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Acoustic analysis of Parkinsonian speech I: Speech characteristics and L-Dopa therapy

Alexander M. Goberman[a,*] and Carl Coelho[b]
[a]*Department of Communication Disorders, Bowling Green State University, Bowling Green, OH, USA*
[b]*Department of Communication Sciences, University of Connecticut, Storrs, CT, USA*

Abstract. This paper reviews the literature pertaining to Parkinson's disease (PD) and the speech dysfunction typically associated with PD, including the effects on respiration, phonation, articulation, resonance, and prosody. The effect of treatment with the drug L-Dopa is also examined, along with the effect of L-Dopa treatment on Parkinsonian speech. This paper is the first of a two-part series. Part two examines the literature pertaining to the fluctuations that can occur during treatment with L-Dopa, the speech changes associated with these fluctuations, and methodological issues affecting the examination of fluctuations and PD speech.

1. Introduction

James Parkinson, in his 1817 essay on the "Shaking Palsy" was the first to describe a disease referred to as "paralysis agitans", now known as idiopathic Parkinson's disease (PD). Parkinson [52] noted that PD patients often became "scarcely intelligible" as the disease progressed. While PD speech has been examined extensively since this first observation, the mechanisms underlying PD speech are incompletely understood. This paper presents a review of the literature with regard to PD and the speech dysfunctions associated with PD, along with L-Dopa treatment and the speech changes associated with L-Dopa treatment.

Idiopathic Parkinson's disease (PD) is a progressive neurological disease that affects over 1 million people in North America [36]. Approximately 10% of patients report symptoms before age 40, and incidence increases with advancing age [26]. The exact etiology of idiopathic PD is unknown [5], but a number of hypotheses have been proposed. Age is the most consistent risk factor for PD, and family history of PD is second, suggesting the possibility of a genetic component [36]. Diet has also been proposed as a possible factor relating to the onset of PD. The discovery that brief exposure to the street drug MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) can cause Parkinson's-like symptoms, led to the belief that PD is caused by exposure to toxins [57]. Lang and Lozano [36] state that there is most likely a genetic predisposition to PD, which, in combination with certain environmental conditions, may lead to the development of PD.

Parkinson's disease is characterized by the progressive death of dopaminergic neurons primarily in the substantia nigra pars compacta, but also in other areas of the brain [5,34,36]. The substantia nigra is the origin of the nigrostriatal pathway, which travels to various structures within the basal ganglia [36]. The dopamine deficiency in this nigrostriatal pathway and the basal ganglia account for most of the typical features of PD. Once the brain is no longer able to compensate for this dopamine loss, there are a number of effects which can occur. Typical symptoms include muscle rigidity, akinesia, bradykinesia, and tremor [5,27]. The rigidity is often accompanied by a feeling of tightness in the muscles. The muscles become resistant to movement in all directions, and through the full range of motion. Akinesia refers to an inability to initiate movement,

*Address for correspondence: Alexander M. Goberman, Ph.D., CCC-SLP, Department of Communication Disorders, Bowling Green State University, 200 Health Center Building, Bowling Green, OH 43403, USA. Tel.: +1 419 372 2518; Fax: +1 419 372 8089; E-mail: goberma@bgnet.bgsu.edu.

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and bradykinesia is characterized by slow movements once initiated. The tremor which occurs with PD is most pronounced at rest, and decreases with purposeful movement. While these and other motor symptoms of PD may start unilaterally, they do eventually become bilateral, as they increase in severity.

In summary, while the exact cause of PD still eludes researchers, a great deal is known regarding the brain structures involved in the disease process. Even though there can be significant variability in the presentation and progression of the disease, the cardinal motor symptoms of rigidity, bradykinesia, and tremor, appear consistently in PD.

2. PD speech

The speech dysfunction resulting from PD is typically classified as hypokinetic dysarthria. The term hypokinetic refers to reduced amplitude of movement [43]. While PD is the most common disorder associated with hypokinetic dysarthria, there are a number of other etiologic factors associated with this type of dysarthria. In the following literature review, all subjects studied had idiopathic PD unless otherwise noted.

Because it would be beyond the scope of this review paper to discuss all research that has been completed on speech in PD, the following review selected studies of speech acoustics in PD. As there are fewer studies examining speech in relation to L-Dopa treatment, non-acoustic speech studies are included to augment the acoustic studies in these sections. A number of other literature reviews concerning Parkinsonian speech have been published with varying foci [4,13,34,58].

