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Quantifying Lenition as a Diagnostic Marker for Parkinson's Disease and Atypical Parkinsonism

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Abstract: Objective: This study aimed to evaluate lenition, a phonological process involving consonant weakening, as a diagnostic marker for differentiating Parkinson's Disease (PD) from Atypical Parkinsonism (APD). Early diagnosis is critical for optimizing treatment outcomes, and lenition patterns in stop consonants may provide valuable insights into the distinct motor speech impairments associated with these conditions. **Methods:** Using Phonet, a machine learning model trained to detect phonological features, we analyzed the posterior probabilities of continuant and sonorant features from the speech of 142 participants (108 PD, 34 APD). Lenition was quantified based on deviations from expected values, and linear mixed-effects models were applied to compare phonological patterns between the two groups. **Results:** PD patients exhibited more stable articulatory patterns, particularly in preserving the contrast between voiced and voiceless stops. In contrast, APD patients showed greater lenition, particularly in voiceless stops, coupled with increased articulatory variability, reflecting a more generalized motor deficit. **Conclusions:** Lenition patterns, especially in voiceless stops, may serve as non-invasive markers for distinguishing PD from APD. These findings suggest potential applications in early diagnosis and tracking disease progression. Future research should expand the analysis to include a broader range of phonological features and contexts to improve diagnostic accuracy.

Keywords: Parkinson's disease (PD); atypical Parkinsonism (APD); consonant weakening; motor control/planning; machine learning

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1. Introduction

Parkinsonism encompasses a range of neurodegenerative disorders characterized by common motor symptoms such as tremors, rigidity, bradykinesia, and postural instability. Parkinson's Disease (PD) is the most prevalent form, whereas Atypical Parkinsonism (APD), which includes conditions such as Multiple System Atrophy (MSA), Progressive Supranuclear Palsy (PSP), Corticobasal Degeneration (CBD), and vascular Parkinsonism (VaP) presents with additional symptoms and generally progresses more rapidly [1]. Accurate differentiation between PD and APD is critical due to their distinct pathophysiologies, treatment responses, and prognoses [1,2]. Traditional diagnostic methods largely rely on clinical observations, including early falls, marked cognitive decline, and poor response to dopaminergic therapies in APD. However, these methods can be subjective, which often leads to misdiagnoses [1,3].

Recent advancements in neuroimaging and genetic testing provide promising tools to improve the differentiation of typical Parkinson's Disease (PD) from Atypical Parkinsonian Disorders (APDs). Techniques like neuromelanin MRI and diffusion tensor imaging help

identify brain degeneration patterns specific to PD, while genetic markers, such as LRRK2 and GBA mutations, link to distinct PD subtypes with varying symptom profiles (see [4] for a review).

Dysarthria, a motor speech disorder affecting muscle control in speech, is commonly observed in Parkinsonian patients. Recently, speech analysis has emerged as a valuable tool in distinguishing between Parkinson's Disease (PD) and Atypical Parkinsonian Syndromes (APSs). For example, a quantitative speech analysis study was 95% accurate in differentiating APSs from PD, and 75% accurate in distinguishing Progressive Supranuclear Palsy (PSP) from Multiple System Atrophy (MSA), highlighting its diagnostic potential [5]. In the study, which included 15 PD, 12 PSP, and 13 MSA patients, and 37 healthy controls, dysarthria was present in all Parkinsonian patients. However, the varying combinations of hypokinetic, spastic, and ataxic dysarthria reflect the distinct underlying pathologies of these conditions. Specifically, PD was characterized by hypokinetic dysarthria, marked by reduced loudness and monopitch. In contrast, MSA displayed ataxic dysarthria, noted for excess pitch and intensity fluctuations, as well as vocal tremors, while PSP presented a combination of hypokinetic and spastic dysarthria, characterized by a strained–strangled voice and a slow speaking rate. These findings underscore the utility of quantitative speech analysis in distinguishing between different forms of Parkinsonism.

Articulatory imprecision is another hallmark feature of Parkinson's Disease (PD), where speech becomes less distinct due to reduced precision in the movement of the articulators. Even in mild cases of PD, imprecise vowel articulation has been observed, highlighting the early impact of motor control impairments characteristic of the disease [6,7]. However, research on the specific speech characteristics that differentiate PD from Progressive Supranuclear Palsy (PSP) and Multiple System Atrophy (MSA) remains limited, with many studies focusing on timing variations in articulation. For instance, ref. [8] evaluated consonant articulation deficits, particularly in voiced and voiceless stops, across participants with PD, PSP, and MSA, as well as in healthy controls, using both acoustic and perceptual methods. Voiced and voiceless stops differ in terms of the timing of the onset of voicing (voice onset time, VOT). Imprecise consonant articulation was observed across all Parkinsonian groups. Notably, PSP and MSA exhibited prolonged VOT for voiceless plosives compared to PD, reflecting greater dysarthria severity and slower articulation. In MSA, the VOT for voiced plosives was significantly shorter, likely due to cerebellar damage. Specifically, the shortening of the negative VOT (voicing lead or negative VOT) caused the voicing to disappear, leaving only a short burst, which contributed to an increased number of voiced plosives being misclassified as voiceless during perceptual evaluation. These timing variations in articulation may offer valuable insights into the underlying disease mechanisms.

However, studies on VOT in individuals with PD have produced inconsistent results. While some studies report longer VOT durations in PD [9,10], others have found no significant changes or even shorter VOT [11–13]. These discrepancies may be due to variations in speaking rate [14]. Although the VOT ratio, a rate-independent measure, has been used to clarify these inconsistencies, it has not fully resolved the conflicting findings [10,13].

Despite promising insights, research on acoustic biomarkers for Parkinson's Disease and atypical Parkinsonian disorders often faces methodological limitations including small sample sizes which restrict the generalizability of findings, highlighting the need for larger, more diverse cohorts to validate speech deterioration patterns across PD and APD subtypes. Additionally, inconsistencies in assessment protocols introduce variability, impacting reliability and replicability. Ref. [15] underscored the importance of standardized, objective speech assessment measures in Alzheimer's Disease and Mild Cognitive Impairment, noting the impact of biases stemming from variable recording conditions and methods—a recommendation also applicable to neuromotor disorders such as Parkinsonism.

In response to these methodological needs, ref. [16] introduced the SMARTSPEECH protocol, employing a smartphone application to systematically investigate speech biomark-

ers for PD and other synucleinopathies. This approach aims to create accessible, diagnostically valuable data collection methods. Furthermore, ref. [17] provided comprehensive guidelines for recording and analyzing speech in movement disorders, establishing protocols for recording environments, vocal tasks, and acoustic features to standardize data collection. This foundational work addresses methodological inconsistencies, enhancing both the replicability and clinical relevance of speech biomarker research.

Although not fully diagnostic alone, speech-based biomarkers offer significant value in settings with limited access to advanced testing, complementing neuroimaging and genetic tools for early diagnosis and personalized treatment strategies in PD and APDs. In contrast to prior studies emphasizing articulation timing, such as VOT or other commonly investigated speech characteristics like fundamental frequency (F0), speech rate, intensity variability, rhythmic patterns, and vowel articulation, the current study examined consonant imprecision related to the ability to achieve complete closure in the vocal tract during stop consonant articulation. This analysis prioritizes articulatory precision over frequency, intensity, or timing-related factors, affecting both voiced and voiceless stops in Parkinsonian speech. The distinct lenition patterns observed between PD and APD suggest that treatment approaches may need to be tailored to address the unique articulation challenges of each group. Using Phonet, a machine learning model [18], we aimed to assess whether stop consonant weakening could reliably distinguish PD from APD. By analyzing these subtle phonological patterns, the study seeks to contribute to early diagnosis, track disease progression, and improve treatment strategies for both PD and APD.

