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Studying Vibratory Patterns of Vocal Folds and Their Impairments in Parkinson's Disease: A Theoretical Approach

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Abstract: Current vocal fold models emphasize either time or space during voice production but leave the source unconsidered. In the present review paper, we studied the vocal fold oscillations by dividing them into two zones. Zone 1 contains the brain, neurons, and vagus nerve, and zone 2 comprises mechanical apparatus including lungs, voice box, and articulators. Then, a zonal view of changes in the voice, and vocal impairments due to Parkinson's disorder is demonstrated. The paper further examines various mathematical models analyzing vocal fold oscillations and their respective limitations. Since voice production is a cause-and-effect relationship, thus we used double point Green's Function method to solve the problem of vocal fold oscillation. This adds the causality factor and delay term to the problem and also illustrates the potential problems that could arise during the creation, conversion, or transition of impulses during speech production. Hence, our approach addresses the problem at the time when sound waves are in the form of electromagnetic impulses in the brain. Due to a lack of test subjects, however, we are unable to evaluate the experimental outcomes of this approach. Thus, currently, our work is a theoretical concept only. Consequently, the validity of the suggested Green's Function treatment in detecting the point source conveying a change in the vibratory pattern of vocal folds can be experimentally verified in future studies with the availability of potential subjects. Moreover, this suggested solution can also be applied to assess other disorders associated with vibratory patterns of vocal folds. Finally, the paper also mentions some unexplored limitations, which can be elucidated in the future.

Keywords: Green's Function, Vocal Fold Vibrations, Acoustic models, Parkinson's Disease

1. Introduction

Among many means of communication, the most frequently used method is verbal communication. It involves a process of conversion of sentiments, feelings, ideas, and emotions into voice. The backbone of verbal communication is the production of sound. It begins from neurons followed by the process of respiration, passing through vocal folds, and other articulators including the mouth, which destined the intelligible output sound wave as either a letter, word, sentence, or speech. Therefore, creating a complex structure and relationship of various mechanisms working together in a fluent and well-coordinated manner. Additionally, the voice is not a mere sound wave. It varies from individual to individual and heavily depends on gender and age. The quality of voice can disclose the personality of a person, like behavior, mood, emotions, and even about their profession, such as singer, teacher, vendor, etc.

The main apparatus responsible for the production of voice is the larynx (or voice box) as portrayed in Figure 1 [1]. The middle of the neck close to Adam's apple (ranging from the 4th to 6th cervical vertebrae) is the larynx [2]. It consists of the epiglottis, true vocal folds, thyroid cartilage (or Adam's apple), and vestibular folds [3]. The pair of muscular flaps protected by mucosa in the middle of the larynx represent the actual vocal folds. The same is also portrayed in Figure 2 [4]. When breathed air leaves the lungs, it travels via the trachea and vibrates these vocal

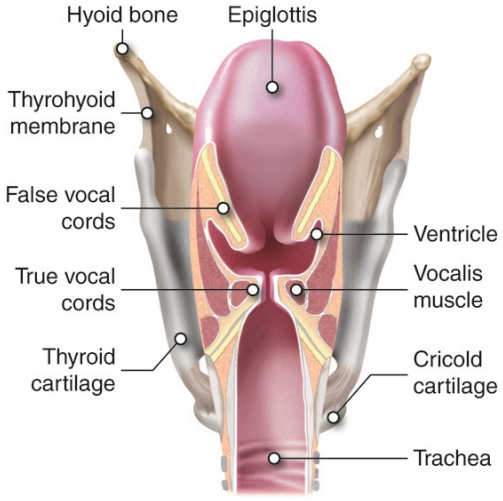

Figure 1. Anatomy of the larynx [1]

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folds. These opening and closing of vocal folds (or vibrations) generate sound waves. These waves move upwards through the epiglottis to the throat (or pharynx) and gets modulated via articulators, such as the teeth, tongue, lips, and oral and nasal cavities.

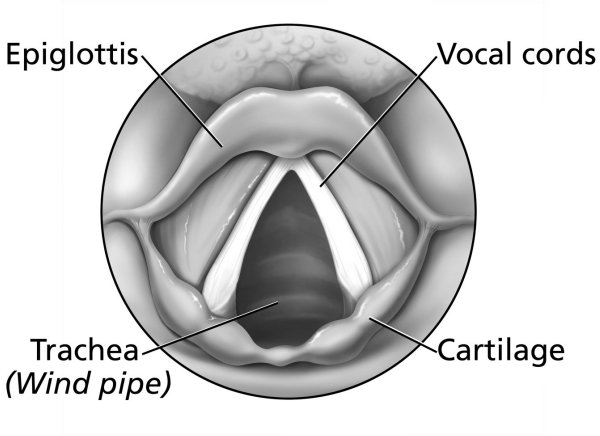

Figure 2. Top view of the larynx [4]

Additionally, the quality of the voice being produced is majorly affected by gender, age, length, density, and thickness of vocal folds, Adam's apple, and disorders. For instance, vocal folds in the adult males are relatively large and thick. They are between 0.75 to 1.0 inches in length. Similarly, in females these vocal folds are less dense and are between 0.5 to 0.75 inches in length [5]. This causes low pitch in males and high pitch in females. It draws attention to the fact that the vocal cord's length and thickness control the pitch of the voice. It suggests that longer and thicker vocal cords result in a higher-pitched, deeper voice.

Besides aging and shrinkage of larynx muscles, Parkinson's Disease causes the muscles in the larynx to go weak. Some disorders, namely Laryngitis, Vocal cord nodules, polyps, and cysts (either benign or cancerous-malignant), Vocal cord paralysis, Laryngeal cancer, Spasmodic dysphonia, vocal cord dysfunction, Alexander disease, Reinke's edema, etc., impact the quality of voice by bringing hoarseness [6], [7], [8]. Other commonly observed changes in vocalization involves swallowing troubles, difficulty in breathing, and recurring coughs [9]. Also, the aforesaid disorders can also affect the ability to speak leading to permanent or complete voice loss.

Moreover, the role of the nervous system cannot be overlooked while considering the voice changes arising due to any damage or injury to the recurrent nerve [10], [11]. The human nervous system carries bidirectional (motor and non-motor) instructions, from the brain to different bodily parts and vice-versa. These signals from the nerves activate the muscles and control their movement. Generally, the Central and Peripheral Nervous Systems are the two major sections of a nervous system. It is further divided into two parts, where activation, control, and coordination required for voice production are handled by the latter section, i.e., the Peripheral Nervous System. There are 12 cranial nerves in this system, among which the longest is the 10th Cranial Nerve called the Vagus Nerve, commencing from the brain and covering the larynx down to the stomach. This Vagus nerve further splits into two branches, namely, the superior and recurrent laryngeal nerves (as portrayed in Figure 3) [12]. The recurrent laryngeal nerve is vital in the opening and shutting of the vocal folds (i.e., adduction and abduction). And, the superior laryngeal nerve merely stimulates the cricothyroid muscle and serves as a sensory nerve for the laryngeal mucosa above the vocal folds [13]. Damage to any of these nerves may culminate in vocal cord paralysis, obstructing the tensing and lengthening of vocal folds. Moreover, damage to both nerves is life-threatening, as it will impair breathing and cause the foreign particles to choke the trachea.