The perceptual characteristics of various types of dysarthrias were examined by Darley, Aronson, and Brown [15,16]. They determined the salient pitch, loudness, respiration, prosody, and articulation characteristics, as well as overall general impressions for each of the classifications of dysarthria. The authors served as listeners, and ranked the perceptual prominence of 38 speech characteristics after listening to speech samples from 212 patients. The characteristics noted to be commonly associated with hypokinetic dysarthria included monopitch, reduced stress, mono-loudness, imprecise consonants, and inappropriate silences. Other perceptual speech characteristics occurring in PD included short rushes of speech, and a harsh breathy voice quality [15]. A number of researchers have found that the most salient features of PD speech were related to phonatory impairment, with articulation being the second most affected speech subsystem [42,45,67].

2.1. Respiration

The rigidity associated with PD can often lead to a disruption of the respiratory process which serves to generate airflow and air pressures for speech. Deficits in respiration may affect the speaker's ability to produce normal phrasing and intensity (i.e., loudness). In addition, a decrease in respiratory pressure may cause further deficits in phonation and articulation [i.e., decreased loudness and decreased ability to alter loudness; 54].

Solomon and Hixon [61] examined speech breathing in 17 patients with idiopathic PD. They found that the respiratory changes associated with PD had an effect on speech during reading and monologue tasks. Specifically, the PD patients produced fewer syllables per breath group, and spoke for less time per breath group than age-matched normal speakers. These patients demonstrated either reduced or inefficient use of respiratory support for speech. Respiratory support for speech may also be measured through vowel prolongation time. Although they did not calculate a mean, Metter and Hanson [48] reported that PD speakers produced the shortest vowel prolongation times compared to normal speakers. King et al. [35] further report that vowel prolongation time decreases as PD progresses. Canter [8,9] found that vowel prolongation time was decreased by an average of fifty percent compared to normal speakers.

Others examined intensity range and ability to vary intensity as a measure of respiratory function. Compared to control speakers, PD speakers have been found to have overall lower intensity levels [12,28,55], deficits in maintaining intensity levels [25], and deficits regulating intensity in response to external cues [24]. Further, Canter [9] found that, when asked to repeat a syllable at four different loudness levels, PD patients produced a smaller intensity range than normal control speakers. The deficits in intensity range and control suggest that PD patients may exhibit decreased breath support and control for speech. The overall reduced respiratory support in PD patients may be evident during reading, monologues, and vowel prolongation, as well as during non-speech tidal breathing [61,62].

2.2. Phonation

In examining phonation in PD speakers, Le Dorze, Ryalls, Brassard, Boulanger, and Ratte [38] found no mean fundamental frequency (F_o) differences between patients with PD and age-matched control speakers for

a sentence reading task. Doyle, Raade, St. Pierre, and Desai [18] found F_o to be significantly higher for PD patients when measured during sustained vowel productions. Doyle et al. [18] examined sustained /a/ produced by 12 mild-to-moderate PD patients who were off medication. They found that the PD patients had significantly higher F_o than age-matched normal speakers. Similar results were found for German-speaking males and females producing vowels [23], as well as for females producing Spanish vowels [31]. Increased F_o has also been found in PD patients when speech was examined during reading passages and monologues [8, 28, 48]. Further, Metter and Hanson [48] found that F_o continued to increase as severity of PD increased. The increased F_o in PD patients is generally attributed to rigidity of the laryngeal musculature, which results in increased stiffness of the vocal folds.

A number of researchers have found increased F_o range and variability in PD patients compared to normal speakers [18,23,31,33,67]. These researchers examined vowel prolongation and measured F_o range, standard deviation of F_o [18,67], or jitter [23,31,33]. In prolonged vowels, Doyle et al. [18] found increased F_o range for PD females, and no difference in range for males. Zwirner and Barnes [67] found increased standard deviation of F_o for PD patients in prolonged vowels. Similarly, increased jitter has been found in prolonged vowel production [23, 31, 33]. This increase in F_o range and variation was assumed to reflect an impaired ability to maintain the laryngeal muscles in a fixed position for vowel prolongation.