2. Lenition

Lenition, a common phonological process, refers to the weakening or softening of consonants. This process is gradient, meaning consonants can undergo varying degrees of weakening, from subtle modifications to complete deletion. Rather than manifesting as a discrete binary shift, lenition typically unfolds progressively, where fully articulated stops transition through stages, transforming into fricatives, approximants, or disappearing altogether. The gradient nature of lenition is especially valuable for tracking phonetic variability across different contexts, as it is sensitive to phonetic environment, speech style, and speaker-specific factors. This gradience also serves as a powerful tool in clinical settings, particularly in the assessment of speech-motor control in degenerative diseases such as Parkinson's Disease (PD). By quantifying incremental changes in lenition patterns, clinicians can more precisely monitor the onset and progression of motor speech impairments and evaluate the effectiveness of interventions over time, offering insights into both linguistic and pathological shifts in speech production.

2.1. Lenition Across Languages

In Spanish, the lenition of the voiced stops /b/, /d/, and /ɡ/ exemplifies this process. When these stops occur between vowels (intervocalic position), they often weaken into approximants [β], [ð], and [ɣ] [19]. For example, sabia "I knew" is pronounced [saβía], where the /b/ lenites to [β], and cada "each" becomes [kaða], with the /d/ leniting to [ð]. In pagar, "to pay", the /ɡ/ is realized as [paɣar], showing lenition into [ɣ]. This phenomenon is most prominent in connected or informal speech but may be less pronounced in formal contexts, where stops retain more articulatory force.

While lenition in Spanish predominantly leads to the production of fricatives and approximants, it may progress further to complete deletion in certain dialects. For instance, word-final /d/ in northwestern Spain can be realized as a voiced approximant, voiceless fricative, or even be deleted altogether. A corpus study on conversational speech in the region demonstrated that /-d/ is frequently deleted or devoiced into a voiceless fricative, often neutralizing the distinction between word-final /-d/ and the phonemic /-θ/ [20]. Such patterns underscore the complexity of lenition in these dialects, where deletion and devoicing represent alternative pathways of reduction.

The principle of articulatory economy helps explain why lenition occurs. Producing stop consonants like /b/, /d/, and /ɡ/ requires significant muscular effort, as they involve full closure of the vocal tract. In contrast, fricatives and approximants, which require only partial closure, are easier and faster to produce, particularly in rapid or connected speech. Lenition thus serves as a natural adaptation in language, allowing speakers to maintain fluency without sacrificing intelligibility [19].

Lenition is not unique to Spanish; it occurs in many languages, though the degree and types of lenition vary. For example, Italian also exhibits lenition of voiced and voiceless stops in intervocalic positions, though the extent varies by dialect [21–23]. In English, lenition is evident in the process of flapping, where intervocalic /t/ and /d/ are realized as a voiced flap [ɾ], particularly in American English. For example, butter is pronounced [bʌɾɚ], where the /t/ weakens to [ɾ]. While this process does not result in fricatives or approximants, it reflects reduced articulatory effort, aligning with the principles of lenition.

2.2. Lenition in Parkinsonism

Lenition holds significant clinical relevance for speech impairments related to neurodegenerative disorders like Parkinsonism. Research on individuals with Parkinson's Disease (PD) reveals reduced lip and jaw displacement and velocity during speech, with more severe dysarthria associated with greater impairments [9,24,25]. PD also affects movement stability, with increased variability in articulatory coordination observed through measures such as the lip aperture variability index [25]. While some studies report no significant differences in movement stability between PD patients and age-matched controls [26], overall articulatory coordination declines as the disease progresses [27].

This reduced articulatory strength in PD often mimics phonological lenition, where consonants weaken or soften as motor control deteriorates. Lenition patterns may correspond to underlying neural changes. In PD, the degeneration of dopaminergic neurons in the substantia nigra impairs motor control, impacting speech production. In Atypical Parkinsonism (APD), more widespread neurodegenerative changes across the basal ganglia, cerebellum, and brainstem [2] could lead to more complex and severe speech deficits. Thus, lenition as a speech marker offers valuable insights into the progression of neurodegenerative diseases.

The study of articulatory weakening in Parkinsonism has significant implications for treatment. Speech therapy, particularly in the early stages of Parkinson's Disease (PD), focuses on improving articulatory precision and vocal strength to mitigate the effects of speech deterioration. Techniques like Lee Silverman Voice Treatment (LSVT LOUD) are designed to increase vocal intensity and articulatory effort, which in turn help reduce articulatory imprecision—a common speech issue in PD. While LSVT LOUD primarily targets vocal loudness, it indirectly strengthens articulation by encouraging more forceful and deliberate speech production, helping to mitigate the weakening of speech sounds like consonants that could otherwise become less distinct [28–30]

However, it is important to note that this does not directly address lenition as understood in phonology, which refers to a systematic weakening of sounds, such as stops becoming fricatives or approximants. Instead, LSVT LOUD reduces articulatory imprecision, which can help prevent the production of weakened consonants in everyday speech, thereby improving speech intelligibility. In more advanced stages, particularly in Atypical Parkinsonism (APD), therapy may shift toward compensatory strategies, such as augmentative and alternative communication (AAC) devices, as speech intelligibility declines due to severe dysarthria and motor control issues [31,32]. Further research into how these techniques can better target specific speech-motor deficits may lead to more personalized treatments [31].

As Parkinsonism progresses, particularly APD, therapy may shift toward compensatory strategies, including the use of augmentative and alternative communication (AAC) devices, as speech intelligibility declines due to severe dysarthria and articulatory weakening [33]. Nevertheless, early interventions like LSVT LOUD remain critical in reducing

the degree of lenition and maintaining intelligible speech for as long as possible. Further research into the neurological mechanisms underlying articulatory weakening could help optimize these interventions, offering more personalized treatment for speech decline [29]. Ongoing research into speech markers of lenition in Parkinsonism increasingly uses machine learning models and automated speech analysis tools.

In this study, we used Phonet, a machine learning model, to quantify the gradient degree of lenition. This approach enables us to capture subtle, fine-grained changes in lenition, providing early diagnostic markers for Parkinson's Disease (PD) and Atypical Parkinsonism (APD), while allowing clinicians to track disease progression and tailor treatment accordingly.

Phonet has been demonstrated to have high accuracy in detecting phonemes and phonological classes in Spanish [18] and the lenition patterns of Spanish stops [34–36] and has been effective in modeling speech impairments in patients with Parkinson's Disease [37], as well as in analyzing speech characteristics in contexts such as intoxicated speech [38,39] and L2 Spanish [40,41].

2.3. Phonological Features and Lenition

Phonological features are the distinctive properties that define how sounds function within a language's sound system. For the study of lenition, two key features—[continuant] and [sonorant]—are particularly important. These features help distinguish sounds based on the degree of airflow and resonance during articulation, both of which are crucial in understanding how consonants weaken in the lenition process.

The [sonorant] feature refers to sounds that allow relatively free airflow and resonance through the vocal tract, such as nasals, liquids, glides, and vowels. These [+sonorant] sounds tend to be voiced and more resonant, contrasting with obstruents like stops and fricatives, which significantly obstruct airflow. The [continuant] feature distinguishes sounds that allow sustained airflow from those that do not. These [+continuant] sounds, including fricatives, liquids, glides, and vowels, permit ongoing airflow through the vocal tract, while stops, which are [-continuant], involve a complete blockage of airflow.

In lenition, stops, such as /b, d, ɡ/, which are [-continuant], often weaken to fricative-like or approximant-like realizations. A fricative-like realization would have a high [continuant] probability but a low [sonorant] probability, indicating sustained airflow but limited resonance. In contrast, an approximant-like realization would exhibit both a high [continuant] probability and a high [sonorant] probability, allowing for both continuous airflow and greater resonance.

