain in order to help reduce PD symptoms. Once the electrodes are implanted in the brain, wires attached to a device called the implantable pulse generator, which generates these electrical impulses, interconnect them to each other. This implantable pulse generator is placed near the patient's collarbone and serves as the "pacemaker for the brain" (*Deep Brain Stimulation*, 2019). This treatment essentially involves an implantation of an electrode in the brain targeting specific parts of the brain imitating a lessoning effect of the

ventrolateral thalamus, internal pallidum, and subthalamic nucleus at frequencies <100 hz (Volkmann, 2020). These areas of the brain are targeted because they mainly control motor movement, so the electrical signals being sent to the ventrolateral thalamus, internal pallidum, and subthalamic nucleus in order to help mitigate the motor symptoms such as tremors and akinesia. Volkmann reviewed two studies to see the affect that thalamic DBS had in Parkinsonian tremor. One study found that thalamic DBS had the ability to suppress Parkinsonian tremors because it was "associated with significant reduction of rCBF (regional cerebral blood flow) in the ipsilateral putamen, sensorimotor cortex, supplementary motor area, and contralateral cerebellum" (Volkmann, 2004). Increasing the blood flow to these specific motor controlling areas of the brain have been associated with improvements in motor functions. The significance of this finding is that it provided insight into the idea that thalamic DBS could potentially alleviate the tremors associated with PD by inactivating "the involuntary running of a central motor program for alternating movements involving the basal ganglia-thalamocortical loop" (Volkmann, 2004). However, this result conflicts with the second study, which found less brain activity during thalamic DBS suggesting that DBS to another portion of the brain, the internal pallidum. With inconclusive results, further research examining the portion of the brain that should be targeted with DBS in order to improve motor deficits in patients with PD.

Deep Brain Stimulation in Relation to Parkinson's Disease Speech Deficits

Deep brain stimulation has had positive effects on improving limb functions in patients with PD, but the effect this treatment has on laryngeal control and the production of phonation contributing to voice and speech is still yet to be explored in greater depth.

Subglottic Air Pressure

Based on current research, it is understood that in order to produce laryngeal tone within the auditory frequency range, it is necessary to have sufficient respiratory driving pressure of air known as the subglottic air pressure in addition to other aerodynamic measures. A study conducted by Michael J. Hammer involved examining the effects of bilateral subthalamic nucleus deep brain stimulation (STN-DBS) on aerodynamic measures of respiratory and laryngeal control in patients with PD given different frequencies of stimulation ranging from 170Hz to 145 Hz. He found that, "PD participants exhibited changes in respiratory and laryngeal control with STN-DBS" (Hammer et al., 2010). Some of these changes included an increase in respiratory driving pressure and vocal fold closure for voicing (Hammer et al., 2010). The most notable finding of this study was the negative correlation between stimulation frequency and aerodynamic measures of respiratory and laryngeal control (Hammer et al., 2010). Hammer found that as one lowers the amount of stimulation frequency of STN-DBS, the respiratory driving pressure/subglottic air pressure and the closure of the vocal folds increases. This increase found in the respiratory driving pressure and vocal fold closure for voicing is important especially for patients with PD because typical speech deficits found in these patients' results from incomplete vocal fold closure or not enough subglottic air pressure to abduct the vocal folds so that the membranous portions of the vocal folds are pushed apart. Incomplete vocal fold closure may be due to vocal fold bowing which is a common characteristic of PD patients. When vocal folds are bowed, they are not able to completely adduct or approximate close enough together so that they can interact with the exhaled airstream resulting in a breathy, hypophonic voice. This finding also suggests that lower frequency STN-DBS may be beneficial for respiratory and speech deficits in patients with PD. By lowering the frequency of the STN-DBS, vocal fold closure is able to increase and enough subglottic air pressure is able to build up

beneath the vocal folds creating a strong driving force needed to abduct the vocal folds engaging in the process of phonation.

Speech and Gait Side Effects

A similar study conducted by Farris and Giroux reviewed patients that reported improved gait and speech within a half hour after turning off STN-DBS and after adjusting stimulation. Their results showed that "turning stimulation off revealed reversible speech and/or gait stimulation side effects within 30 minutes" (Farris & Giroux, 2015). In addition to these results, they concluded, "that disease progression may not be the only cause for a decline in speech and or gait after STN-DBS and detection of stimulation side effects requires up to 30 minutes of time off stimulation" (Farris & Giroux, 2015). The researchers determined that the speech and/or gait side effects such as worsening speech, swallowing, balance and gait were due to the stimulation treatment since these side effects were reported within 30 minutes of the STN-DBS off time. It also recognizes that it is important to determine the appropriate amount of stimulation for the patient and their needs based on their symptoms and deficits. The average amount of stimulation provided through the STN-DBS treatment was 159.7 Hz, but the researchers acknowledge the fact that for therapy and clinical purposes, there is an upper threshold of a therapeutic window, which they found to be approximately 130 Hz. This study supports the findings in Hammer's study because both believe that lower frequency stimulation of STN-DBS treatment has positive effects on PD patients' laryngeal control contribution to speech benefits.