A third phonatory measure that has been studied in PD is voice onset time (VOT). The measure of VOT is defined as the duration of time from articulatory release of a consonant to the onset of voicing for the following vowel [40]. Because VOT changes in PD are generally attributed to phonatory disruptions, VOT is categorized here as a phonatory measure. Forrest, Weismer, and Turner [22] found increased mean VOT in PD patients. They attributed this finding to deficits in coordination of the laryngeal musculature. Other researchers have noted decreased VOT in PD patients [21]. Weismer [63] proposed that rigidity of the laryngeal musculature caused reduction in vocal fold opening compared to normal speakers. The PD patients, therefore, were able to achieve full vocal fold closure in a shorter amount of time, as evidenced in a shorter VOT [63].

Other research has found that subjects with PD have deficits in the coordination of phonation and articulation. Some PD patients have been shown to continue voicing into a voiceless consonant in the post-vocalic position [10,63], while some demonstrated a delay in initial consonant voicing. Canter [10] reported that some subjects omitted initial /l/ and /r/ sounds. The patients appeared to move their supra-laryngeal articulators appropriately, but voicing did not accompany this articulation, leading to an omission of an initial consonant.

Overall, PD patients generally present with increased mean F_o, which is attributed to stiffness of the laryngeal musculature. In addition, they often demonstrate increased F_o variation in prolonged vowels, deficits in producing normal VOT, and discoordination in the timing of phonation. See Fig. 1 for a summary of the phonatory deficits associated with PD, and the relationship between speech measures and the anatomy of PD.

2.3. Articulation

In examining articulation in subjects with PD, a number of researchers have reported that stop consonants (p, t, k, b, d, g) were imprecise, and produced as fricatives [10,41,63]. Logemann and Fisher [41] noted that the articulatory deficits may have been a result of inadequate tongue elevation and resulting inadequate constriction for stops and fricatives. Ackermann and Zeigler [3] compared the intensity of stop consonant production in 12 PD and 12 normal speakers. These authors found that normal speakers demonstrated significantly decreased intensity at the moment of stop closure (i.e., oral closure). The PD patients did not show any decrease in intensity at the moment of stop closure, therefore complete closure may not have been achieved. This may have been an example of reduced amplitude of articulator movement, or reduced articulator strength, leading to an inability to adequately close off the oral cavity in PD.

Another method of evaluating articulatory skills in patients with PD has been the use of oral diadochokinetic (DDK) tasks. Typically DDK tasks involve production of syllable trains containing consonant-vowel combinations with bilabial, alveolar, and velar places of articulation, such as /p^p^p^.../, /t^t^t^.../, or /k^k^k^.../ [20]. The DDK rates are used to examine the patient's ability to make rapidly alternating articulatory movements [60]. A number of issues relating to the use of DDK rates in PD speech are raised by Ackermann, Konczak, and Hertrich [2]. First, the impaired prosody (i.e., rate of speech) characteristic of PD speech could hypothetically cause a decrease or an increase in DDK rates produced, in spite of ade-