This distinction between fricative-like and approximant-like outcomes is crucial for understanding the gradient nature of lenition. In languages like Spanish, lenition involves a shift in voiced stops toward more fricative-like or approximant-like forms, with stops becoming [+continuant] and/or [+sonorant] in certain contexts. Capturing the interaction between these two features is essential for analyzing how sounds change and weaken over time in the lenition process.

3. Phonet

Phonet, developed by Ref. [18], is a machine learning model that estimates the posterior probabilities of phonological features using bi-directional recurrent neural networks (RNNs) with gated recurrent units (GRUs). The model processes acoustic features such as log-energy distributed across triangular Mel filters from short windows (25 ms) of the input signal with predictions computed over 10 ms frames, ensuring fine-grained temporal resolution. When multiple frames occur within a phoneme token, the average prediction from the middle frame(s) is used for classification. These features are then analyzed using GRU layers, which capture both past and future context in the speech signal. Phonet ultimately classifies phonological features using a softmax-activated output layer.

In this study, Phonet was trained on 23 Spanish phonological classes and 26 phonemes using a weighted categorical cross-entropy loss function to address class imbalance. Al-

though the PD and AD data are in English, we opted to use the Spanish-trained model due to its proven cross-linguistic effectiveness. As shown by Ref. [42], models trained on Spanish speech data slightly outperform those trained on English data in classifying PD vs. healthy controls.

Model training was performed using an NVIDIA GeForce RTX 3090 GPU, and the corpus was split into a training subset (80%) and a test subset (20%) using the Python scikit-learn library [43]. Ambiguous targets such as /b, d, g/ were excluded to avoid contamination. The model achieved an unweighted average recall (UAR) ranging from 94% to 98% for phonological class detection, with UARs of 97% and 96% for the sonorant and continuant features, respectively. Phoneme detection accuracy ranged from 42% for speech-like noise /spn/ to 96% for /f/, with most phonemes falling between 59% and 96%. Please see [34] for model training procedures.

4. Methods

4.1. Data

The data consisted of sentence readings by 142 participants, 108 of whom had been diagnosed with Parkinson's Disease (typical PD) and 34 of whom had been diagnosed with other forms of Parkinsonism (atypical PD). Diagnoses were based on comprehensive neurological evaluations combined with assessments of dysarthria types, conducted as part of routine care. Patients with PD all exhibited hypokinetic dysarthria, while those with Atypical Parkinsonism presented with various dysarthria types, including hypokinetic-hyperkinetic dysarthria, spastic-ataxic dysarthria, ataxic dysarthria, and mixed forms such as hypokinetic-flaccid dysarthria. The specific diagnoses are summarized in Table 1.

Table 1. PD and APD diagnoses.

Diagnosis	Number of Participants
Parkinson's Disease (typical PD)	108
Multiple System Atrophy	11 Autonomic-1 Cerebellar ataxia-3 Parkinsonism-6 Unspecified-1
Progressive Supranuclear Palsy	10
Parkinsonism	4
Lewy Body Dementia	4 2 LBD 1 Dementia with Lewy bodies 1 PD with LBD
Corticobasal Degeneration/Corticobasal Syndrome	3 2 CBD 1 CBS
ALS-PD	1
Cervical dystonia with associated hand tremor	1

The participants read the following four sentences:

1. The valuable watch was missing.
2. Please put the groceries in the refrigerator.
3. The shipwreck washed up on the shore.
4. In the summer they sell vegetables.

The target sounds were the stops across all four sentences. Because of the limited variety of sentences, only five of the six English stops (/b/, /g/, /p/, /t/, and /k/) were attested (Table 2).

Table 2. Tokens across groups and phones.

	Typical	Atypical
/b/	216	58
/ɡ/	108	29
/p/	432	116
/t/	315	81
/k/	108	29

4.2. Procedure

The PD and APD data were forced aligned using the Montreal Forced Aligner (version: 2.0) [44]. The trained Phonet model described above was applied only to the stop tokens /b, d, g, p, t, k/, with other phones silenced out. The predicted posterior probabilities for the [continuant] and [sonorant features] were computed over 10 ms frames. If a phone token contained multiple frames, then the average of the prediction of the middle frame(s) was used as the prediction of that phone.

4.3. Analysis

Data were analyzed using linear mixed-effects models [45] in R [46]. Continuant and sonorant posterior probabilities, respectively, were the dependent variables in each model. Predictor variables were group (APD vs. PD), voicing (voiced vs. voiceless), and place (bilabial vs. alveolar vs. velar), and speaker was a random effect. The group and voicing variables were deviation-coded, and the place of articulation variable was forward difference-coded. The contrasts examined were bilabial vs. alveolar and alveolar vs. velar, as well as atypical vs. typical, voiced vs. voiceless, and interactions with the group. The model formula is given below:

$$\text{Lmer(posterior probability} \sim \text{group + voice + place + group:voice + group:place + (1 | speaker)}$$

To capture the shape of the posterior probability distribution beyond the conventional difference in mean, additional analyses were performed using the four statistical moments calculated over the deviation value of each token. The deviation is the difference between the "expected" value for each feature and the posterior probability calculated by Phonet. The expected value is 0 and all deviations are negative if the phone has a negative value for the feature (e.g., /p/ is [-sonorant]). The expected value is 1 and all deviations are positive if the phone has a positive value for the feature (e.g., /j/ is [+sonorant]). In this experiment, since the target stops are [-continuant] and [-sonorant], the deviations are always negative.

The four statistical moments (mean, variance, skewness, and kurtosis) were calculated for each speaker using the deviation values of both continuant and sonorant features. Analyzing these moments allows us to capture unique distributional characteristics in articulation that may help distinguish PD from APD. The mean reflects central tendency, offering insight into the typical level of articulatory deviation in each group. Variance indicates the spread, shedding light on consistency or variability in articulatory patterns, which may differ due to the more pronounced motor control irregularities often seen in APD. Skewness assesses asymmetry, potentially identifying directional biases linked to motor deficits. Lastly, kurtosis highlights 'tailedness', identifying extreme deviations that may occur more frequently in APD. Together, these moments allow for a comprehensive comparison across groups, capturing subtle distributional differences that can illuminate distinctions in motor speech characteristics between PD and APD.

A logistic regression was run for each feature, with typical vs. atypical group as the dependent variable, and the z-scores of the statistical moments for each speaker as the predictors. In this analysis, the group variable was treatment-coded, with PD as the null level, and APD as the treatment level. The logistic regression formula is given below:

glm(group~feature.mean.deviation.z + feature.variance.deviation.z + feature.kurtosis.deviation.z + feature.skewness.deviation.z)

5. Results

5.1. Continuant Posterior Probability

As shown in Figure 1, PD and APD patients did not differ in continuant posterior probability (b = −0.010, SE = 0.013, t = −0.770, $p = 0.442$)

