AERODYNAMIC AND ACOUSTIC MEASURES OF VOICE IN PERSONS WITH PARKINSON'S DISEASE

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Register Number: 12SLP001

A Dissertation Submitted in Part Fulfillment of Degree of

Master of Science (Speech Language Pathology)

University of Mysore, Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING

MANASAGANGOTHRI, MYSORE – 570 006

MAY, 2014.

CERTIFICATE

This is to certify that this dissertation entitled **"Aerodynamic and Acoustic Measures of Voice in Persons with Parkinson's Disease"** is a bonafide work submitted in part fulfillment for the Degree of Master of Science (Speech Language Pathology) of the student (Registration No: 12SLP001). This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any of the University for the Award of any other Diploma or Degree.

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This is to certify that this dissertation entitled **"Aerodynamic and Acoustic Measures of Voice in Persons with Parkinson's Disease"** has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in other University for the award of any Diploma or Degree.

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DECLARATION

This is to certify that this dissertation entitled **"Aerodynamic and Acoustic Measures of Voice in Persons with Parkinson's Disease"** is the result of my own study under the guidance of Dr. Jayakumar T., Lecturer in Speech Sciences, Department of Speech-Language Sciences, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier in other University for the award of any Diploma or Degree.

Mysore
May, 2014

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Dedicated to.....

To my dearest and sweetest "uppuppaas" for their blessings and also for spending their valuable time with me. Despite their difficulties, they just came along with me and helped me in bringing out this data. They really did all that they can without considering their physical difficulties. You are appreciated and whole heartedly thanked.

Acknowledgement

I thank God Almighty for his wonderful creations on which human beings are continuing their explorations. I thank for the blessings that he showers on all of us involved in this study the and for listening to all of our prayers.

I thank my dear Dad, Mom and my sisters and in-laws for their care and support throughout my life.

Dr. Jayakumar T, my guide is a person with excellent patience. He listens as well as explains patiently and clearly. Most of the time he would be silent and it indicates he is in deep thought of our doubts. He never hurries for conclusion and that is what he should be appreciated. I got the mental strength even at times of extreme pressure only because of you sir….you are cool…

I thank Prof. S.R. Savithri, our Director, for giving us the platform to exhibit our abilities.

I thank all the staff in the department of SLP and SLS for their assistance in using the instruments. I thank Prof. R. Manjula for giving us the permission to use the instrument. Special thanks to Ms. Irfana and Ms. Gayathri and Ms. Sheela.

I thank Mrs. Vasanthalakshmi and Mr. Santhosha and for assisting and giving us the knowledge and training to use SPSS.

I thank Ms. Amulya P, for her valuable information and linking me with the BGS members – Mr. Venkat and Co.

I am blank when I wanted to thank my best friend…..

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CHAPTER I

INTRODUCTION

Voice production in human requires coordinated interaction of respiratory, phonatory, and resonatoryand nervous systems. Voice production occurs in three stages: (1) lungs generate air which is forced up through the larynx (respiratory level) (2) this air flows through the vocal cords, causing vibration of vocal cord mucosa and the sound is generated (phonatory level) (3) the generated sound is then shaped by the articulators to produce speech (resonatory and articulatory level). All these processes are controlled by the nervous system. Pathology anywhere along this path can result in voice problems. Thus expiratory air from the lungs is the source for voice production. The air exhaled from the lungs (respiratory) is modified by laryngeal modulations (phonatory) to create the acoustic waves. Resonatory system, a part of the vocal tract structures, amplifies and filters these acoustic waves resulting in voice production. Any impairment or incoordination in the above mentioned systems and mechanisms leads to disorders of voice.

Voice disorders can be congenital or acquired. The etiology may be psychogenic, neurologic, and endocrinologic in origin. Vocal abuse and misuse can lead to hyper functional voice disorders. Neurogenic voice disorders may be caused due to lesions at various parts of the brain and its associated structures/pathways. For example, lesions in the extrapyramidal system that consists of reticular substance, corpus striatum and the basal ganglia will affect the coordination of laryngeal function (Ward et al., 1981).

4Athetoid movements, Parkinsonian characteristic progressive abductor paralysis due to Shy-Drager syndrome are few examples.

Dysarthria, according to Darley et al. (1969), is characterized by all speech disorders related to disturbances of muscular control of the speech organs, whose origin could be a central or peripheral nervous system. Based on site of lesion and presenting symptoms, dysarthria is classified into different types. For examples Upper motor neuron system (UMN) lesions leads to spastic dysarthria, lower motor neuron (LMN) lesions lead to flaccid dysarthria while Basal ganglia lesions lead to either hyper or hypokinetic dysarthria. Generally all or either of the respiratory, phonatory, resonatory and articulatory systems may be affected, respiratory dysfunction being the commonest.Dysarthrophonia is the term used to refer to the voice problems occurring subsequent to dysarthria.