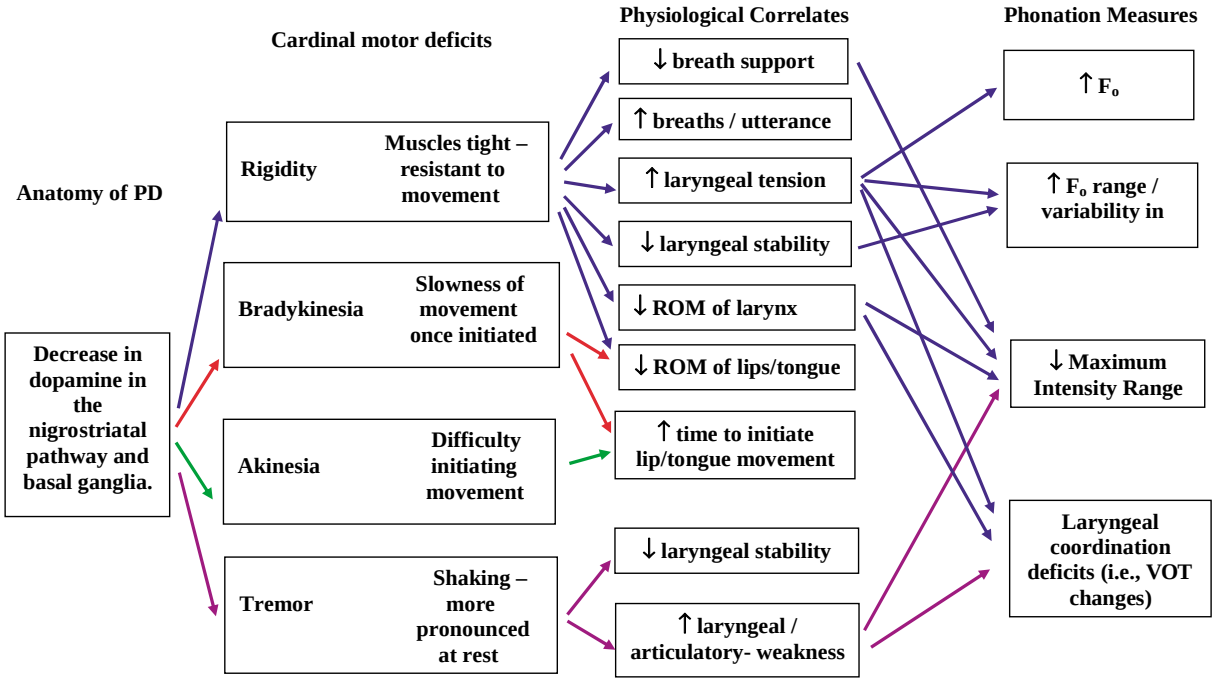

Fig. 1. Hypothesized relationship between anatomy of PD and speech phonation measures. Note: ↑ = Increased; ↓ = Decreased; ROM = Range of motion; F_o = Fundamental frequency; VOT = Voice Onset Time.

quate articulatory movement. Second, there may be a trade-off between amplitude of articulator movement and rate of speech. Non-acoustic studies have shown that the latter issue may affect the results of PD patients' productions of DDK rate tasks. For example, Ackermann et al. [2] examined DDK rates in two PD patients using an optoelectric movement analysis system. They reported that these patients had unimpaired DDK rates, but the patients achieved these rates at the expense of amplitude of movement. They adjusted for the abnormally slow movement of the articulators (bradykinesia) by reducing the amplitude of movement (articulatory undershoot). Ackermann, Hertrich, and Hehr [1] reported similar results for 17 PD patients. Some patients apparently used articulatory undershoot to successfully compensate for bradykinesia, however the more severe patients were unable to fully compensate. These severely involved PD patients produced abnormally slow speech in spite of their attempts to compensate with articulatory undershooting.

Vowel production may also be affected by deficits in articulator control and mobility as evidenced by differences in vocal tract resonances (i.e., formants). Zwirner and Barnes [67] found increased variability of first formant (F1) values during vowel prolongations, indicating possible articulator instability. The area created by plotting the first and second formant (F1–F2) values for the four corner vowels can be a metric of tongue movement [64]. Speakers with PD have been found to have reduced F1–F2 vowel space, compared to control speakers, but this difference was not found to be significant [64]. Connor, Ludlow, and Schulz [11] examined F1 and F2 transitions from syllable repetitions to infer information about articulator movement. Both F1 and F2 transition rates were flatter compared to control subjects. Similarly, Flint et al. [21] examined F2 characteristics for PD and normal subjects and found flatter F2 transition rates in the PD patients during sentence reading. These results were presumed to represent reduced speed of articulator movement during the tasks examined.

In summary, PD patients generally demonstrate deficits in articulation which affect oral closure for stop production and the ability to quickly move the articulators for DDK tasks. Also, amplitude and velocity of lip and mandible movements have been shown to be defective, in addition to slowed articulator movement during vowel productions. See Fig. 2 for a summary of the articulatory deficits associated with PD, and the relationship between speech measures and the anatomy of PD.