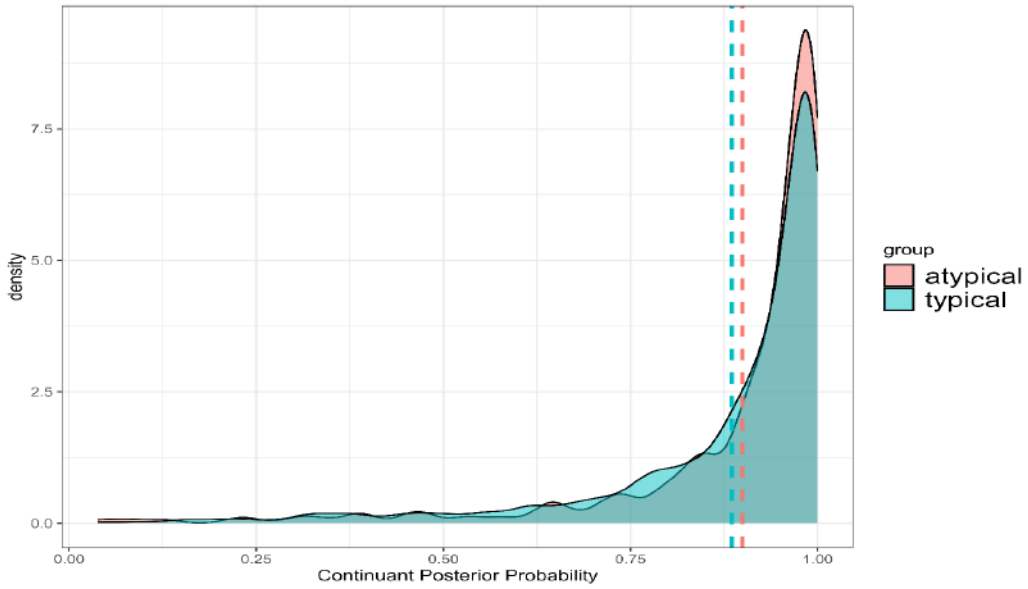

Figure 1. Continuant posterior probability for typical (PD) and atypical (APD) groups: higher probabilities indicate greater weakening (more fricative-like articulation) of stop consonants across different groups and places of articulation Dash lines indicate mean values for typical (teal) and atypical (pink) groups.

Alveolar stops had a lower posterior probability than velar stops (b = −0.072, SE = 0.017, t = −4.320, $p < 0.001$) (Figure 2). Post hoc analyses, calculated using estimated marginal means [47] showed that this place difference only applies to typical PD patients (b = −0.074, SE = 0.015, t = −4.911, $p < 0.001$), but not atypical PD patients (b = −0.069, SE = 0.029, t = −2.332, $p = 0.182$).

Voiced stops had a significantly higher posterior probability than voiceless stops (b = −0.037, SE = 0.013, t = −3.010, $p = 0.002$). Post hoc analyses revealed that voiced stops had significantly higher posterior probabilities than voiceless stops for PD patients (b = 0.049, SE = 0.011, t = 4.363, $p < 0.001$), but not for APD patients (b = 0.002, SE = 0.021, t = 1.130, $p = 0.671$) (Figure 3). There were no significant interactions (group × voice: b = −0.025, SE = 0.025, t = −1.003, $p = 0.316$; group × place, bilabial vs. alveolar: b = 0.012, SE = 0.0331, t = 0.459, $p = 0.647$; group × place, alveolar vs. velar: b = −0.006, SE = 0.033, t = −0.171, $p = 0.864$).

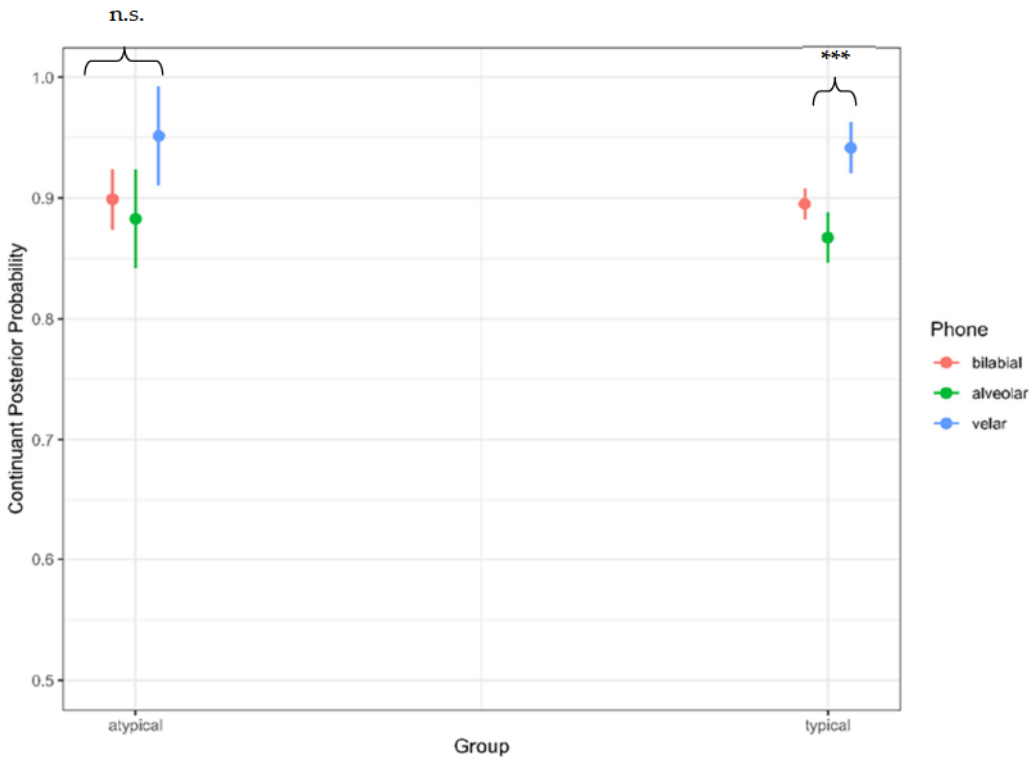

Figure 2. Continuant posterior probability by group and place of articulation: higher probabilities indicate greater weakening (more fricative-like articulation) of stop consonants across different groups and places of articulation. "n.s." denotes no significant difference; "***" indicates a significant difference at $p < 0.0001$.

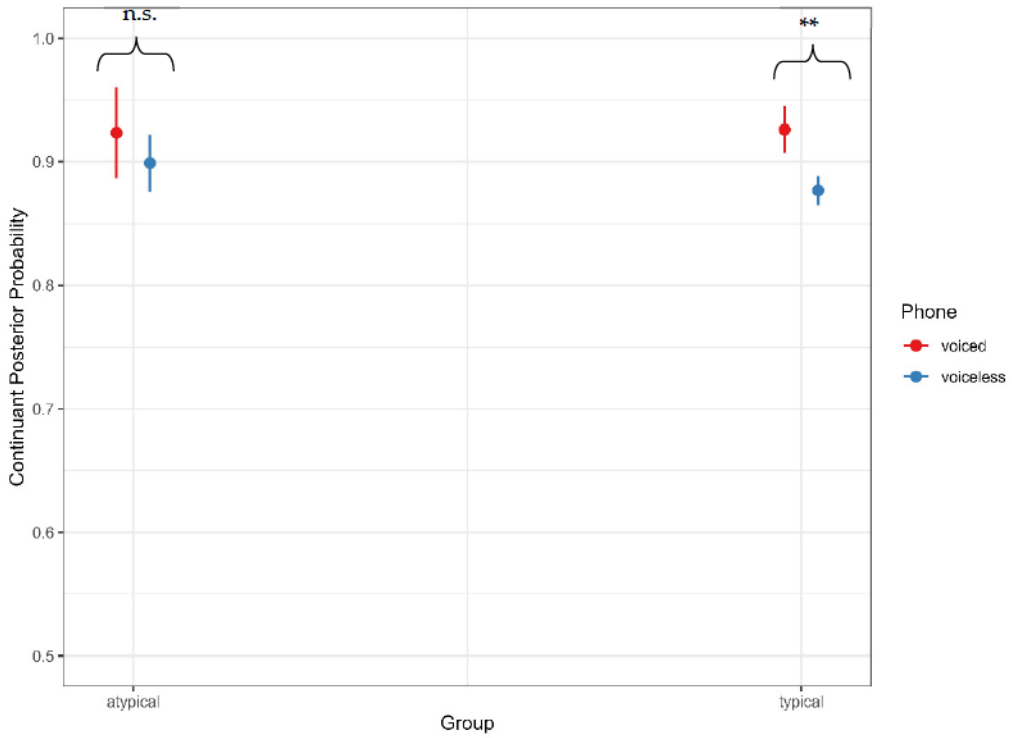

Figure 3. Continuant posterior probability by group and voicing: higher probabilities indicate more fricative-like production of stop consonants. "n.s." denotes no significant difference; "**" indicates a significant difference at $p < 0.001$.