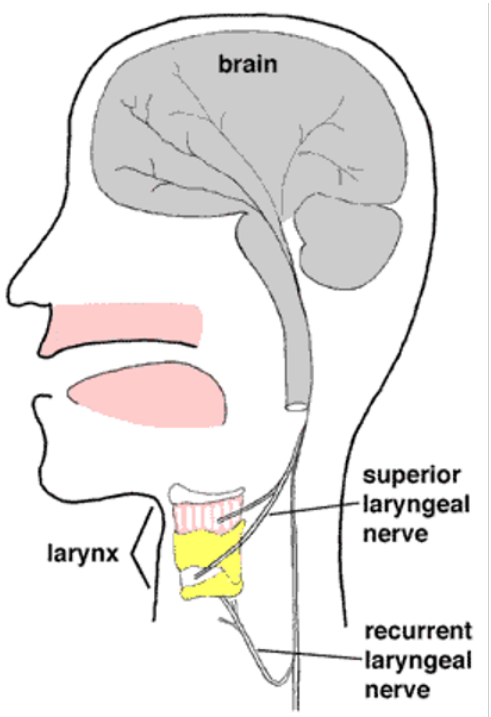

Figure 3. Two branches of the Vagus nerves accessing the larynx [12]

Comprehensively, it seems that various factors contribute to the creation of acoustic waves that ultimately form words, sentences, and speech. Among these elements, the lungs are generally considered as the source. But, in our study, rather than viewing the lungs as the source of vocalization, we assumed that genuine sound generation began with neurons. These neurons provide an electrical signal to the lungs, instructing them to begin sound production via the process of respiration. Therefore, our study addresses the topic of voice creation in an innovative way,

which subsequently contributes to the existing literature in the following way:

- For the anatomization of the vocal fold vibrations, we break down the voice creation process between two zones. Zone 1 incorporates the brain, neurons, and the two branches of the vagus nerve. And, Zone 2 comprises mechanical apparatus including lungs, and the articulators of voice, i.e., lips, tongue, teeth, jaws, larynx, pharynx, vocal cords, vocal folds, nasal cavity, oral cavity, and epiglottis.
- Communication between these two zones is only possible via impulses. Such that zone 1 sends electronic impulses to activate the mechanical apparatus in zone 2. In Zone 2, these electric impulses are converted into mechanical impulses or force. For a healthy person, this mechanical force is equivalent to the electric impulses generated in Zone 1. Thus, both zones work together to produce sound waves, including amplitude, time, and space, from the source to the outcome.
- We also discussed and reviewed the effect of neurodegenerative Parkinson's Disease on vocal folds, especially to identify the probable impact of dysphonia, dysarthria, and hypophonia on the anatomy of the voice box.
- The paper further examines various mathematical models analyzing vocal fold oscillations and their respective limitations. Since voice production is a cause-and-effect relationship, thus we used double point Green's Function method to solve the problem of vocal fold oscillation. This adds the causality factor and delay term to the problem and also illustrates the potential problems that could arise during the creation, conversion, or transition of impulses during speech production. Hence, our approach addresses the problem at the time when sound waves are in the form of electromagnetic impulses in the brain.
- However, due to the lack of test subjects, the proposed treatment for analyzing the vibratory patterns of vocal folds is limited to a theoretical approach. Finally, we also identified some unexplored limitations of current mathematical models, which can be addressed in the future.

Since our approach divides the entire study of voice production into two zones, thence the anatomy of sound production in Zone 1 and the process of voice production with mechanical apparatus in Zone 2 are discussed in Section 2. The process of voicing in speech is summarized in Section 3. Similarly, changes observed as vocal impairments in any or both zones during a neurodegenerative problem like Parkinson's Disease (or PD) are deliberated in Section 4. Some of the renowned mathematical models representing the physical and functional structure of vocal folds are discussed in Section 5 accompanying their benefits and drawbacks. Section 6 outlines our view of the entire process and suggests a probable method to identify the cause-and-effect relationship observed in voice production. The significant contribution of this theoretical approach is comparatively discussed in Section 7 followed by a Conclusion in Section 8 highlighting the future possibilities.

2. Zonal View of Voicing in Speech

A. The Brain and Voice

Besides the involvement of the vagus nerve's recurrent and superior laryngeal nerves in triggering vocal fold abduction and adduction, and stimulating the cricothyroid muscles. Some studies often suggest that certain regions of the brain exhibit higher levels of neuronal activity than the rest of the brain when hearing, analyzing, and preparing a response to a voice. According to functional Magnetic Resonance Imaging (fMRI), these regions known as Wernicke's and Broca's areas, are located in the left lobe of the brain. The Broca's region named after French surgeon Paul Broca in 1861 [14] is located at the frontal lobe of the brain in the motor cortex region above the Sylvian fissure. While German neurologist Carl Wernicke identified the Wernicke's region in 1874 [15] situated near the auditory cortex in the left hemisphere of the brain. These two areas are together connected via the arcuate fasciculus, which further attaches them to the parts of the brain. Flinker and colleagues [16] recast Broca's area in the role of scriptwriter. As it not only frames a plan for speaking and shares a copy of it with the motor cortex (in zone 1), but also shapes the speech by coordinating the movement of various articulators (in zone 2). Language comprehension (in written as well as articulated dialectal), sensorimotor learning and certain hand movements are too controlled by this region. Similarly, the Wernicke area manages the articulation of phonemes (or phonologic retrieval) and controls the temporoparietal junction [17].

To study the role of neuronal oscillations in the production of language, Piai and Zhang reviewed several works describing the motor and memory aspects of speaking [18]. They asserted that theta frequency bands of ElectroEncephaloGram (EEG) could clarify the motor components of speech. While memory-related task like language comprehension could be explained by using the alpha-beta bands (ranges between 8 to 30 Hz). Dichter and colleagues performed experiments on 12 epilepsy patients who had electrodes implanted in their brains to determine which area of the brain was responsible for controlling pitch [13]. "I never said she took my money", was the phrase provided to these patients to speak while stressing different words. The neuronal activity recorded by the electrodes in the dorsal laryngeal motor cortex (dLMC) suggests its accountability for altering pitch when emphasizing different words. Continuing the same work, Chartier et al. [19] discovered that stimulating the neurons in the dLMC and the nearby region elicited movement in larynx muscles that resembled the vocal fold oscillations.

Several other studies were also carried out to find out the role of basal ganglia on vocalization. In this series, Turk and colleagues added the impact of dopamine on the process of speeching [20]. They revealed the brain as a controller of the orofacial region and respiratory movements that was further connected to the basal ganglia-thalamocortical circuits in the brain. These circuits comprises mechano- and chemo-receptors, which took proprioceptive information, and initiated the motor movements in the laryngeal muscles. Apart from other roles of dopamine, i.e., controlling the coordination among movements, cognitive function, sense of emotional stability, and so forth. Interestingly, dopamine was also found to be capable of generating appropriate pitch and fluidity during vocalization. This result was demonstrated by Poeppel and Assaneo [21] by experimenting on songbirds, rats, and primates. As per studies from animal models, the basal ganglia and cerebral cortex's interactions with dopamine also govern our ability to produce voice. Ford et al. [22] were the initial researchers who thoroughly demonstrated Broca's, Thalamus, and Basal Ganglia circuitries via diffusion-MRI tractography study of 10 persons (aged between 19 and 35 years) without any neurological condition. They explored that cortical initiation of phonological tasks was handled by the basal ganglia while the cerebellum was responsible for the amplification and refinement. The work discussed in [23] suggested that Broca's region along with pre-SMA-basal ganglia circuitry was intricated in the selection of lexical terms, grammatical correction, intonation, and articulatory representation to ensure phonological unification.