Thus depending upon certain characteristic features, dysarthria can be of various types. Hypokinetic dysarthria is one among them. The term hypokinetic dysarthria was introduced by Darley, Aronson, and Brown, 1969a). As with the term, the physiological basis for this type of dysarthria is 'reduction in mobility of movements' which is reflected in speech movements as well. Since the characteristics were identified by one specific disease: Idiopathic Parkinson's disease (IPD), Duffy (2005) consistently states that "hypokinetic dysarthria is the dysarthria of Parkinson's disease". Hypokinetic dysarthria occurs subsequent to basal ganglia lesion.**Parkinson's disease** (PD) is caused due to loss of dopamine secreting neurons in the substantianigra (basal ganglia structure located in the midbrain). As such the dopamine is deficient in such individuals. The functions of dopamine include motor control, motivation and reward, cognition and also in assisting

the transmission of other hormones. Reduction in motor control due to dopamine deficiency leads to features such as tremors, slowness of movements and rigidity. These features are the hallmark for the diagnosis of PD. In most of the cases the cause for PD is unknown and hence the term 'Idiopathic PD' is used. In addition there can be drug-induced PD and Parkinson- plus syndromes which are usually rare. Onset of PD is insidious and generally occurs after the age of 40 and later (middle and later stages of life).The speech symptoms that characterize hypokinetic dysarthria include: imprecise consonants, low speech intensity, reduced prosody (monotone), harsh and breathy voice quality and fast or inconsistent speech rate. The term Parkinsonian dysarthria is synonymously used with this type of dysarthria as suggested by Darley (1969). It was because the speech and voice characteristics resembled those of patients with Parkinson's disease (PD).

In general voice can be assessed by subjective or perceptual and objective or instrumental methods and techniques. Qualitative/subjective evaluations are done perceptually using standardized rating scales like GRABS, CAPE V, and VHI and so on. Quantitative/objective evaluations are done using instruments. It can be invasive or non-invasive. Invasive methods are usually carried out by medical personnel or under their supervision. It includes procedures such as video laryngoscopy, stoboscopy, which provide visual information about vocal fold vibratory characteristics and thus the structure and function of vocal folds and related structures can be observed. Non-invasive methods include recording and subsequent analysis of quantified values by the examiner or by the instrument itself (semi-objective or purely objective). Aerodynamic analysis provide information regarding respiratory capacities, pressure and flow and its related

measures while acoustic analysis provide information regarding frequency and its related measures, intensity and its related measures, noise related measures, tremor related measures and so on. These two are among the few examples of non-invasive objective voice analysis methods.

The coordination between the respiratory and phonatory mechanisms is called pneumophonic co-ordination. It is very important in normal voice production. The assessment of this pneumophonic coordination can be done using aerodynamic analysis methods. The aerodynamic analysis provides information related to the valving efficiency of the glottis during phonation. Aerodynamic analysis gives two kinds of information: (a). the static measures of respiration which includes lung volume or lung capacity measures like vital capacity, tidal volume, and functional residual volume and so on. It thus helps in knowing capacities of an individual's respiratory system; (b).dynamic measures of phonatory/laryngeal system that provide information about the laryngeal valvingefficiency in converting the expiratory airstream to acoustic energy. These measures include maximum phonation duration, s/z ratio, estimated subglottic pressure, mean airflow rate, laryngeal airway resistance, laryngeal airway conductance, phonation threshold pressure, and vocal efficiency.

Estimated subglottic pressure (ESGP) is the amount of pressure exerted on the vocal folds during adduction. The unit for ESGP is cmH_20. Mean airflow rate (MAFR) is the volume of air flow across the vocal folds during phonation in one second. The unit for measurement is milliliters/second (mL/s) or liters/second (L/s). The ratio of ESGP and MAFR gives the laryngeal airway resistance (LAR). It reflects the resistance offered by the vocal folds to airflow at the glottis level. Similarly, ratio of MARF and ESGP gives

the laryngeal airway conductance (LAC). It reflects the easy flow of airflow at the glottis level by the vocal folds. These parameters play an important role in diagnosis and management of voice disorders.