In the deviation analysis, the distribution of PD deviations had a significantly higher mean (b = −1.367, SE = 0.202, z = −6.783, $p < 0.001$) than the APD distribution. PD patients also had a smaller standard deviation (b = 1.227, SE = 0.208, z = 5.907, $p < 0.001$) and a smaller skewness (b = −0.515, SE = 0.240, z = −2.146, $p = 0.032$) than APD patients. There was no difference in the kurtosis of deviations between groups (b = 0.142, SE = 0.226, z = 0.631, $p = 0.528$). A larger mean deviation score has less distance from the expected value of 0, because all deviations are negative here. Therefore, the larger deviation value implies less lenition in the PD group than in the APD group. The smaller skewness score for PD patients means that the distribution is less right-tailed than for the APD group, which follows from the lower mean score. The lower standard deviation for PD patients suggests less variability.

Figure 4 shows the continuant deviation statistical moments. Mean, in the top left, has a maximum value of 0 (no deviation from the expected posterior probability) and a minimum value of −1 (expected posterior probability of 0, observed posterior probability of 1). Here, the deviations are clustered on the lower end of the scale, demonstrating a high continuant posterior probability and a lenited production in comparison to the expected stop-like production. Variance and kurtosis have a minimum value of 0 and no maximum value, and skewness is unbounded.

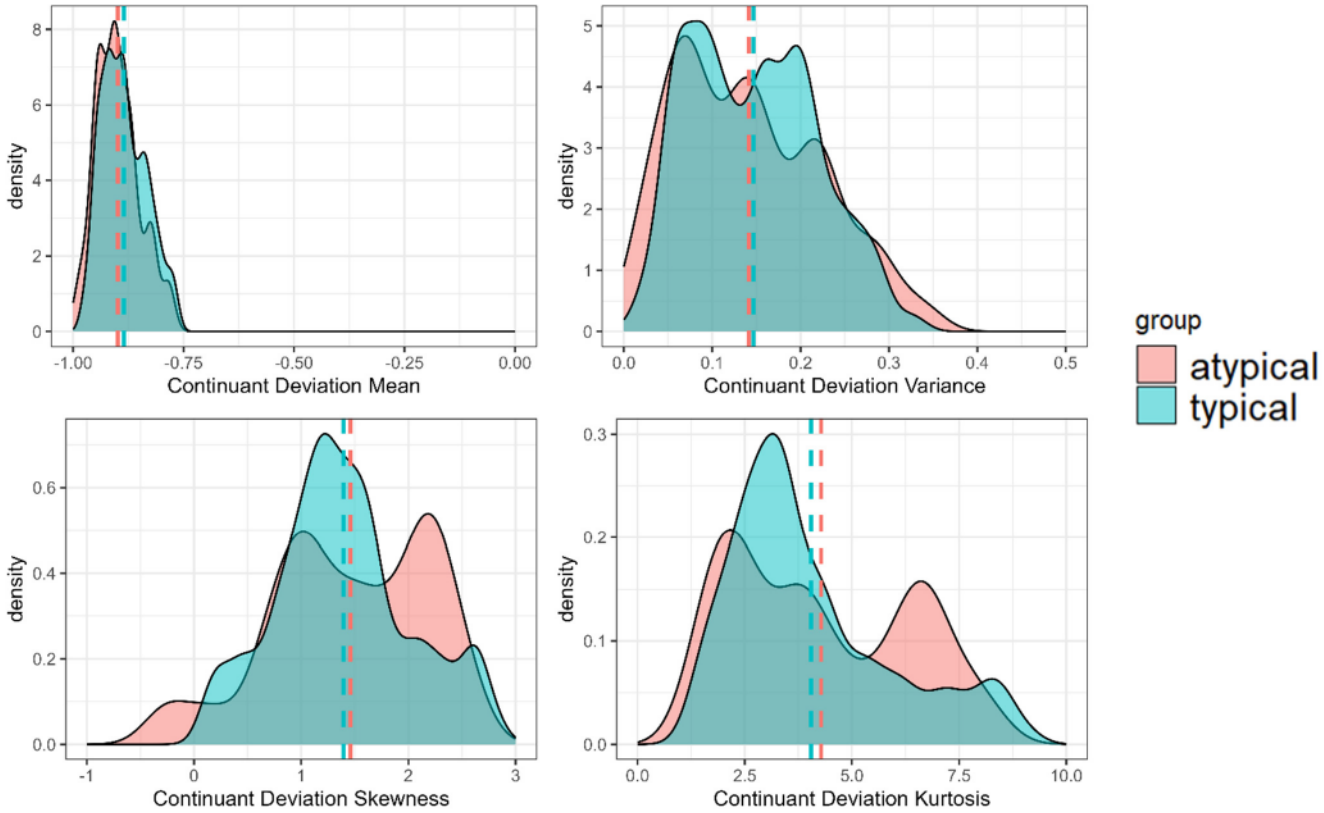

Figure 4. Density plots of continuant deviation mean, variance, skewness, and kurtosis for typical (teal) and atypical (pink) Parkinsonian Disorder groups. Dashed lines indicate group means.

5.2. Sonorant Posterior Probability

PD and APD patients did not differ in sonorant posterior probability (b = −0.014, SE = 0.028, t = 0.524, $p = 0.601$) (Figure 5).

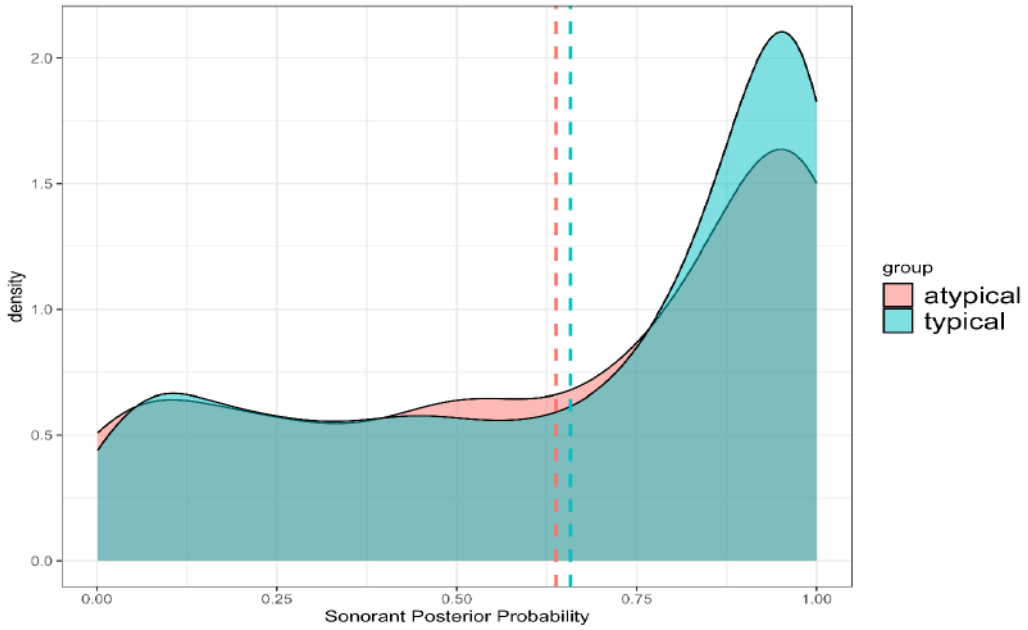

Figure 5. Sonorant posterior probability for typical (PD) and atypical (APD) groups: Higher probabilities indicate greater sonorant (more approximant-like) articulation across groups. Dashed lines represent the mean probability for each group.