Recently, Serra and colleagues performed intensive research to locate the region responsible for commanding the voluntary movements as well as depression [24]. For this, an optogenetic simulation was carried out on mice to activate their subthalamic nuclei. As a result, a habit of avoiding this simulation environment was observed in those mice, along with a persisting feeling of discomfort. After a few weeks, when the same mice were again tested without optogenetic simulation, they again exhibited strong avoidance behavior when placed in the same environment. This aversion assists in evading those circumstances and things that induce a feeling of disappointment. Activation of such strong senses in the subthalamus leads to depressive behavior, apathy, and other similar psychiatric symptoms. This revelation also disclosed the reason behind the side effects, especially depression, that appeared after subthalamic Deep Brain Stimulation (STN-DBS) used to treat essential tremors, Obsessive-Compulsive or Parkinson's Disorders. Further, during PD, as is known, the subthalamus region is overly active. And, simulating this region by implanting electrodes during DBS eliminates the tremors and related motor issues by simulating the subthalamus nuclei.

These studies highlight the importance of Broca's, Weickner's and subthalamic regions of zone 1 in coordinating the muscles associated with the production of voice. Further, dorsal pre-motor cortex's hypoactivation leads to inappropriate phonatory control. With respect to our study, this highlights that the generation of either incorrect or unnecessary electric impulses in this region (i.e., the brain) can impair the consequent signal. The same can be observed in the neurodegenerative diseases affecting muscular coordination like Parkinson's Disease.

B. The Mechanical Apparatus for Vocal Fold Vibrations

As also discussed in Section 1, Zone 2 includes mechanical apparatus such as the lungs and voice articulators, including the tongue, lips, teeth, jaws, larynx, pharynx, vocal cords, vocal folds, nasal passages, the mouth, and epiglottis. Zone 1 sends electronic impulses to stimulate (or trigger) the mechanical apparatus in Zone 2 which transforms these electric impulses into equivalent mechanical force (in case a person is healthy). In humans, speech production generally requires phonation and articulation. Phonation entails inhaling, allowing air to flow along the respiratory tract which is obviously from the lungs to the oral cavity, and finally exhalation. Whilst, articulation encompasses the shaping of sound with the aid of articulators. Besides these, the thyroid (singular), cricoid, and a few arytenoid cartilages all play an important role in phonation [25]. When stimulated by the laryngeal nerves in zone 1, these cartilages activate intrinsic laryngeal muscles to enable adduction and abduction of the vocal folds in zone 2 [26]. As is known, the vocal folds (or thyroarytenoid muscle) are fabricated by coatings of lamina propria and squamous epithelium [2]. So, in order to open vocal folds, the posterior cricoarytenoid muscle exhibits an outward rotation. Contrariwise, to close the vocal folds, inter arytenoid muscles along with the lateral cricoarytenoid muscles perform an inward rotation [25] [26]. In this way, changes such as lengthening, tension, and thickness needed for the modulation of the pitch are managed by the aforesaid cartilages.

Using a one-dimensional Euler-Bernoulli's type beam model, Serry and colleagues detected numerous intrinsic and extrinsic muscle motions generating diverse geometrical configurations of vocal folds [27]. The convex shape of the vocal folds was produced by co-activating the lateral criarytenoidal and inter-arytenoid muscles while leaving the thyroarytenoid muscle inactive. The only need to activate the thyroarytenoid muscle was to generate the concave geometry of the vocal folds, depicting the posterior glottal opening. Furthermore, an hourglass-shaped vocal fold arrangement can be created by engaging all adductory muscles. Similarly, via glottal source analysis, Novotny and others [28] reported less phonatory disturbance in the very beginning stages of Parkinson's disease. While gender differences had a perplexing influence on the evaluation of voice. Arias, Cortés, and Olmo conducted experiments to show that biomechanical voice characteristics such as duration of phonation, amplitude and frequency deviation, and pitch alter with gender [29].

Likewise, Padmanabhan et al. performed a test on 10 male and 9 female Parkinsonian patients having an average

age of 67 years [30]. Their study tried to assess the amount of effort generated by these patients during a simple task and the role of dopamine in performing it. The job was to squeeze a sensor-equipped handgrip on two different days within 4 weeks. For this, two groups were created, where one group could continue their regular dosage of dopamine and the rest had skipped their dosage before 12 hours of testing. With different levels of effort, the experimental results inferred that patients on a continual dosage of dopamine exhibited less variability and more consistency in squeezing the handgrip. While patients who skipped the dopamine dosage found this task harder than the other group. Moreover, they exhibited more variability in grips. Again, another coin-flipping experiment was conducted on these groups, yielding more or less the same results. In summary, this study demonstrated that the amount of effort demanded to accomplish a task was substantially influenced by dopamine levels. Alternatively, for instance, if an activity involves more physical exertion, it motivates fewer individuals. Thereby, it was revealed that dopamine regulates how much effort a person willingly puts out to finish a future task. Further inadequate dopamine levels signified persistent weariness, which was also observed in illnesses like anxiety, cancer, depression, and COVID. As this work suggests, inadequate levels of dopamine cause a patient to put more energy in usual tasks.

Werner and colleagues examined the influence of airflow direction (i.e., inhalation and exhalation) on the acoustic qualities and different configurations of the vocal tract [31]. They analyzed breathy noises produced by 100 men and 34 women while phonating four vowels and four fricatives with static vocal tract configurations, and developing a three-dimensional spectrum. During analysis, the spectra below 3 kHz exhibited moderate to strong overlap with resonances, from female to male voice samples. These results demonstrated that subglottal resonances have more significance in the quality of voice produced with varying vocal tract configuration rather than the flow of air.

This section discusses various configurations of muscles, and different articulators to allow voicing in speech. Suggesting that the low level of dopamine in Parkinson's patients compels such patients to exert more effort into inhaling and exhaling the air, as well as in modulating the air in zone 2. Additionally, the changes caused by decreasing muscle strength or decreased coordination between muscle and brain highlight the need for in-depth analysis in this area.

3. The Process of Vocalization

Therefore, the entire process of voicing in speech can be summarized into two main functions performed by the larynx. Beginning with setting up of vibrations, and then modulating the air.

A. *Setting up vibrations*

Since the actual power behind the production of voice is breathing. Thus, the foremost step is inhaling the air into the lungs. This air flows upwards through the trachea, and then to the voice box (or in between vocal folds). This airflow initiates vibration in vocal folds, which continues until one stops talking or is out of breath.

B. *Modulation of the air*

The mouth comprises several articulators, such as the tongue, teeth, lips, and nasal and oral cavities. Now, the air which initiated the vibration in the vocal folds reaches the supraglottal tract, where it experiences certain constrictions including the expanding or contracting of the vocal tract walls [32]. It causes the subglottal pressure to fluctuate between 600-1200 Pascal, thus producing turbulence in the airflow. Then, the different amounts of resistance and passage to air offered by the articulators while phonating, generates the voice [33]. During this entire aerodynamic phenomenon, the opening phase is controlled by the air pressure from the lungs and the closing phase is handled by the trailing Bernoulli effect, occurred due to the pressure difference created by the air passing through the laryngeal column [34].

In short, air from the lungs move upwards to the laryngeal pathway, where the force created by breath from the lungs causes the vocal folds to strike together enabling them to oscillate. These vibrations take the form of phonation, letter, word, or sentence, with the help of articulators, especially, the oral cavity, teeth, lips, and tongue. Meanwhile, Cricothyroid muscles controls the tension of the vocal folds coordinating the alteration of pitch [25] [26] [35].