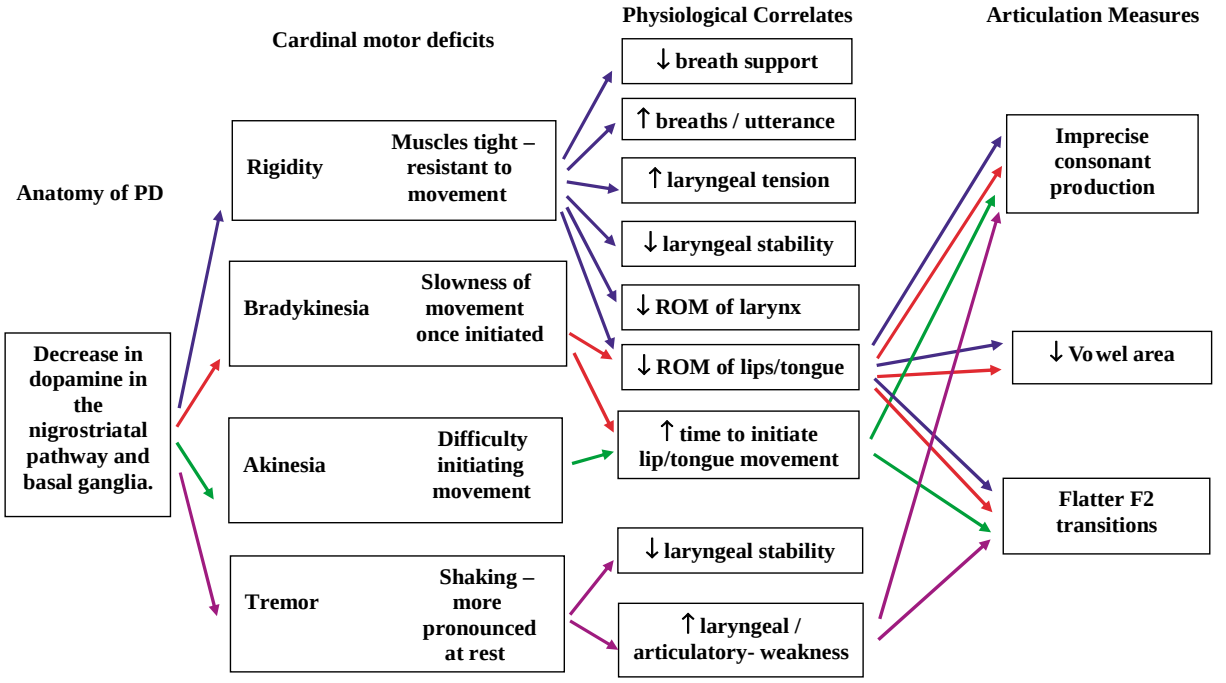

Fig. 2. Hypothesized relationship between anatomy of PD and speech articulation measures. Note: ↑ = Increased; ↓ = Decreased; ROM = Range of motion; F2 = Second Formant.

2.4. Resonance

There is a paucity of research examining the characteristics of resonance in PD speech. The reason for this shortage may be that deficits in this area of speech production are not readily apparent in the speech of PD speakers [19]. Darley et al. [15] did not include resonatory disturbances among the perceptually salient features of hypokinetic (PD) speech. Hoodin and Gilbert [29] found no significant difference in nasality between PD and age-matched normal speakers, as rated by seven trained judges. On the other hand, Ludlow and Bassich [44] reported that nasality was a strong perceptual feature in differentiating the speech of patients with PD and normal speakers. Acoustic analysis has shown evidence of excess nasality in the speech of patients with PD [19]. Duffy [19] further concluded that excess nasality in PD patients may be the result of slow movement and rigidity of the muscles involved in the velopharyngeal mechanism.

2.5. Prosody

Prosody is the term applied to the natural variations in pitch, intensity, and rhythm occurring during running speech [32]. A number of studies have found that patients with PD have impaired speech prosody. Comparing the F_o of the final syllable of a sentence produced as both a question and a statement has been used to examine prosody [37, 38]. Le Dorze et al. [38] examined F_o differences in 20 question-statement pairs produced in the speakers' native French language. They compared the F_o of the last syllable in each sentence and found that the 10 PD patients produced significantly smaller F_o differences compared to age-matched normal speakers. While the normal speakers produced the questions with higher F_o on the last syllable, the PD patients did not produce this F_o difference. Le Dorze et al. [37] investigated various types of dysarthria including hypokinetic dysarthria, and found a similar low mean F_o difference for question-statement pairs produced by dysarthric speakers. Another semantically governed frequency change was used in differentiating a noun phrase (e.g., black board) from a compound noun (e.g., blackboard). Darkins, Fromkin, and Benson [14] examined the prosody of 30 male PD patients and age-matched normal speakers. Their results indicated that normal speakers produced a significant frequency declination in the second word of compound nouns, and not in the second word of noun phrases. The PD patients did not make this distinction, as there was no difference in frequency between noun phrases and compound nouns.