Voiced stops had a higher sonorant posterior probability than voiceless stops (b = −0.222, SE = 0.024, t = −9.278, $p < 0.001$) (Figure 6). This difference was significant for both PD (b = 0.224, SE = 0.022, t = 10.223, $p < 0.001$) and APD patients (b = 0.219, SE = 0.043, t = 5.160, $p < 0.001$). There was no difference in posterior probability between places of articulation (bilabial vs. alveolar: b = −0.026, SE = −0.025, t = −1.056, $p = 0.291$, alveolar vs. velar: b = 0.052, SE = 0.032, t = −1.603, $p = 0.109$) (Figure 7) and no significant interactions (group × voice: b = −0.005, SE = 0.048, t = −0.109, $p = 0.913$; group × place, bilabial vs. alveolar: b = 0.012, SE = 0.0331, t = 0.459, $p = 0.647$; group × place, alveolar vs. velar: b = −0.006, SE = 0.033, t = −0.171, $p = 0.864$)

In the deviation analysis, PD patients had a significantly smaller skewness deviation score than APD patients (b = −0.625, SE = 0.238, z = −2.625, $p = 0.009$). As in the continuant model, the PD distribution is less right-tailed (Figure 8). The other three statistical moments did not differ between groups (mean: b = −0.310, SE = 0.177, z = −1.754, $p = 0.079$; variance: b = 0.048, SE = 0.102, z = 0.472, $p = 0.637$; kurtosis: b = 0.107, SE = 0.176, z = 0.611, $p = 0.542$).

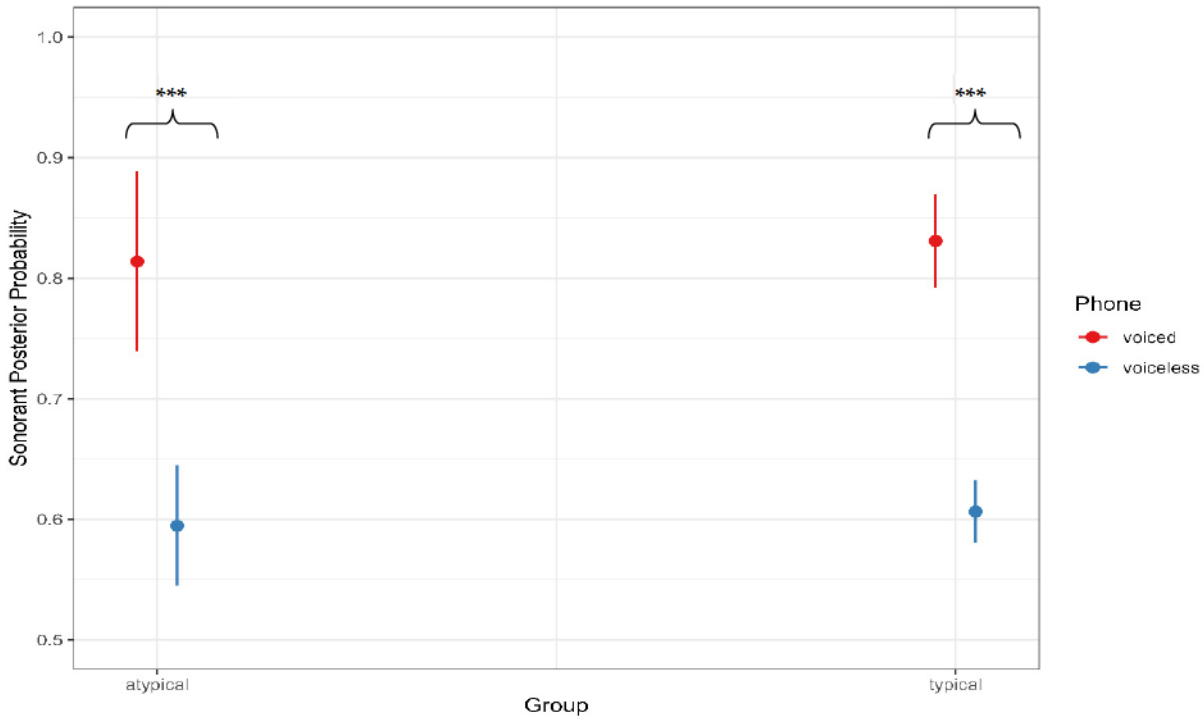

Figure 6. Sonorant posterior probability by group and voicing: Higher probabilities indicate more approximant-like articulation across typical and atypical groups by voicing. *** denotes significance at $p < 0.0001$.

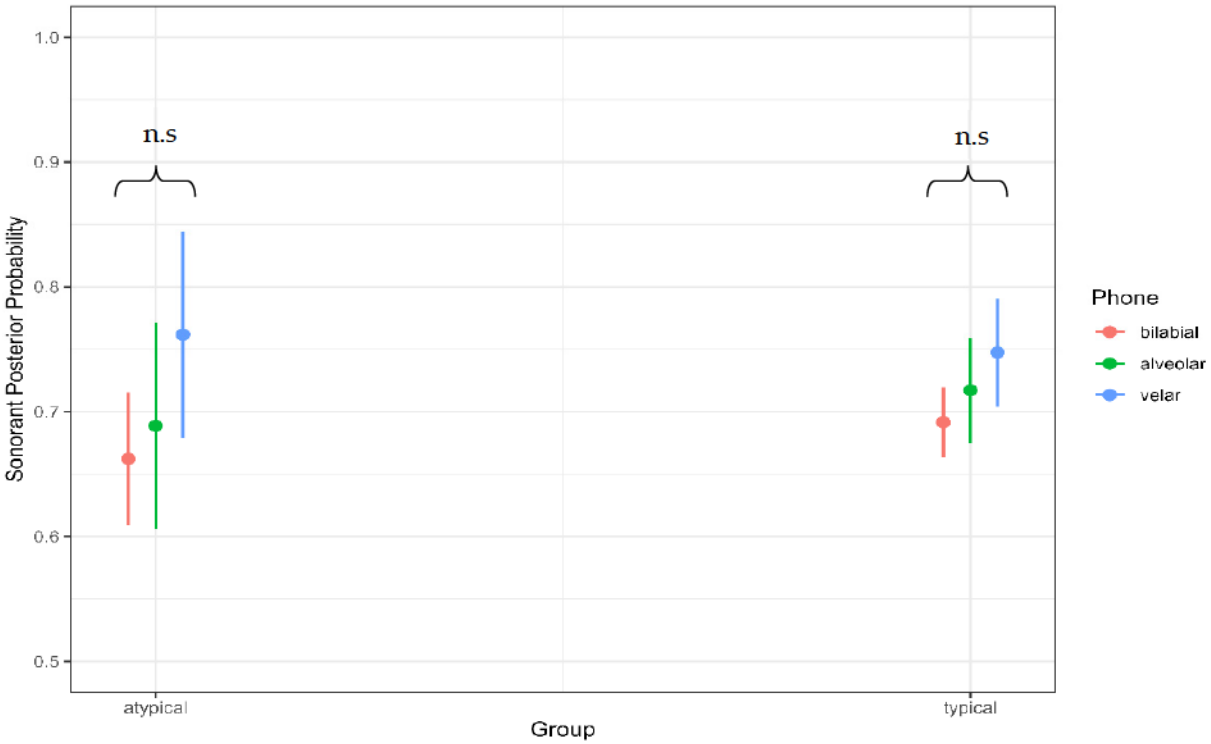

Figure 7. Sonorant posterior probability by group and place of articulation: Higher probabilities indicate more approximant-like articulation across typical and atypical groups for different places of articulation. n.s. denotes non-significance.