In contrast to existing methods, the present work explains the process of voice production by dividing it into two distinct zones. Therefore, the forces responsible for this vibratory motion can be described in terms of zones, where the brain from Zone 1 sends electric impulses to Zone 2 to breathe and activates the posterior cricoarytenoid muscles. These electromagnetic impulses are transformed into mechanical force in Zone 2, where the pressure of the air coming out of the lungs rotates the larynx's arytenoid cartilage. This causes the vocal folds to provide a small narrow passage to air. Now, once the vocal folds get open, this flow of air can move up toward articulators. Since the air is passing through a constricted tube-like region, thus air pressure is quite high. This force drags the vocal folds together causing them to close. This phenomenon is known as the Bernoulli Effect and is usually observed in Zone 2. On the other hand, the lateral cricoarytenoid muscle shrinks as a result of electric impulses sent by the brain from Zone 1. This contraction of lateral cricoarytenoid muscles is opposed by oblique arytenoid muscles. In this way, the vocal folds meet in the middle or reach the equilibrium position at least once in a cycle. First initially (at the time of opening), and then at the time of closing, thereby creating a regular as well as repeating wave. This cycle continues until the person stops talking or the lungs are out of air. Moreover, the damping factor also plays a significant role in steady-state motion by acting as a viscous force introduced

by the lamina propria and squamous epithelium layers on the vocal folds. This entire aerodynamic phenomenon is identical to a harmonic oscillator [36].

4. Speech Problems in Parkinson's Disorder

As we know, any damage or injury to any of the apparatus in any of the zones causes impairment in the functions associated with that zone. According to our concept of dividing voicing in speech into two zones, two kinds of disorders may appear. The first is a language disorder in which the patient finds it difficult to understand other people and explain their thoughts and feelings. Similarly, any damage to either of the Broca or Wernicke areas causes aphasia in that region [37]. Like, during Broca (or expressive) aphasia there is a lack of ability to form sentences, where patients can conceptualize an idea but find themselves incapable of demonstrating it. Contrary to it, during Wernicke aphasia (or receptive/sensory/fluent aphasia) due to lack of understandability, the patient speaks jumbled words. They speak with fluency, rhythm, and proper grammar, but the uttered words make no sense. Other is associated with an injury to cartilage, muscles, nerves, brain, articulators, etc., and any disorder distorting the voice, such as laryngitis, vocal cord paralysis, precancerous and cancerous growths, cysts, thyroid, Parkinson's disease, over-use of voice, smoking, screaming, and so forth [6-10]. One such disorder, which appears due to the hypoactivity of dopamine-producing neurons, thereby inhibiting motor control is named Parkinson's Disease (PD). It causes Dysphonia, Dysarthria, and Hypophonia, and results in a monotonous, breathy, and hoarse voice with reduced loudness [38] [39]. Moreover, in a review study, Wang and others found that during Parkinson's disease, defective signals were transmitted to the frontal areas of the brain (including the Broca's region) by the basal ganglia [40].

Numerous researches successfully detected Parkinson's-induced voice disorders, firstly using Recurrence Period Density Entropy (RPDE) [41] and Pitch Period Entropy (PPE) [42]. Then Mel Frequency Cepstral Coefficients (MFCCs), and their first derivatives (Delta) [43]. And, now Relative Fundamental Frequency (RFF) [44] [45]. In this series, the association among acoustic, clinical, and perceptual changes detected in the voice of PD patients, was studied by Ma, Lau, and Thyagarajan [25], using aerodynamic assessments, Laryngeal Electromyography, Laryngoscopy, Computed Tomography, Photoglottography, Stroboscopy, and Pulmonary Function Testing. They re-established the known facts observed as Parkinsonian symptoms, which were escalating pitch, jitter, shimmer, and glottal opening time, and a decrease in the Harmonics-to-Noise ratio. As obvious, jitter signifies unstable oscillation in vocal folds causing roughness in voice. While shimmer and glottal air leakage were related to breathy voice. Similarly, the Videostroboscopy analysis of Parkinson's patients demonstrated the asymmetric oscillations of the vocal folds, where the glottal opening time was found to be longer than the glottal closing time. The same was cross-verified with Photoglottography results. The aerodynamic assessment of respiration in PD patients revealed a reduction in maximal inspiratory, expiratory, subglottal pressures, laryngeal resistance, and peak airflow. Laryngeal Electromyography and Laryngoscopy identified an increase in the activities of thyroarytenoid and cricothyroid muscles and a reduction in the posterior cricoarytenoid muscle activity during PD. Both of these changes occurred regardless of disease severity. Thus, the devoiding typical reciprocal forces between antagonistic muscles demonstrate the onset of vocal fold bowing (or atrophy).

In another work based on Laryngoscopy of the larynx of Parkinsonian patients, Castro et al. [46] showed that the raising breathiness and dropping intensity of voice could be correlated with trouble in sustained prolonged phonation. These changes, generally, arose due to increased glottic gap, and asymmetry in vocal fold closure. Similarly, Yücetürk, Yilmaz, and Egrilmez [47] laryngoscopically analyzed PD patients under dopamine treatment and found a high amount of abnormality in glottal closure, the amplitude of vibration, and vocal fold edges of Parkinsonian patients rather than healthy individuals. Zhang et al. [48] proposed increasing the thickness of the vocal folds as a treatment for elevated glottal tremors, chaotic vocal fold oscillations, and phonation threshold pressure. Moreover, Tsai, Wu, and Liou [49] associated the vocal cord impairments observed in Parkinsonian patients with nigrostriatal dopaminergic dysfunction. An in-depth review of various speech disorders associated with PD and methods such as Lee Silverman Voice Treatment (LSVT), Deep Brain Stimulation (DBS), and speech therapy to treat those disorders were discussed in [50]. Rapoport and others discussed the appearance and changes brought by dysphonia in aged persons [51]. Aging also has an inevitable effect on the larynx, which reduces the quality of life. However, this can be improved through vocal work practice. While slowness in the vocal fold closure and decreased respiratory driving pressure during PD can be correlated with a systemic decline in laryngeal control and the progression of the disease. In this way, they posited that during PD, the inadequacy observed in the conversion of the aerodynamic energy of the inhaled air into acoustic sound was due to the weakened motor function.

Recently, Castillo and colleagues examined 42 PD patients and assessed that the peak subglottal pressure was found to be linearly associated with maximum expiratory pressure symbolizing expiratory muscle strength and voluntary cough [52]. Yang et al. discussed the vocal changes observed during the second phase of PD [53]. These considered changes appear due to the trunk, vocal, and facial muscle hypertension resulting in declined voice quality, poor articulation, reduced rhythm, trembling voice, and high-frequency voice with reduced volume. Other noticeable changes were evident as a lack of fine control over muscles of various articulators of Zone 2, which are the larynx, chest, face, and mouth. Meanwhile, reduction in dopamine receptors and muscle tone, increases bass, voice

tremors, and apathy. In another work, Yang and others compared acoustic parameters between Parkinsonian and healthy individuals, and identified significant differences in the fundamental frequency of both gender's participants [54]. During monosyllabic and disyllabic tests, male Parkinson's patients exhibited a change in duration and jitter, whereas, in female PD patients, changes in the median intensity were observed.

Illner et al. conducted an audio analysis of 459 vowel articulation samples using a phoneme recognizer and formant tracker [55]. With this, they screened dysarthria-related vowel articulation impairment in a wide range of progressive neurodegenerative conditions such as PD, Progressive Supranuclear Palsy, Multiple-System Atrophy, Huntington's disease, and many more. Manes and colleagues utilized functional MRI to investigate motor cortex activity in the brains of PD patients and elderly healthy people during sustained vowel phonation [56]. In addition to equal bilateral activity of the laryngeal-phonatory and supplementary motor regions of the primary motor cortex in both types of participants, Parkinson's patients showed a decline in the activities of the right dorsal premotor cortex. Thereby, suggesting that functions in the right dorsal premotor cortex were found to be positively connected with maximal phonation time in the PD group but inversely associated with the regions controlling loudness and pitch.