Acoustic analysis of voice is done using various instruments and software. The voice sample can be recorded in live or a stored sample can be analyzed. There are various measures that are extracted from the sample using these instruments/software. These measures can give us information regarding the voice of a person. Few of them are fundamental frequency (F_0) (unit- Hz) and its standard deviation (SD F_0), intensity (in dB), perturbation measures like jitter and shimmer. Jitter is the peak to peak variation of pitch is represented by the term jitter, while that of loudness is represented as shimmer (both are represented in percent). Any abnormal values of these measures (as compared to appropriate norms) indicate a disorder in voice.

Acoustic analysis is one of the advantageous among all the instrumental analysis. The literature concerning motor speech disorders are well studied using acoustic analysis. Perkell, Guenther and Lane et al., (2000) indicate that the speech production models hypothesize that acoustic targets serve as control variables such that the neural systems specify acoustic goals to guide articulatory movements. Thus acoustic analysis provides insight into the disturbances caused by neural deficits. Various instruments and software are available for the acoustic analysis of voice. Spectrograph is one of the earliest inventions among them (1930s). Nowadays computer-based speech analyses are emerging with advanced options, making the voice assessment more accurate and reliable. Computerized speech lab (CSL) is one such example. Certain software are free of cost (Praat) while some are relatively inexpensive (Lingwaves). Recently quantitative

evaluations of voice quality using indices like Dysphonia Severity Index (DSI) are emerging. DSIis an objective and quantitative correlate of the perceived vocal quality (Wuytset. al., 2000). The DSI is based on the weighted combination of selected set of voice measurements such as: highest frequency (High-F_0in Hz), lowest intensity (Low-In in dB), maximum phonation duration (MPD in seconds), and jitter (%).

1.1 Need for the study

Respiratory dysfunctions and phonatory impairments (voice disorders) occur frequently in dysarthria with respiratory dysfunctions being the major cause of death in individuals with PD in advanced stage. Also it is estimated that 60 to 80 % of those individuals with PD develop speech symptoms as the disease progresses with hypophonia or reduced loudness being the important and earlier symptom.Hypophonia in addition to other speech symptoms like reduced stress, imprecise consonant articulation, inconsistent and rapid rate of speech adds to the frustration of the person with PD (for example, theymay be frequently requested to speak louder and clearer). Voice quality is also observed to be harsh and breathy. Hoarseness is also noted. The incoordination of the respiratory and phonatory system as well as the reduced mobility of the structures within these systems could be the reasons for these symptoms. Hence a thorough analysis of these systems is necessary, which would help us in knowing the disease better and in formulating the areas to be concentrated for intervention

Assessment of physiological functions of phonatory and respiratory coordination in patients with PD using aerodynamic measures is limited to the western studies. The lack of literature with respect to aerodynamic and acoustic measures in Indian context brings forth the importance of the same. This study also makes an attempt to assess the

relevance of acoustic and aerodynamic measures in the assessment of voice quality in Parkinson's disease, using Dysphonia Severity Index(DSI). Hence the present study aims at investigating the coordination between the respiratory and phonatory systems using few aerodynamic parameters, and acoustic parameters in persons with Parkinson's disease in comparison with their peer group

1.2 Objectives of the study

- To obtain few aerodynamic and acoustic measures of voice in persons with Parkinson's disease and compare with their peer group
- To obtain voice quality (DSI) measures in persons with Parkinson's disease and compare with their peer group

CHAPTER II

REVIEW OF LITERATURE

Parkinson's disease is a progressive, degenerative, neurological disease associated with selective loss of dopaminergic neurones in the pars compacta of the substantianigra (Uitte and Calne, 1993). Hypokinetic dysarthria is most commonly associated with PD. In the absence of other influences (e.g., medication effects), hypokinetic dysarthria is *the dysarthria of Parkinson's disease* or "parkinsonian dysarthria". The term PD is usually reserved for Parkinsonism of unknown cause/idiopathic that is responsive to medical treatment (Duffy, J., 2005)

2.1 Etiological factors

There are several factors that can cause PD. These factors can be divided into three major categories. They are *primary causes; secondary causes;* and *PD plus syndromes* (Fahn&Przedborski, 2005). The primary cause for PD is unknown or *idiopathic* and it also includes sporadic disease which can cause few PD symptoms. The secondary PD, as the term indicates, includes PD that occurs as a secondary effect and thus the cause is known and identifiable. These causes include certain drug usage (neuroleptics), encephalitis, toxins (manganese, carbon monoxide, MPTP, cyanide), vascular insults, brain tumour, and head trauma. The last category is the PD plus syndromes where the parkinsonism symptoms may be caused by a known gene defect and have distinctive pathology, which includes progressive supranuclear palsy, multiple system atrophy, dementia syndromes (Alzheimer's, normal pressure hydrocephalous, frontotemporal dementia), hereditary disorders like Wilson's disease and Huntington's disease. In more than 80 % of the cases the cause for PD is unknown(Fahn&Przedborski,

2005). Thus Idiopathic PD is the most common among all the three followed by Parkinson plus syndromes (15%).