Fundamental frequency range has been measured in sentences to examine prosody. Studies indicate a significantly reduced F_o range and variability in PD patients producing sentence length material. Canter [8, 9] investigated speech production in 17 PD males, and noted decreased F_o range during syllable production and during paragraph reading. Flint et al. [21] found decreased F_o range in 30 PD patients compared to 31 normal speakers during a reading task. Metter and Hanson [48] examined F_o variability in a reading passage and found a significant decrease in F_o variability compared to normal speakers. This variability decreased further as severity of PD increased. More recently, Jimenez-Jimenez et al. [31] also found decreased F_o variability in sentence productions of untreated PD patients. Normal speakers typically demonstrate a high F_o range and variability during reading tasks, corresponding with normal rising and falling intonations during speech. A decrease in F_o variation during reading tasks may reflect a prosodic deficit.

Prosodic intensity changes have also been examined [6,48]. Caekebeke et al. [6] examined emotion-related intensity changes, with subjects producing sentences containing varying emotional content (i.e., angry versus neutral). The 21 PD patients produced smaller intensity changes than normal control subjects. The authors note that the greatest difference occurred in the production of sentences with the emotion anger. The PD patients did not produce the intensity changes necessary to signify anger [6]. Metter and Hanson [48] also reported reduced ability to use intensity changes, stating that PD patients produced significantly smaller intensity variation compared to normal speakers during reading of a standard passage. In summary, patients with PD demonstrate reduced ability to use pitch and intensity changes to signify semantic or emotional differences during sentence and paragraph reading.

Rate of speech has been shown to be influenced by prosodic disturbances in PD. The rate disturbance associated with PD can cause speech rate to be accelerated or slowed. When compared to normal speakers, PD patients can exhibit fast speaking rates [21,63]. Weismer [63] stated that PD patients may produce speech at a faster rate because of articulatory difficulties, in which patients might "blur contrasts" between different speech sounds, causing an increase in rate. Impaired self-timing for motor movements has also been offered as an explanation for the increased rate of speech sometimes seen in PD [2]. Alternatively, Ludlow, Connor, and Bassich [46] found that PD subjects demonstrated slower speaking rates than control subjects, but that this difference was not significant. Still other research has found that there are no group differences between the speaking rates of PD subjects and normal speakers, while some individual PD subjects exhibited slow rates and others exhibited fast rates [8]. Patients with mild PD have been shown to have relatively normal rates of speech, while patients with more severe symptoms of PD demonstrate abnormally slow and fast rates [48]. As these extreme rate disturbances were in both directions (i.e., slower and faster), the mean rate differences between PD and control subjects were not significant [48]. Overall, patients with PD demonstrate production deficits in frequency and intensity changes, variations in speech rate, and pause characteristics of reading tasks. See Figure 3 for a summary of the prosody deficits associated with PD, and the relationship between speech measures and the anatomy of PD.

3. Treatment for PD

In the 1950s, a decrease in parkinsonian symptoms was noted after an accidental lesioning in the brain of a PD patient. This led to efforts to treat PD with lesions of the thalamus, globus pallidus, or other brain structures [59]. In surgical treatment of PD, lesions have been made in the region of the thalamus (thalamotomy), or the internal segment of the globus pallidus (pallidotomy). This type of surgery has been shown to be reasonably successful in relieving the rigidity and tremor in PD, but it is not without its drawbacks [50]. There are often dangers and severe side effects to lesioning any part of the brain, and such surgical techniques are often only used after other treatment options have been exhausted [50]. Other forms of treatment for PD include fetal cell transplantation, deep brain stimulation, and pharmacological treatments.

After a depletion of the neurotransmitter dopamine was discovered in the brains of PD patients, attempts were made to reintroduce dopamine into the brain [5]. As dopamine itself does not cross the blood-brain barrier, it was discovered that the chemical precursor to dopamine, L-Dopa, does pass freely into the brain, and can then be converted into dopamine within the brain [5,66]. While early reports of the efficacy of L-Dopa ranged from complete reversal of parkinsonian symptoms, to no effect [66], the benefit of L-Dopa on parkinsonian symptoms is now widely accepted. Early in the history of L-Dopa administration, large quantities of L-Dopa were required to achieve a therapeutic effect since it was often converted into dopamine