So, it can be said that the source of the problem can be either in zone-1 affecting the function of the apparatus in zone 2, or it could be in the cricothyroid muscles, larynx, and rest of the mechanical apparatus of zone 2, which perverted the consequences of signals triggered by zone 1.

5. Mathematical Models for Analyzing Vocal Fold Vibrations

Several complex and high-dimensional one-[57], two-[35], and multi-mass biomechanical models [58] exhibiting oscillation of the vocal folds had been designed. The very basic among these is the one-mass model, which solely simulates flow-induced vibration of the vocal folds [57]. It considers two vocal folds as a single mass hanging from a spring with non-linear damping properties. Thus, behaving as a simple mechanical oscillator, it lacks self-sustained oscillation and can produce only lateral displacement of mass. In this way, one mass model is unable to explain the detailed behavior of vocal cords. However, such another simplified model in accordance with the body-cover concept of physics was only able to explain small amplitude oscillations in low-dimension [59].

To overcome the drawbacks of the one-mass model, Ishizaka and Flanagan demonstrated a two-mass model [35], comprising two distinct masses m_1 and m_2 suspended as springs and having a damping coefficient to represent their viscous characteristics. This introduced the self-oscillation in the model and addressed other minor and major limitations of the previous model. However, this model did not include the impact of multiple layers of tissues of vocal folds on sound production and was also unable to explain soprano singing, which was later on addressed in [60]. Adachi and Yu also proposed a two-dimensional model of vocal fold vibration in which the vocal folds were thought to be a pair of single masses oscillating perpendicularly and parallelly to the outflow of air. Both the suspended masses form a parallelogram shape, whose center is the equilibrium state of the vocal folds. It means the mass M of vocal folds is supposed to be centrally located, such that the displacement vector $\mathbf{r}$ depending on time t is given by $\mathbf{r}(t) = 0$. Consequently, after abduction and adduction, the vocal folds must have to return this position, i.e., the position of rest. In other words, at the equilibrium position, no force is acting on the vocal folds. Now, during oscillation, the parallelogram shape assumed for these vocal folds gets deformed, thus executing both elastic and swinging motions. In this way, the vocal fold vibratory pattern is similar to a harmonic oscillator as established by many physicists both theoretically as well as experimentally [60] [61] [62] [63]. Hence, their model not only attained the self-sustained oscillation but also achieved stability of pitch F_0 near the First Formant frequency F_1. This also demonstrates a smooth transition between oscillations, even while F_0 sometimes surpasses F_1 during high-pitched soprano singing.

Besides this, the multi-mass models not only captured the effect of separate tissue layers of vocal folds but also possess various degrees of freedom while computing various parameters associated with vocal fold oscillations [58]. Moreover, it explained the production of voice from various perspectives, but the multi-mass models have a drawback, which is high computational expenses [61]. Another three-dimensional body-cover phonation model by Zhanga [64] studied vocal folds to identify the conditions responsible for irregular vibration. Based on variations in stiffness, geometry, and shape of the vocal tract, they categorized voice into regular, subharmonic, and chaotic phonation. Their study further demonstrated that high subglottal pressure, low transverse stiffness, and large medial surface vertical thickness of vocal folds exhibit irregular vocal fold vibration. However, their work is unable to explain the conditions under which the redistribution of energy across vocal fold eigenmodes turns to vocal instabilities.

Additionally, to reduce the complexity of the mathematical simulation of vibratory patterns of vocal folds, a fixed boundary constraint is imposed at the lateral surface of the vocal folds. This causes neglection of the paraglottic area, which is a region between the thyroid cartilage and the vocal folds. To address this, in 2021, Li and colleagues developed a one-dimensional model using equations of conservation of mass and momentum, and machine learning techniques [65]. Since their model was a viscous flow model, thus it was able to effectively interpret different glottal shape geometries during vocal fold oscillation. They incorporated the pressure loss in the glottis observed during the entrance of air in the paraglottic area in subject-specific anatomies

as well. Thereby, providing a model, which is better and more explanatory than the previous one-dimensional models. Similarly, Wu and Zhang [66] considered the soft tissue layer forming the posterolateral border of the vocal folds, a component of the paraglottal area in their three-dimensional MRI-based model. They experientially inferred that if stiffness of below 10kPa was applied on this soft paraglottic space, it reduced the capacity of vocal folds to bear the subglottal pressure and maintain the closing position. This causes the vocal folds to vibrate with a reduced closed quotient and a larger mean glottal opening.

Another three-dimensional flow-structure interaction phonation model was developed by Wang et al. [67]. This model tried to overcome the limitation of the existing three-dimensional model by considering three layers of vocal folds. Their impact was to generate a stiffness parameter including both transverse- and longitudinal-elastic modulus. Consequently, a monotonic effect on the pitch, glottal opening, and flow rate was observed. Similarly, a significant non-monotonic influence was also observed on longitudinal rather than transverse stiffness parameters. Further, the most dominant effect on the output sound was the ligament layer. Zhao and Singh readdressed the self-sustained oscillatory motion of vocal folds using the body cover model [68]. They used the Adjoint Least Squares Estimation (ADLES) and ADLES-Vocal Fold Oscillation (VFO) algorithm to solve the vocal fold dynamics. Further, to simulate the expiration and vocal fold simulation Yokota and colleagues [69] suggested a coupled analysis method. Unlike the traditional two-mass model, their approach did not require parameter identification and was much swifter. Their work also identified the significant eigenmode among other eigenmodes of the vocal folds, which aided in generating vocal fold vibration. The numerical analysis yielded that during the self-excited mode of vibrating vocal folds second and fourth-order modes were dominant, air pressure was high, and Young's modulus was low.

Gender-specific variations in the glottal dynamics of vocal folds and deformation of airflow were assessed in [70]. This study considered elasticity, thickness, length, and depth of vocal folds via the aid of a fluid model, and airflow was modeled with Navier–Stokes equations. The experimental results suggested a decrease and increase in the thickness and length of vocal fold respectively, led to maximal tissue displacement. Similarly, flow rates significantly vary with variation in the depth of the vocal folds. Zhang reviewed various studies focusing on different laryngeal adjustments during phonation [71]. They inferred that the glottal closing and spectrum of voice produced were handled majorly by medial surface and vertical thickness. The pitch was controlled by the stiffness of the vocal fold, and a small glottal gap was a must for initiating sustained phonation. Jiang and colleagues suggested another two-dimensional simulation model, where intraglottal pressure was practically found negative after flow separation vortices improved vocal fold vibration [72]. As a result, an increase in aeroelastic energy transfer during vibration lowered the vibration intensity and the rate of flow by 20%, while the closure speed and maximum flow declination rate had been decreased by 40%. In another review study [73], authors highlighted the use of various ordinary as well as partial differential equations in the numerical modeling of phonatory systems in humans during the past decade. Moreover, Titze focused on how the shape of the airway and vocal fold adduction regulated with the lung pressure controls loudness [74]. They suggested that a bell shape airway configuration increase loudness with decrease in pressure. Similarly, a ring-shaped airway configuration again suggests increased loudness with a minute increase in pressure.

6. Our View of the Problem

History speaks of the presence of a cause behind every observed natural and man-made phenomenon. Cause and effect are two intertwined concepts, where the cause is an action that brings out reaction(s). It can be formally stated as the two events occurring at the same time and in the same place immediately preceding the other, such that the second event will never happen if the first event would not occur. Supporting this concept, many researchers worked to identify such cause-and-effect relationships in Parkinson's Disorder also. Some of them were the relationship between smoking and PD [75], caffeine consumption and improvement in PD [76], Nicotine and PD [77], the impact of environmental factors on the appearance of Parkinsonian symptoms [78], pesticides and PD [79] [80], the correlation between the death of dopaminergic neurons and α-synuclein [81], the impact of lifestyle on PD [82], vitamin D deficiency and PD [83], the relation of gastrointestinal disorder with the appearance of PD [84], oropharyngeal geometry and alterations in vocal attributes during PD [85], comparing dysphonia severity index with vocal severity in Parkinson's patients [86], and association of immune system with PD [87].