High Frequency vs Low Frequency DBS

In order to differentiate the benefits of high frequency STN-DBS in comparison to low frequency STN-DBS, "high frequency STN-DBS has become a neuromodulation treatment of choice for advanced PD and is associated with marked improvements in limb motor function"

(Arocho-Quinones et al., 2017). This finding suggests that higher frequency STN-DBS is more beneficial for PD patients experiencing limb function deficits since patients reported improvements in their limb motor function after receiving high frequency STN-DBS. While limb control benefits from higher frequency STN-DBS, "laryngeal and respiratory sensorimotor control may benefit more from low frequency STN-DBS" (Arocho-Quinones et al., 2017). With a frequency as low as 60 Hz, these researchers found that laryngeal function benefitted from this lower stimulation from the STN-DBS treatment in comparison to higher stimulation which benefits limb motor function. This is because limb muscles are greater and stronger than laryngeal muscles, and therefore, exert a greater force and need higher stimulation to have better control of these movements (Arocho-Quinones et al., 2017).

Another potential explanation for the differences between motor limb benefits and speech benefits through STN-DBS was proposed in a study examining the relationship between voice and motor disabilities of PD. The researchers suggest that the motor speech control system differs from peripheral motor control mechanisms and thus concluding that speech actually contains a separate motor control mechanism from the motor control mechanism (Majdinasab et al., 2016). This finding indicates that the motor control mechanism is responsible for the axial symptoms of PD such as balance, posture, and instability. On the other hand, the speech control system is responsible for non-axial (appendicular) symptoms such as tremors and involuntary muscle movements. By synthesizing the findings from Arocho-Quinones and Majdinasab, it could be possible that higher stimulation frequency benefits motor limbs. Higher frequency STN-DBS stimulates the motor control mechanism with greater force, improving the axial symptoms of PD. Lower stimulation frequency benefits laryngeal muscles because it stimulates

the phonatory or speech control mechanism improving the appendicular symptoms such as vocal tremors in the voicing of patients with PD.

These studies explored the effect of STN-DBS and speech related issues associated with PD. All three agree that STN-DBS can have positive speech benefits for these patients by increasing the respiratory driving pressure in order to build up a sufficient amount of subglottic air pressure needed to abduct the vocal folds during the process of phonation. In addition, this treatment increases vocal fold closure, which helps mitigate the characteristic of breathy vocal quality present in many patients with PD due to bowed vocal folds. It is suggested to provide lower frequency STN-DBS treatment in order to target the speech mechanisms, mitigating the voice and speech problems associated with PD. However, another study assessed 47 PD patients (22 treated with bilateral STN-DBS of a frequency of 135.4 Hz +/- 17.0 Hz and 25 treated medically with LEDD levodopa equivalent daily dose of 842 mg +/- 292 mg) specifically using laryngoscopic examinations to compare the two groups and their laryngeal functions. Their laryngoscopic findings demonstrated that the "PD-DBS patients showed a significantly higher rate of incomplete glottal closure and hyperadduction of the false vocal folds than PD-Med patients" (Tsuboi et al., 2015). Other research conducted also found that "deep brain stimulation… has shown limited improvements to voice and speech function despite significant changes in limb movements" (Merati et al., 2005). In fact, one study reported that STN –DBS actually reduced spontaneous speech intelligibility and that "two of seven subjects were observed to have post-operatively worsened dysarthria" (Spielman et al., 2011). These studies challenge the effectiveness of DBS in terms of improving speech deficits in PD patients because their findings indicate that patients who were treated with PD-DBS actually showed a higher rate of incomplete glottal closure and hyperadduction of the false vocal folds in comparison to the

patient treated medically. The incomplete glottal closure leads to a breathier tone of vocal quality and hyperadduction of the false vocal folds contributes to more tense vocal folds and well as the rigidity of the vocal folds, which are vocal deficits in PD patients. These studies also qualify that DBS was more beneficial for correcting limb movements rather than for treating voice and speech implying that DBS should be a treatment for individuals looking to correct their larger motor musculature. In fact, Spielman found that subjects actually displayed worse speech symptoms after the patients received STN-DBS treatment.

By comparing the findings of all these studies and analyzing the effect of STN-DBS on Parkinsonian speech, it is important to analyze the amount of stimulation frequency utilized. Tsuboi's study used a range of stimulation from about 118.4 Hz to 152.4 Hz. The study conducted by Arocho-Quinones utilized a stimulation frequency significantly smaller than Tsuboi's study as well as Hammer's and Farris & Giroux's study. Based on Archo-Quinones conclusion that lower frequency stimulation benefits laryngeal musculature, it is possible that Tsuboi's study did not have congruent findings with the other studies because the amount of frequency stimulation utilized in the treatment plan was too high. Higher stimulation frequency tends to benefit the larger musculatures since they require more force and therefore laryngeal musculatures, being smaller, exert less force and need a smaller amount of frequency to improve vocal fold closure and better the patient's speech.