2.2 Incidence and prevalence

The prevalence of PD in the general population has been estimated to be 1 or 2 cases per 1000 (Weiner and Lang, 1989). It rises to approximately to 10 in 1000 as the age increases to 65 and above (Tanner and Goldman, 1996). The average age of onset is noted to be 60 years with10 % exhibiting PD as early as 40 years of age (young-onset PD). In India, PD is a common neurological disease. Around 5-60% of total movement disorders constitute PD with variations in different geographical area (Razdan, Kaul, Motta, Kaul, & Bhatt, 1994; Das, &Sanyal, 1996; Gouri e-Devi, Gururaj, Satishchandra, &Subbakrishna, 1999). PD was observed to be more prevalent in rural than in urban areas and it is more common in men (Gouri e-Devi, Gururaj, Satishchandra, &Subbakrishna, 1999. In a study conducted in Bangalore, Ragothaman,Murgod, Gururaj, Louis, Subbakrishna, &Muthane, (2006) reported that 24 % of their elderly population (above 60 years) studied had Parkinsonism with IPD being the commonest (71 %), followed by drug induced PD (2.5%), multiple system atrophy (2.5%), vascularParkinsonism (1.7%), progressive supranuclear palsy (0.8%) and the remaining unclassified. The average annual mortality rate was found to be 2.89/100,000 per year and the relative risk of death was found to be 8.98 (Das, Misra, Ray, Hazra, Ghosal, Chaudhuri, Roy, Banerjee, &Raut, 2010).

2.3 Course and neuropathology

There are two distinct phases that occurs during the course of the disease. They are the *pre-symptomatic* phase (early stage) and the *symptomatic phase.* As the term

indicates, during the *pre-symptomatic phase,* no overt signs or symptoms of the disorder are observed but the physiological changes have just begun. In the*symptomatic* phase (middle to later stages) the signs and symptoms become overt and the severity increases (Del Tredici, Rüb, Vos RAI de.,Bohl, &Braak, 2002; Braak, Del Tredici, Rüb, Vos RAI de., Jansen Steur, &Braak, 2003). Within the above two phases there are six neuropathological stages. The pre-symptomatic phase includes the first and the second neuropathological stage of the PD where the pathology is confined to the medulla oblongata/pontinetegmentum and olfactory bulb/anterior olfactory nucleus. The symptomatic stage includes the third stage to the sixth stage. In the third and fourth stages the pathology initially extends to the substantianigra and other nuclear grays, the midbrain and forebrain and then severe pathological changes occur. Most of the individuals at this stage cross the threshold point of the symptomatic phase of illness. During the fifth to the sixth stage, the pathology extends to the mature neocortex and the disease manifests in its entire clinical dimension. During the sixth stage the person with PD usually becomes bedridden. Table 1 shows different stages of PD suggested by Hoehn and Yahr (1967).

Table 1
Motor staging of PD by Hoehn and Yahr (1967)

Stages	Charecteristics
0	Asymptomatic
1	Unilateral involvement only
2	Bilateral involvement without impairment of balance
3	Mild to moderate involvement; some postural instability but physically independent; needs assistance to recover from pull test
4	Severe disability; able to walk or stand unassisted
5	Wheelchair bound or bedridden unless aided

2.4 Clinical features

The clinical features of PD include *bradykinesia, rigidity, resting tremor, postural instabilityand asymmetric onset* (Fahn, 1986). At least two of the first three symptoms should be present for a person to be diagnosed as having Idiopathic Parkinson's disease, with one of them being either tremor or bradykinesia, with a sustainable response to a dopaminergic medication. Bradykinesia is defined as "the unusual decrease in the amplitude and velocity of the movements which are voluntary in nature" (DeLong, 1990). It is manifested by masked face, decreased eye-blink, micrographia, loss loss of facial expression (hypomimia) and shuffling gait (Jankovic, 2008).Rigidity is defined as "the unusual increase in muscle tone resisting to the passive movements" (DeLong, 1990). It results in stooped posture and cogwheel phenomenon (resistance to passive stretch of the limbs). Resting tremors of frequencies 4 to 6 Hz are typical in PD. They may be unilateral at early stage. "Pill rolling" phenomenon is seen, which is defined as "the supination-pronation tremors of the distal parts of the extremities" (Jankovic, 2008). At the later stages there occurs postural instability leading to short and shuffling gait and imbalance (leading to frequent falls). Freezing episodes are noted during walking, wherein the patient feels difficulty in initiating a movement thus becomes immobile for few seconds.