Since vocal folds behave as slightly damped harmonic oscillators, thus during the phonation of vowels the flow of air remains almost smooth. The main articulators that work are the tongue and lips [88]. The possible positions for the lips are neutral (for pronouncing vowels /a, and /e), spread (for /i), and rounded (for phonating /o, and /u). Similarly, the tongue can take back, front, and central positions with height adjustments from low, middle, and high. Additionally, the manifestation of the vocal folds during the unvoiced phonation (or whisper), commonly produced by Parkinsonian patients during the later years of the third stage shows that the vibration between vocal folds is missing as they remain stretched and abducted. In this way, the air only gets a very narrow passage between the arytenoid cartilages such that the audible sound is due to the turbulence of air passing through the larynx.

Consider the vocal folds to be a pair of suspended masses oscillating perpendicularly and parallelly to the lungs' airflow. Initially, vocal folds are closed and in an

equilibrium position, i.e., no force is implied on vocal folds symbolizing the position of rest. Then, the expunge of air from the lungs creates a subglottal pressure just below the glottis to ensure the opening of vocal cords. Once this subglottal pressure goes beyond the glottic closure force, it opens the vocal folds. So that air can pass through it and enter the larynx. In this way, the air coming from the lungs acts as a source of energy (or Driving Force) to begin phonation [89] [90]. These vocal folds remain open until the pressure of the air from the lungs is greater than the viscous force of the vocal folds. When the supraglottal pressure created by the high flow of air moving through the small opening of the vocal cords becomes weak, a suction effect is generated that draws vocal folds together. This is also known as the Bernoulli Effect. This is one complete cycle of vocal fold vibration.

Similarly, another force acting to slow down the vocal fold oscillation is the damping force. When a damping force acts on an oscillatory system, the energy of the system begins to dissipate. It leads to a decrease in amplitude and a change in the angular velocity of the vocal fold oscillations depending on the applied damping. As a result, when the damping ratio is one, the vocal folds remain in their resting state with no vibration. While the vibration remains unattenuated in amplitude at a damping ratio equivalent to zero [90]. It means the vocal fold's vibration too depends on the viscous force generated by the thickness and mass of the mucosal membrane of the vocal folds. A very low damping ratio appears to be detrimental to phonation because it causes uneven vibration and impairs the ability to produce a quick change in vocal pitch. Thence, a long and sustained phonation is fairly supported with a small amount of damping ratio as also suggested in [91].

A. *Solving the Equation of Motion of Each Vocal Fold Using Green's Function*

Adhering to the concept that left and right vocal folds exhibit symmetric vibrations, and as per the two-dimensional model of vocal fold vibration proposed in [60]. Each vocal fold's equation of motion can be given as:

$$\frac{1}{2}M\frac{d^2\mathbf{r}(t)}{dt^2}+\frac{1}{2}\gamma\frac{d^2\mathbf{r}(t)}{dt^2}=\sum f_i \tag{1}$$

where M is the mass of the vocal fold, $\mathbf{r}(t)$ is a position vector depending on the X- and Y-coordinates that themselves depend on time t, and γ is the damping or resistance coefficient of two dampers (or vocal folds). In order to consider the problem more sensitively, we introduced the term effective mass $m_e=\frac{1}{2}M$, effective damping coefficient γ_e equivalent to $\frac{1}{2}\gamma$, and effective force f_e replacing $\sum f_i$, which is the resultant term indicating various forces. All these terms transform Equation 1 as:

$$m_e\frac{d^2\mathbf{r}(t)}{dt^2}+\gamma_e\frac{d\mathbf{r}(t)}{dt}=f_e \tag{2}$$

Generally, the equations suggested for voice production talk about either time or space, and leave the source unconsidered. So, the present approach tried to include both space and time constraints, and the other parameters associated with Zones 1 and 2 also. To achieve this, Equation 2 can be solved using the double point Green Function [92], instead of using the traditional numerical methods as fourth-order Runge Kutta [60] and other methods described in [35] [59] [60] [61] [62] [89] [91] [93].

According to the Green's Function method (GF), the equivalent equation of motion for Equation 2 with a delay term t' is [90]:

$$m_e\frac{d^2G(t,t')}{dt^2}+\gamma_e\frac{dG(t,t')}{dt}=-\delta(t,t') \tag{3}$$

The conventional GF solution can be obtained by multiplying Equation 2 by $G(t,t')$ and Equation 3 by $r(t)$, which gives:

$$m_eG(t,t')\frac{d^2\mathbf{r}(t)}{dt^2}+\gamma_eG(t,t')\frac{d\mathbf{r}(t)}{dt}=G(t,t')f_e \tag{4}$$

$$m_e\mathbf{r}(t)\frac{d^2G(t,t')}{dt^2}+\gamma_e(\mathbf{r}(t))\frac{dG(t,t')}{dt}=-\mathbf{r}(t)\delta(t,t') \tag{5}$$

Thus,

$$\begin{aligned}m_e\left[G(t,t\prime)\frac{d^2\mathbf{r}(t)}{dt^2}-\mathbf{r}(t)\frac{d^2G(t,t')}{dt^2}\right]+\\ \gamma_e\left[G(t,t\prime)\frac{d\mathbf{r}(t)}{dt}\mathbf{r}(t)\frac{dG(t,t\prime)}{dt}\right]=G(t,t\prime)f_e-\mathbf{r}(t)\delta(t,t\prime)\end{aligned} \tag{6}$$

Now, integrating both sides of the above equation from $-\infty$ to ∞ gives:

$$\begin{aligned}m_e\int_{-\infty}^{\infty}\left[G(t,t')\frac{d^2\mathbf{r}(t)}{dt^2}-\mathbf{r}(t)\frac{d^2G(t,t')}{dt^2}\right]dt+\\ \gamma_e\int_{-\infty}^{\infty}\left[G(t,t')\frac{d\mathbf{r}(t)}{dt}-\mathbf{r}(t)\frac{dG(t,t')}{dt}\right]dt=\\ \int_{-\infty}^{\infty}G(t,t')f_e-\int_{-\infty}^{\infty}\mathbf{r}(t)\delta(t,t')\end{aligned} \tag{7}$$

From fixed-end boundary conditions, L.H.S. in above Equation 7 is equivalent to 0, yielding:

$$\begin{aligned}m_eG(t,t')\frac{d\mathbf{r}(t)}{dt}\Big|_{-\infty}^{\infty}-m_e\mathbf{r}\frac{dG(t,t')}{dt}\Big|_{-\infty}^{\infty}-\\ \gamma_e\int_{-\infty}^{\infty}\frac{dG(t,t')}{dt}\mathbf{r}dt+\gamma_e\int_{-\infty}^{\infty}\frac{d\mathbf{r}}{dt}G(t,t')dt=0\end{aligned} \tag{8}$$