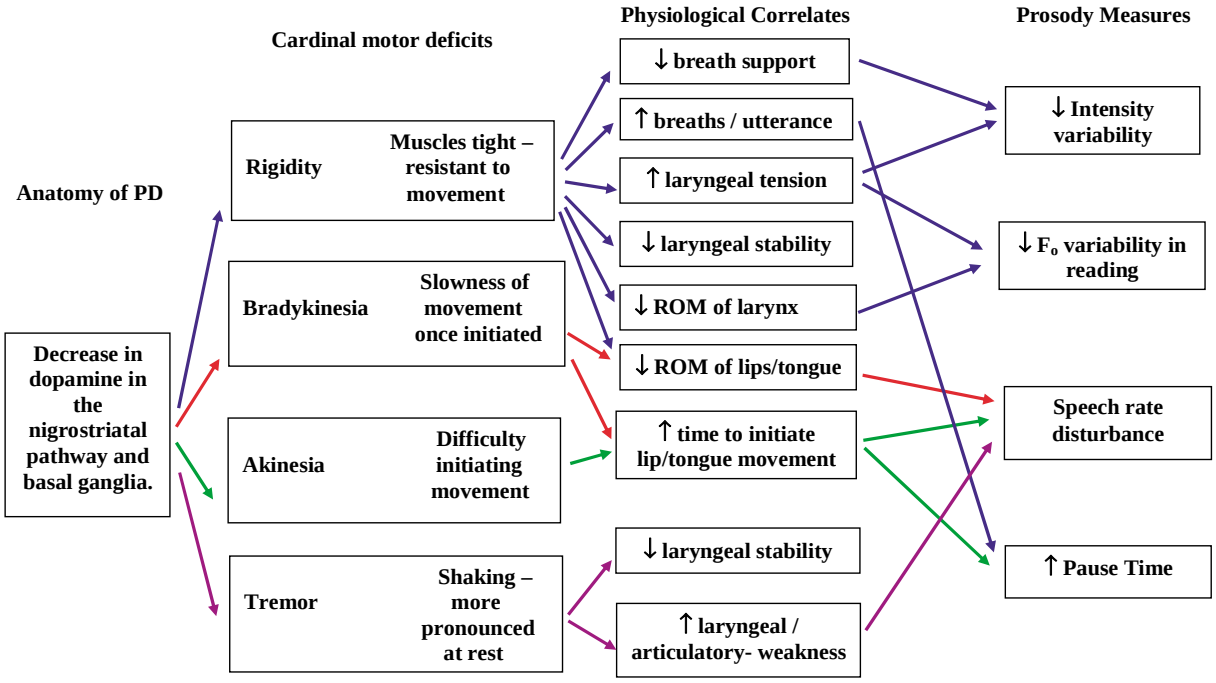

Fig. 3. Hypothesized relationship between anatomy of PD and speech prosody measures. Note: ↑ = Increased; ↓ = Decreased; ROM = Range of motion; F_o = Fundamental frequency.

before entering the brain. This process is called peripheral decarboxylation. An advancement was made in the 1970s when L-Dopa was combined with carbidopa to form Sinemet. Carbidopa decreases peripheral decarboxylation, enabling a greater portion of the administered drug to enter the brain. The combination of carbidopa with L-Dopa in Sinemet works as well as L-Dopa alone, but at a much lower dosage, and with fewer side-effects [30]. Other pharmacological treatments for parkinsonism are often prescribed in addition to L-Dopa, including dopamine agonists [51], monoamine oxidase – MAO-B [30], and controlled release Sinemet [30].

L-Dopa, alone or in combination with other dopamine enhancing drugs, has a number of benefits for the parkinsonian patient, as well as a number of side effects which may occur years after initial L-Dopa administration. After initially starting L-Dopa therapy, there is often a period of two to five years in which there is a smooth and stable response to the drug [17,51]. During this period of time (often called the Levodopa honeymoon period) the severity of parkinsonian symptoms is decreased (i.e., antiparkinsonian benefit) in most patients. The original clinical trial report characterizing the effect of L-Dopa before it was available for general use reported at least minimal decrease in symptoms in 34 of 38 patients studied [66]. These antiparkinsonian benefits were noted primarily in the areas of rigidity and bradykinesia. A smaller, while still significant, benefit was seen in parkinsonian tremor [66]. Yahr et al. [66] state, "Patients who had been unable to arise from a chair, walk unassisted, feed themselves, or care for personal hygiene could once again perform these routine activities of daily living." (p. 58).