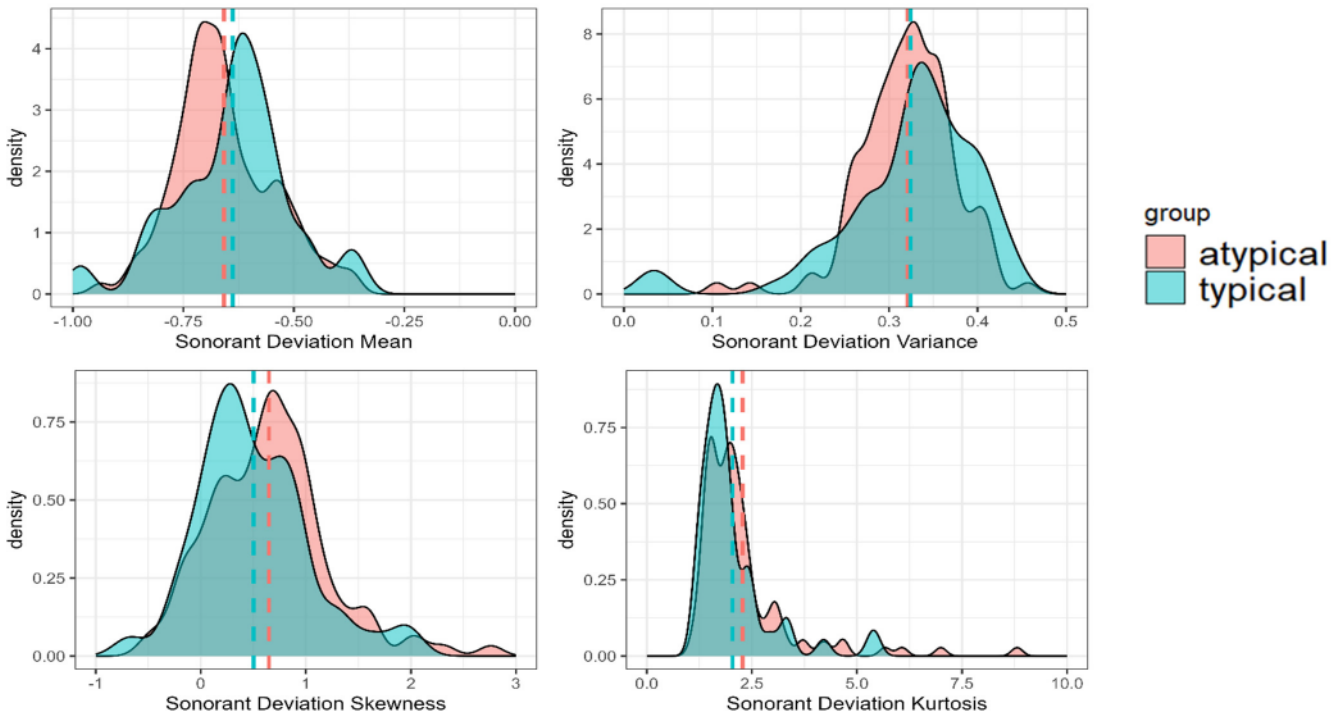

Figure 8. Sonorant deviation moments (mean, variance, skewness, and kurtosis) by group: Density plots display the distribution of sonorant deviation across typical and atypical groups. Dashed lines indicate mean values for each group, illustrating differences in articulatory variability and distribution patterns.

6. Discussion and Summary

The findings from this study highlight the potential of lenition, quantified through posterior probabilities of phonological features, as a diagnostic marker for distinguishing Parkinson's Disease (PD) from Atypical Parkinsonism (APD). Although no significant differences were observed in the overall posterior probabilities of continuant and sonorant features, critical distinctions emerged through deviation analysis. Specifically, PD patients exhibited less variability and more stable articulatory patterns, while APD patients demonstrated greater variability, indicating less precise motor control. These findings suggest that lenition is more systematic and controlled in PD, whereas APD is characterized by more erratic and unpredictable articulatory patterns.

The lack of significant overall group differences could result from several factors. Variation in disease severity among participants may play a role, as more advanced stages of APD typically involve greater motor control deficits. Dopaminergic treatment effects, particularly in the PD group, could also mask articulatory differences by mitigating symptom severity. Additionally, variability in individual characteristics, such as age and general health, may reduce statistical power, highlighting the importance of future studies to clarify articulatory patterns through these variables.

Moreover, the broader neurodegenerative impact of APD likely contributes to the increased variability in articulation, affecting motor control more severely and broadly than in PD. While PD primarily affects the substantia nigra and its dopaminergic pathways, APD involves more widespread neural disruptions, including in the cerebellum, basal ganglia, and brainstem [2]. These wider disruptions align with observed lenition patterns in APD, with motor control deficits in this population being more generalized than those typically seen in PD.

A key finding in this study was the interaction between group and voicing. In PD patients, voiced stops exhibited higher continuant and sonorant posterior probabilities than voiceless stops. This suggests that, although PD patients struggle to form complete

oral closures for both voiced and voiceless stops, they maintain finer control over the oral aperture, preserving the contrast between voiced and voiceless stops. In contrast, APD patients showed the distinction between voiced and voiceless stops in sonorant posterior probabilities but failed to maintain this distinction in continuant probabilities, indicating a breakdown in their ability to control smaller oral aperture variations, though they could manage larger oral openings. These findings align with [8], which reported a similar breakdown in voicing distinctions in APD. Specifically, [8] found that APD patients were unable to produce prevoicing (lead voicing or negative VOT), which caused voiced stops to be perceived as voiceless stops. This inability is likely due to increased supraglottal pressure during oral closure, which makes it more difficult to initiate and sustain vocal fold vibration. This increased pressure counteracts the subglottal pressure needed for voicing, exacerbating motor execution deficits and complex vocal fold movements. The more extensive neural degeneration in APD likely contributes to this reduced capacity for maintaining contrastive features. Regarding place of articulation, PD patients demonstrated less lenition in alveolar stops compared to velar stops, consistent with aerodynamic principles [48,49], which predict greater lenition for sounds articulated further back in the vocal tract. APD patients, however, showed no such distinction, suggesting a generalized motor deficit that affects stops across all places of articulation equally, reinforcing the hypothesis of widespread neural damage in APD.

The higher variability in speech production observed in APD, particularly in maintaining phonological contrasts, suggests a more significant disruption of the fine motor control necessary for articulation. By contrast, PD patients exhibited more stable articulatory patterns, consistent with the more localized neurodegenerative impact of the disease. This pattern is consistent with the findings of [5], who noted that dysarthria in Progressive Supranuclear Palsy (PSP) and Multiple System Atrophy (MSA) differed from that of PD due to its greater severity and the presence of spastic and ataxic components. Specifically, PSP was marked by increased dysfluency, slower speaking rates, inappropriate silences, and vowel articulation deficits, while MSA was characterized by vocal tremors, pitch fluctuations, and prolonged phonemes. The broader motor impairments seen in APD are reflected in the more frequent and severe lenition observed in this study, consistent with the findings of [4].

These findings highlight crucial differences in how PD and APD patients manage articulation, especially in sustaining distinctions between voiced and voiceless sounds. PD patients, while experiencing some articulatory limitations, retain a degree of control over the oral aperture and are able to achieve the transglottal pressure necessary for voicing. This suggests a relatively preserved coordination of laryngeal and respiratory functions, supporting the maintenance of voicing contrasts. Conversely, the extensive neural degeneration in APD, particularly affecting the cerebellum and basal ganglia, disrupts finer control over both oral and laryngeal articulation, leading to greater challenges in sustaining voicing distinctions and contributing to reduced intelligibility in APD patients.