Now, using the Dirac Delta function, the conventional

GF solution of Equation 2 is:

$$\int_{-\infty}^{\infty} G(t,t')\, f_e - \int_{-\infty}^{\infty} \mathbf{r}(t)\delta(t,t') = 0 \tag{9}$$

$$\int_{-\infty}^{\infty} G(t,t')\, f_e = \int_{-\infty}^{\infty} \mathbf{r}(t)\delta(t,t') \tag{10}$$

$$\int_{-\infty}^{\infty} G(t,t')\, f_e = \mathbf{r}(t) \tag{11}$$

Now, using Fourier transformation integrals, Equation 3 can be written irrespective of $\mathbf{r}(t)$ as:

$$\begin{aligned} m_e \frac{d^2}{dt^2}\left(\frac{1}{2\pi}\int_{-\infty}^{\infty} G(\omega)\, e^{-i\omega(t-t')}d\omega\right) + \\ \gamma_e \frac{d}{dt}\left(\frac{1}{2\pi}\int_{-\infty}^{\infty} G(\omega)\, e^{-i\omega(t-t')}d\omega\right) = \\ \frac{1}{2\pi}\int_{-\infty}^{\infty} e^{-i\omega(t-t')}d\omega \end{aligned} \tag{12}$$

On solving,

$$\begin{aligned} m_e \int_{-\infty}^{\infty} G(\omega)\,\omega^2 e^{-i\omega(t-t\prime)}d\omega - \gamma_e \int_{-\infty}^{\infty} G(\omega) i\omega e^{-i\omega(t-t\prime)}d\omega = \\ \int_{-\infty}^{\infty} e^{-i\omega(t-t\prime)}d\omega \end{aligned} \tag{13}$$

Since the integration limit extends over a large interval, i.e., from $-\infty$ to ∞, hence by continuity we can write

$$m_e G(\omega)\omega^2 e^{-i\omega(t-t')} - \gamma_e G(\omega) i\omega e^{-i\omega(t-t')} = e^{-i\omega(t-t')} \tag{14}$$

$$G(\omega)\left[m_e\omega^2 - \gamma_e i\omega\right] = 1 \tag{15}$$

$$G(\omega) = \frac{1}{m_e\omega^2 - \gamma_e i\omega} \tag{16}$$

Therefore, $G(t,t')$ can be written as:

$$\begin{aligned} G(t,t') &= \frac{1}{2\pi}\int_{-\infty}^{\infty} G(\omega)\, e^{-i\omega(t-t')}d\omega \\ &= \frac{1}{2\pi}\int_{-\infty}^{\infty} \frac{e^{-i\omega(t-t')}}{m_e\omega^2 - \gamma_e i\omega}d\omega \end{aligned} \tag{17}$$

Solving this by residue method, such that in Equation 17, there exist two poles. One at $z = 0$, i.e., Zone 1 (the brain source) and the other at $z = \frac{i\gamma_e}{m_e}$ in the lower half plane, i.e., Zone 2 (mechanical apparatus).

$$ResQ(z)_{z=0} = \frac{d}{dz} zQ(z)\Big|_{z=0} \tag{18}$$

where $Q(z) = \frac{e^{-izt}}{z(m_e z - i\gamma_e)}dz$. Thus,

$$ResQ(z)_{z=0} = \frac{d}{dz}\frac{e^{-izt}}{z(m_e z - i\gamma_e)}\Big|_{z=0} = \frac{1}{\gamma_e}\left(t + \frac{m_e}{\gamma_e}\right) \tag{19}$$

Similarly, at the second pole,

$$ResQ(z)_{z=\frac{i\gamma_e}{m_e}} = \frac{d}{dz}(m_e z - i\gamma_e)Q(z)\Big|_{z=\frac{i\gamma_e}{m_e}} \tag{20}$$

Thus,

$$ResQ(z)_{z=\frac{i\gamma_e}{m_e}} = \frac{e^{izt}}{z}\Big|_{z=\frac{i\gamma_e}{m_e}} = \frac{m_e e^{\frac{\gamma_e t}{m_e}}}{i\gamma_e} \tag{21}$$

Substituting Equations 19 and 21 in

$$\int Q(z)dz = 2\pi i \sum ResQ(z)\Big|_{z=0,\frac{i\gamma_e}{m_e}} \tag{22}$$

gives $G(t,t\prime)$:

$$G(t,t\prime) = \frac{m_e e^{\gamma_e(t-t')/m_e}}{\gamma_e} + \frac{i}{\gamma_e}\left(t - t' + \frac{m_e}{\gamma_e}\right) \tag{23}$$

Now substituting Equation 21 in the conventional solution of GF, i.e., in Equation 23. The displacement vector $\mathbf{r}(t)$ will be

$$\mathbf{r}(t) = \int_{-\infty}^{\infty}\left[\frac{m_e e^{\gamma_e(t-t')/m_e}}{\gamma_e} + \frac{i}{\gamma_e}\left(t - t' + \frac{m_e}{\gamma_e}\right)\right] f_e dt \tag{24}$$

Equation 24 represents the effect produced by m_e, γ_e, and f_e on vocal folds with time t and delay term t'. Since the formation of the signal cannot be traced in Zone 1, only its consequential actions are observed as the setting up of vibrations in the vocal folds (lying in Zone 2).

In this way, considering the problem of vocal fold vibrations as a boundary value problem helps in analyzing the real-world aspects of the problem. Green's function $G(t,t\prime)$ with a two-point function of position, depends only on the boundary conditions applied to Equation 2 and is devoid of the effective force term $F_e(t)$. The function is referred to as "two-point" as it represents the functions of two points in space and time [94]. Moreover, the use of the Dirac delta (as in Equation 3) assigns a symbolic meaning and an interesting physical interpretation to $G(t,t\prime)$, which is the displacement of the vocal folds because of an applied effective force of density delta. In short, a two-point Green's Function denoted by $G(t,t\prime)$ signifies the displacement of the vocal fold at time t (with delay term $t\prime$) caused by

a unit force applied on it by the air pressure from the lungs, generated via activating certain nerves in Zone 1. Additionally, the integration represented in Equation 11 over all possible points in the region of interest provides the total displacement of the vocal folds caused due to an applied force distributed according to the behavior of the function $F_e(t)$. In this way, we obtain the solution to Equation 2 pertaining to vocal fold oscillation. GF can also assess the causality, which depends on the driving force $F_e(t)$ at some pastime.

In simpler terms, utilizing the Green's Function approach for studying vocal fold vibrations reveal how the vibrations in the vocal folds change with different point sources. Concerning present work, it explains the consequences of any change in the zone 1, zone 2 or during transition in the vibrations of vocal folds. For example, during Parkinson's disease vocal fold remains open for a longer duration than normal persons. It means the probable cause of this repercussion lie somewhere either in dopamine chemical responders or in the inappropriate conversion of signals, i.e., generated mechanical impulse is not equivalent to the input electric impulse initially transmitted by the brain.

7. Discussion

Different disorders associated with the voice box are characterized by various symptoms and laryngeal muscle alignment. As is known, the inflammation of the voice box (or laryngitis) alters the normal cycle or symmetry of vocal fold vibration, particularly during adduction. Since this disease majorly appears as a result of pathogenic infection the afflicted region is likely limited to Zone 2. Similarly, an additional piece of tissue develops during polyps (or Reinke's edema) as the consequence of friction among vocal folds or frequent smoking. Aside from a low-pitched, rough, and breathy voice, an extra effort in voicing is observed as the late commencement of voice. Again, aberrant cell proliferation in laryngeal cancer affects exclusively the Zone 2 apparatus. The presence of a big fibrotic nodule in the topmost layer of the vocal folds hinders the mucosal wave. However, the harshness produced is limited to a particular pitch and involves both folds. Conversely, accumulated mucous on any of the vocal folds during cysts causes improper closure. This discussion suggests that the aforementioned ailments exclusively interfere with Zone 2.