Schulz and Grant [58] recently completed a review of the effects of an array of different PD treatments on speech and voice. The present paper, however, focuses specifically on the effects of L-Dopa on voice and speech production in PD. The completion of the present review was somewhat limited by the fact that many research reports do not include specific information on their recording protocols. As a result, some assumptions had to be made to categorize the research into the categories of PD Speech, and Speech and L-Dopa Treatment. The research reviewed in the PD Speech section (above) included comparisons of PD patients and control subjects, and an effort was made to exclude post-treatment or post-medication studies from this category. The research in the Speech and L-Dopa Treatment section (below) includes articles that examined speech before versus after initiation of L-Dopa based treatment. It was assumed that none of the subjects in these studies were experiencing fluctuations.

3.1. Speech and L-Dopa treatment

Prior to the use of L-Dopa therapy for treatment of PD, the pharmacological and surgical treatments available were often not effective in improving speech function [8]. However, speech improvements have been documented since the emergence of L-Dopa in treatment of PD [e.g., 53]. Improvements have been reported in various speech characteristics including overall intelligibility, and the features of phonation, articulation, and rate of speech.

The effect of L-Dopa on overall speech characteristics was examined by Quaglieri and Celesia [53]. Fourteen PD patients who had not received any surgical treatment were examined before and after initiation of L-Dopa therapy. A subjective 'global' speech score (on a scale of 1 to 5) was given to each of the patients before and after treatment. While these authors found little speech change with the addition of L-Dopa (2 of 14 patients demonstrated improved speech), others have found subjective overall speech improvement after L-Dopa administration [47,49]. Based on a counting task (1 to 10) and a spontaneous speech task, Mawdsley and Gamsu [47] reported a significant increase in speech intelligibility after initiation of L-Dopa treatment, although no objective data were given to support this result. Significant speech intelligibility improvements were also noted with L-Dopa initiation in a double blind placebo study of 18 PD patients [49]. Test stimuli included words and short phrases, and speech intelligibility was ranked from least to most intelligible by 10 untrained listeners.

Wolfe, Garvin, Bacon, and Waldrop [65] examined the speech characteristics of PD patients before starting L-Dopa, and again after a stable motor response to the drug was achieved. Three speech pathologists evaluated a 98 word reading passage and rated adequacy of voice quality, pitch variation, and articulation on a five point equal-appearing interval scale. Based upon these ratings, the authors found voice quality improvements as well as improvements in pitch variation and articulation. Other studies have found quantitative L-Dopa related improvements in articulatory performance [7,39]. Caliguiri [7] measured labial rigidity through passive displacement of the lower lip using a transduction system. Labial movement was measured through a strain gauge system attached to a headmount. Results indicated that non-speech labial rigidity was decreased, and amplitude of labial movement was increased after L-Dopa treatment. Leanderson et al. [39] noted abnormal lip muscle activation in PD patients during rest and during movement, as detected through EMG patterns. The abnormal patterns were eliminated during both rest and movement after starting L-Dopa. While the relationship between non-speech oral motor movement and speech movement has been debated [7], speech-related labial movements have also been shown to improve. With EMG analysis of lip function during speech, Nakano et al. [49] noted that abnormal tonic lip movements were eliminated with L-Dopa treatment.

Conflicting results have been reported on changes in speaking rate after initiation of L-Dopa therapy. Examining perceptual adequacy of speaking rate, Rigrodsky and Morrison [56] reported significant improvements after administration of L-Dopa. Based on a 7 point perceptual rating scale, average "speech rate adequacy" score improved from 3.8 to 4.9. Conversely, Wolfe et al. [65] reported no measured or perceived speech rate changes after administration of L-Dopa. These conflicting reports may have been expected based upon the speech rate variability in untreated PD patients. Specifically, untreated PD patients may demonstrate increased [21, 63], decreased [46], or relatively normal [48] speaking rates.

In summary, various speech measures have been shown to improve with the administration of L-Dopa. Subjective evaluations of PD speech revealed improvements in intelligibility, voice quality, pitch variation, and articulation. Objective analysis revealed improvements in non-speech and speech related labial movements.

4. Summary

Speech disorders commonly associated with Parkinson's disease were reviewed in this paper, in addition to the general medical characteristics of PD. L-Dopa is the most common and most effective treatment for the symptoms of PD, and L-Dopa treatment can result in dramatic improvements in motor function, as well as overall speech intelligibility, voice quality, pitch variation, and labial movements [e.g., 65]. This paper is part one of a two part series. Part two examines the literature pertaining to the fluctuations that can occur during treatment with L-Dopa, the speech changes associated with these fluctuations, as well as methodological issues affecting the examination of L-Dopa fluctuations on PD speech.

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