The findings also suggest the need for differential therapeutic interventions for PD and APD to address these specific articulatory challenges. For PD, exercises that strengthen respiratory and phonatory functions—such as those in the Lee Silverman Voice Treatment (LSVT LOUD)—are highly beneficial. These exercises focus on enhancing respiratory support and improving vocal fold adduction, both crucial for maintaining voiced-voiceless distinctions. LSVT LOUD, in particular, has shown efficacy in increasing vocal loudness and improving speech clarity, which are key to sustaining voicing contrasts in PD [28]. Additionally, respiratory support exercises that focus on breath control and coordination with phonation can mitigate supraglottal pressure issues, potentially promoting more stable voicing onset and maintenance [50].

Targeted tongue exercises, as described by [51], designed to strengthen the suprahyoid muscles and improve swallowing, may also enhance muscle control and support oral closure. This improvement can help PD patients produce clearer stop consonants and maintain voiced–voiceless distinctions, ultimately boosting articulatory precision.

For APD patients, techniques like exaggerated articulation—employed in the Be Clear program [52]—and pacing strategies can improve overall speech intelligibility by stabilizing timing and reducing the slurred speech common in ataxic dysarthria [53]. The Be Clear program, an intensive treatment for non-progressive dysarthria, incorporates these techniques to improve intelligibility in individuals with similar impairments following traumatic brain injury (TBI). Through the repeated practice of exaggerated articulation, it has shown promising results for speech clarity, supporting gains in intelligibility, vowel space, and articulatory precision.

Beyond speech-specific interventions, non-pharmacological management strategies can enhance functional communication and quality of life for APD patients. These include physical therapy and balance training for motor symptoms, occupational therapy for fine motor skills essential to daily tasks, and cognitive therapy for addressing potential cognitive deficits. Together, these approaches offer comprehensive support for APD patients, enhancing both voicing control and functional communication [54]. Taken together, these results suggest that lenition, particularly in articulatory variability and the maintenance of voicing contrasts, could serve as a valuable diagnostic marker for distinguishing PD from APD. The more severe and unpredictable lenition in APD, along with the breakdown in the ability to differentiate voiced and voiceless stops, reflects the broader neurodegenerative changes that affect motor control in this population. Future research should explore additional phonological features, such as nasality and frication, to further refine the differential diagnosis of Parkinsonian Syndromes.

7. Conclusions

This study demonstrates the potential of lenition as a diagnostic marker for differentiating Parkinson's Disease (PD) from Atypical Parkinsonism (APD). Through the use of Phonet, a machine learning model, we captured subtle distinctions in the speech production patterns of PD and APD patients by analyzing the posterior probabilities of continuant and sonorant features. While the overall posterior probabilities did not significantly differ between the groups, deviation analysis revealed critical differences in speech variability and articulatory precision.

PD patients exhibited more consistent and stable lenition patterns, especially in maintaining voiced stop articulations, while APD patients showed greater variability and more pronounced lenition. These findings suggest that lenition patterns, particularly when analyzed alongside articulatory variability, could serve as valuable diagnostic markers for distinguishing between these forms of Parkinsonism.

Overall, this study reinforces the growing body of evidence supporting speech analysis as a non-invasive, objective tool for diagnosing and monitoring neurodegenerative diseases. By quantifying speech features such as lenition, clinicians may be better equipped to detect early signs of APD, track disease progression, and develop more personalized treatment interventions.

8. Limitations and Future Research

While this study provides valuable insights into the potential of using lenition as a diagnostic marker for Parkinson's Disease (PD) and Atypical Parkinsonism (APD), several limitations should be considered. First, the relatively small and imbalanced sample size—particularly the smaller number of APD participants compared to PD participants—may limit the generalizability of the results. A larger and more balanced dataset, including a broader range of APD subtypes, is needed to confirm these findings and strengthen the validity of the diagnostic patterns observed.

Second, the variability in disease stage and severity among participants is a significant factor to consider. While our analysis provides valuable insights into articulatory patterns in both PD and APD, the clinical utility of our method would be most beneficial for early-stage diagnosis, where distinguishing between typical and atypical Parkinsonism is often most challenging. However, our study includes participants across a range of disease stages,

which may affect the generalizability of our findings if severity levels differ significantly between groups. For example, comparing more advanced stages of atypical Parkinsonism to early stages of typical Parkinsonism could impact the observed differences in articulatory patterns.

Third, the analysis relied on a logistic regression model, which assumes a linear relationship between the predictors and the log odds of group classification. This approach may oversimplify the complex relationships inherent in articulatory deviations between PD and APD, potentially overlooking non-linear patterns or interactions. Although non-linear or machine learning methods could capture these complexities more effectively, we chose logistic regression for its interpretability and to provide clear, coefficient-based insights in this exploratory study. Future studies could investigate non-linear models to determine if they enhance classification accuracy in distinguishing PD from APD.

Fourth, the study focused exclusively on a limited set of phonological features—continuant and sonorant probabilities—within a narrow set of phonemes. While these features are crucial for analyzing lenition, the speech of individuals with Parkinsonism is affected by a wide array of articulatory and phonological factors. Future research should explore additional phonological features, such as nasality, stridency (frication), and phonation (e.g., breathiness and hoarseness), to provide a more comprehensive understanding of how speech changes in PD and APD.

Moreover, the sentences used in the data collection process were limited in variety and complexity. A greater range of linguistic contexts, including spontaneous speech and conversational data, could reveal more about how lenition manifests in natural communication. Since lenition is often gradient and context-dependent, evaluating speech across different types of discourse (e.g., formal vs. informal) and in more natural settings would offer richer insights into the full extent of speech impairments in Parkinsonism. Another limitation concerns the use of the Phonet model, which, while effective in this study, may be sensitive to variability in language and dialect. The model was trained on Spanish phonological features, which may not fully align with the phonetic and phonological characteristics of English, the language used in this study. Future studies should explore how models trained on different languages perform in similar diagnostic tasks and whether language-specific models provide better diagnostic accuracy for speakers of various languages [30].

In terms of future research directions, additional studies could investigate whether the patterns of lenition observed in this study are consistent across different stages of PD and APD. By tracking speech changes longitudinally, researchers could assess whether lenition patterns become more pronounced as these diseases progress. This could contribute to the development of speech-based biomarkers not only for diagnosis but also for monitoring disease progression.

Furthermore, future work could integrate speech analysis with other diagnostic tools, such as neuroimaging or physiological assessments, to develop more comprehensive models of disease detection and progression. Multimodal approaches may reveal correlations between speech impairments and neural degeneration, thereby providing deeper insights into the mechanisms behind speech deficits in neurodegenerative disorders.

Additionally, a focused evaluation of early-stage PD and APD patients, paired with the longitudinal tracking of lenition patterns and integration with neuroimaging and genetic testing, holds considerable potential to advance early differential diagnosis and reveal progression-specific changes in articulatory control. Such comprehensive longitudinal data could establish lenition as a reliable biomarker for tracking disease progression, directly informing and refining treatment strategies to be more responsive to the evolving needs of patients.

Lastly, exploring the effects of therapeutic interventions, such as speech therapy or pharmacological treatments, on lenition patterns in PD and APD patients would be valuable. Understanding how treatment impacts articulatory precision and lenition could lead to improved therapeutic strategies that better address the specific motor speech deficits experienced by individuals with these conditions.

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Institutional Review Board Statement: The data were obtained from the database of a different study which was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board (or Ethics Committee) of the University of Florida for studies involving humans. We were approved by the UFIRB to access and use the data for this study (UF IRB201602473).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study from which the data were obtained.

Data Availability Statement: Data are available upon request to the authors. The data are not publicly available due to the sensitive information contained therein.

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