Besides these, vocal fold paralysis and Parkinson's-related vocal issues influence both zones. During vocal fold paralysis, one or both vocal folds are paralyzed and stay open by remaining in the medial line, paramedian, or transverse orientation, even when gulping food. Generally, vocal fold paralysis is a neuro-architecture palsy in both branches of the vagus nerve caused by any lesion from the brain stem to the laryngeal pathway. In this way, the disorder's influence may be seen in both zones, where the main cause is disguised in zone 1 as a brain injury and the consequences are shown in zone 2 as paralyzed vocal folds. Further, multiple sclerosis and PD can also lead to this disorder in some cases. Likewise, during PD, hypoadduction (or vocal fold deformation) causes dysphonia, and slow speech reflects cognitive issues. The overactive superior laryngeal nerve elevates activity of the laryngeal and thyroarytenoid muscles resulting in Hypophonia. The circuitry of the basal ganglia has been demonstrated as a significant component for modulating dopaminergic processes and autostriato-thalamo-cortical circuits for context-dependent modifications (such as those required in singing). Furthermore, the substantia Nigra pars reticulata and subthalamic nuclei contribute significantly to the voice control mechanism of humans. Such that the striato-pallidal structure regulates motor activities generated as a response to emotional and motivational stimuli, with the dorsal portion handling emotional stimuli and the ventral portion managing motivational stimuli. Additionally, the amount of effort required in pitch modulation is determined by the release of dopamine. In this way, alterations in zone 2 have been linked to a decline in dopamine level, and other neuronal deficits in zone 1.

Since vocal fold paralysis also impacts both zones, thus can also be handled with our proposed theoretical concept for vocal fold vibrations. Thus, the mathematics of the aforementioned vocal disorders can be adequately explained with the present approach. Again, by constructing the double-point Green's function, a real-world interpretation of oscillating vocal folds in terms of applied effective force and obtained resultant displacement can be generated. Further, our view of the problem can also estimate the causality factors like impulse generation and transition behind such disorders. However, due to a lack of experimental data, we are unable to offer the empirical contribution and quantitative quantification of the amount of delay, as well as other factors such as effective force. Consequently, our study is limited to a theoretical idea. In terms of future experimental validation, procedures that can be utilized for this empirical estimation can be given as:

For measuring the hoarseness in the voice, stroboscopy and video-stroboscopy can be utilized. Both examinations monitor the vibration of the vocal folds to determine problematic regions. Photoglottography, on the other hand, follows the transmission of light across the glottis to measure the opening and shutting of the glottis. Recently, near-infrared Photoglottography (PGP) gained popularity. This technique not only retained the conventional functionality of PGP, but the added continuous transillumination captured the glottal cycles, glottal aperture, vocal contact, and vertical vocal fold edge movements. MRI and Computed Tomography can detect nerve damage and superfluous growth on the voice cords. Findings include anterior and medial orientation of the vocal folds, dilatation of the ipsilateral and laryngeal ventricles, thickness of the vocal folds, etc. Moreover, Laryngeal electromyography investigates how nerves govern the muscles of the voice box. For this, electrical impulses transmitting in these muscles were recorded and evaluated. To ensure that the lungs are performing properly,

Pulmonary function testing is conducted. Unlike the rest of the diagnosis tests, it is a non-invasive examination that measures lung volume capacity, flow rate, and gas exchange. The quantity of airflow and pressure needed during voice production is determined by aerodynamic evaluations of the voice. It also investigates the association between transglottal airflow, subglottal and phonation threshold pressure, and voice acoustics.

The other state-of-the-art algorithms for studying vocal fold oscillations either consider time or space and neglect the source. Therefore, firstly, the present approach tried to simplify the entire problem by dividing the entire process of vocalization into two separate zones. Then, the equation representing the harmonic oscillation of vocal folds is solved using the double point Green's function. The advantages of using Green's function are:

- In this approach, the Green's Function acts as a two-point correlation function. It assesses the likelihood of any alterations in the vibration of the vocal folds, while the source of this change lies in a different area, i.e., either in zone 1, zone 2, or somewhere in between the transformation of impulses.
- While solving any non-homogeneous differential equation like Equation 2, where to provide an immediate solution is quite complex with traditional numerical methods. The GF solution is constructed by performing point-by-point integration of convolutional GF against the term effective force f_e. This can be viewed as a combination of countless solutions to Equation 2 with a point source, all summed up to match the arbitrary forcing term f_e. At this point, the Dirac delta function helps in approximating the force of the impact.
- Likewise, the Fourier transformation of GF helps in swift yet mature conversion towards the solution in position space constraint with time $G(t, t\prime)$.
- Finally, GF helps in evaluating the amount of response produced in the form of displacement in the vocal folds due to a delta function source subject to time.

The absence of potential subjects is the prime limitation of this study. Since with no experimental input, the quantitative assessment of the amount of delay and effective force cannot be made. Thus, currently our work is a theoretical concept only. Consequently, the validity of this suggested GF solution in detecting the point source contributing to bring a change in the vibratory pattern of vocal folds can be experimentally verified in future studies with the availability of potential subjects. Moreover, this suggested solution can also be applied to assess other disorders associated with vibratory patterns of vocal folds.

8. Conclusion

The work presents a theoretical analysis of researches regarding vocal fold vibration by categorizing it into zones 1 and 2 signifying the brain and the mechanical apparatus respectively. Recent works suggest that the dorsal Laryngeal Motor Cortex and Broca's area of zone 1 are found responsible to manage voluntary vocalization. While the role of effective forces such as Bernoulli's, driving, and damping forces are also highlighted. Based on the reviews of laryngoscopic studies, the work further discusses the changes observed in voice quality as well as on a zonal basis during a neurodegenerative disease like Parkinson's Disorder. Correspondingly, we treated the phenomenon of vocal fold vibration as a harmonic oscillator and thus used double point Green's Function for resolving the equation exhibiting vocal fold oscillation instead of using the traditional numerical methods. Thus, as an analytical complex function, the analysis of effective displacement helps in estimating the role of the effective damping coefficient and effective force in the vocal fold vibrations. Further, delay term ($t\prime$) aids in identifying the changes observed in voice production. These changes may occur either (i) while generating electronic impulses in Zone 1, (ii) when the electromagnetic impulses from the brain are transformed into mechanical impulses required to initiate vibrations in the vocal folds, or (iii) due to any impairment in the mechanical apparatus and articulators of Zone 2 or because of any voice disorder or disease.

However, due to the lack of experimental subjects, the present computation is a theoretical assessment only, which is yet to be tested subject to the availability of cohorts. Besides this, considerable advancement has been made in reconnoitering the vocal fold oscillations and the physics of phonation, but some questions are still required to be elucidated in future works. These are:

- During EEG, the memory- and language-related operations are identified either under the alpha band or beta band, whereas in [18], such neuronal processes were unexplainably found in between alpha-beta bands.
- Some experimental results like those demonstrated in [64] are based on animal models, which need to be simulated on human subjects due to the difference in the anatomies of phonation.
- The role of the Bernoulli effect, tension, and rigidity of vocal folds, and thyroarytenoid muscles in phonation has been undervalued, necessitating a more detailed examination of the effects of the produced voice's acoustics on voice transmission.
- More knowledge regarding the cause-effect relationship between physiological alteration and perceived changes in voice needs to be outlined. Any such information not only helps in building a thorough knowledge of the laryngeal changes needed to im-

prove voice quality but also will aid in the betterment of the diagnosis, treatment, and clinical management of certain disorders impairing the voice.

Furthermore, such relationships help in highlighting the emotion behind a speaker-specific speech. Thus, will also help to enhance the efficiency of existing machine learning tactics in detecting emotion from sound synthesis.

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