Implications

These results are inconclusive, as they do not provide a clear answer regarding the effect of DBS treatment on speech indicating that future research in this specialized area is necessary. Perhaps in future studies, researchers should explore a range of stimulation frequencies in order to determine the optimal amount of stimulation needed that specifically targets the laryngeal

muscles in order to mitigate the voice and speech issues associated with PD. If any quantity of stimulation is beneficial for improving speech and phonation deficits in PD patients, it is necessary to acknowledge and recognize that each individual is different and displays a unique, personal response to treatment.

Conclusion

In conclusion, communication is vital in order for an individual to communicate their wants, needs, thoughts, feelings, opinions, and ideas. Many accomplish this through utilizing their voice. The act of speaking and producing voice begins with the larynx at the level of the vocal folds as the arytenoid cartilages glide medially along the axis of the cricoid cartilage while the thyroid cartilage simultaneously rocks forward in order to adduct the vocal folds, approximating them close enough to interact with the exhaled airstream provided by the lungs. This produces laryngeal tone, a raw sound that becomes refined through the oral and nasal cavities and ultimately turns into voice. Disruptions in the phonatory process can be attributed to a multitude of problems; however, a common disruption in this process is due to disease.

By examining the relationship between laryngeal physiology and Parkinson's disease (PD), this research specifically sought out to explore Parkinson's disease, the etiology, and the general population affected. In addition, the main objective was to understand how does Parkinson's disease affects phonation and speech and determine some of the characteristics of Parkinsonian speech. Finally, another purpose was to look at deep brain stimulation treatment as a potential treatment and determine its effectiveness for targeting speech deficits in Parkinson's disease patients.

Through analyzing various Parkinson's disease literature and studies, PD is a common neurodegenerative disease that mainly affects motor movements due to lower productions of the

neurotransmitter, dopamine. There have been questions regarding the etiology of this disease, but studies agree that both nature and nurture contribute to its etiology. Genetic factors include a mutation of the genes: SNCA, UCH-L1, PRKN, LRRK 2, PINK 1, and DJ-1 genes in addition to an abnormal aggregation of proteins, including the ones encoded for by the six specific genes, contributing to lewy bodies. Environment factors such as exposure to pesticides have also been correlated with PD, which is why the etiology of this disease is best described as a combination of both genetic and environmental factors. Approximately one million Americans are affected by this disease around the age of sixty. While motor movements are a common sign and indicator of this disease, PD can also manifest itself thorough speech deficits by interfering with the process of phonation, which is why speech language pathologists need to be aware of this disease and ways in which they can provide therapy to help mitigate these speech deficits.

Specifically looking at how PD affects voice and phonation, studies have described Parkinsonian speech as breathy, hoarse, mono-loud, low volume monotone voice with vocal tremors and micropertubations. A possible explanation for these characteristics could be attributed to bowed vocal folds found in many PD patients. When the vocal folds are bowed, the vocal folds are abducted too far apart, even when the CT and TA muscles are activated and therefore the vocal folds cannot adduct or approximate close enough to interact with the exhaled airstream to produce normal laryngeal tone. In addition, incomplete glottal closure is another explanation for the speech deficits seen in PD patients because the vocal folds are not adducted enough to minimize the glottis and utilize the subglottic air pressure efficiently to turn into laryngeal tone. It is also important to recognize that these speech deficits could be affected by other factors including lower activation of the laryngeal muscles and somatosensory deficits. All

of these factors contribute to vocal characteristics like breathy voice lacking a variety of tone, loudness, and pitch in addition to vocal tremors, which are all seen in Parkinsonian speech.

After understanding the etiology of PD and how it affects speech and voice, the next objective was to look at treatment for this disease. While there are many treatments available for PD patients, this study specifically looked at deep brain stimulation (DBS). This type of surgical treatment involves utilizing an implantable pulse generator device to generate electrical impulses at various frequencies, functioning like a pacemaker for the brain or order to minimize PD symptoms. Deep brain stimulation studies have utilized a wide variety of frequencies. When comparing low frequency and high frequency DBS treatment, multiple studies have seen that lower frequency DBS provides adequate stimulation to the laryngeal muscles contributing to improved speech while other studies have actually seen worsened symptoms. These inconclusive results indicate that further research is needed to truly understand the effectiveness of DBS on speech in PD patients. In order to explore DBS and its relationship with phonation, future research should focus on determining a DBS frequency threshold to define a range of stimulation frequency to target laryngeal muscles and improving speech and voice deficits as well as a range of stimulation frequency that targets improving motor movements.

Implications from this research indicate that every individual is unique displaying different combinations of PD symptoms and different reactions, both positive and negative, to various treatments. It is important to implement a personalized treatment plan for each PD patient as each person will respond to the treatment differently and it is most important to cater to that specific individual's needs and treat the most severely displayed symptom in order to improve his/her overall quality of life.

Overall, Parkinson's disease negatively affects phonation and speech making it difficult for the individual to communicate with others or be understood by their communicative partner due to Parkinsonian speech characteristics, which is why this is a significant topic in the field of communication sciences and disorders. Speech language pathologist prioritize their patients through providing treatment and therapy to improve their communication abilities because everyone deserves a voice and every voice deserves to be heard.

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