During the early stages (1-5 years) the symptoms noted are unilateral resting tremor (usually the upper limb), unable to perform finer movements including dexterity which results in micrographic handwriting. Complaints of slowness of movements, stiffness and lack of power in one limb are milder. Reduction in facial expression and reduction in arm swing during walking are notices. Posture is slightly asymmetric and

stooped. During the middle and the later stages (5 to 15 years and above after the disease onset) the above mentioned symptoms increase in severity. Symptoms like freezing episodes (immobility) and dyskinesia (uncontrolled, rapid movements of limbs, trunk and head) occur. Dyskineticfeatures are the after effect medication (levodopa) used for treatment of the disease itself.

In addition to the above mentioned motor symptoms, the persons with PDalso exhibit few non-motor symptoms. These non-motor symptoms include cognitive impairments (deficits in attention and memory, executive functions), word finding difficulties, sleep disorders including vivid dreams, sleep fragmentation and Rapid Eye Movement behavior disorder, drowsiness during the day, restless legs syndrome, depression, apathy and fatigue, urinary and sexual dysfunctions and weight loss. These symptoms may vary from person to person. Swallowing difficulties are also noted

Among the speech motor symptoms, the most common perceptual features ofthese disorders are reduced loudness (hypophonia), reduced prosodic pitch inflection (monotone speech), hoarse voice, and imprecise articulation (Canter, 1965; Logemann, Fisher, Boshes, &Blonsky, 1978).

2.5 Laryngeal Aerodynamics

Speech production is a complex multisystem process that is accomplished through coordination of the sensory and motoric components of the respiratory, phonatory, resonatory and articulatory subsystems. Deficits in any one of these subsystems may likely cause deficits in others as well. Specifically events that alter the sensorimotor functioning such as progressive neurological diseases, cerebro-vascular accidents or neoplasms may affect the performance of these subystems.

Since speech is produced on exhalation, adequate respiratory control and coordination is a necessity for normal oral communication. Patients with a deficit in the sensory or motor components of respiratory system might thus have difficulty in normal speech production and in coordinating the respiratory and phonatory subsystems. It may also produce weakness and dyscoordination in the respiratory muscles, which may limit the exhalatory air available for subglottal pressure maintenance.

Aerodynamics is the branch of science that is concerned with the study of gas motion in objects and the forces that are created. Laryngeal aerodynamics is the specific field within this branch of science that studies the airflow and pressure that are produced during voice production and is considered as essential tool in the voice laboratory as part of the clinical voice evaluation (Dejonckere, 2000). Airway resistance is the relationship between pressure and flow, which provides information regarding the impedance of the airway during voice production.

Improvement in the technology in the past decades, had led to inventions of variety of instruments that are capable of measuring aerodynamic variables in a precise and better manner. Thus we have come a long way from simple manometric sensing devices to elaborate combinations of pressure transducers and airflow meters. The basic components of these aerodynamic measuring systems are pressure transducers (that record airway pressures within the vocal tract) and flowmeters/flow transducers (those record volume rates).

Few among the parameters that are used while studying the aerodynamic measures include Estimated Subglottic Pressure (ESGP), Mean Airflow Rate (MAFR) and Laryngeal Airway Resistance (LAR).

2.5.1 Subglottic pressure

Subglottic pressure (SGP) is the pressure exerted by the expiratory air column on the vocal folds. In voice production it acts as a force building up below the adducted vocal folds, rising until the folds overcome resistance and set the vocal folds into oscillation. Thus it indicates the valving characteristics of the vocal folds. Since direct measurement of the subglottal pressure poses serious technical limitations to SLPs, researchers of our field assume intra oral pressure as an indirect reflection of the subglottic pressure. Baken and Orkiloff (2000) states that the subglottal pressure is an important aerodynamic parameter and could allow a better understanding of somedysfunctions in speech production system.

Subglottic pressure is estimated indirectly by "InterruptedAirway Method" (Smitheran&Hixon, 1981), a non-invasive method validated notably by Demolin et al (1997).In this method, the participants were asked to phonate the syllable /pi/ in one stretch of several repetitions. During production of vowel /i/, the vocal folds are vibrating and the lips are open, hence the intra-oral pressure is equal to atmospheric pressure. During the production of unvoiced phoneme /p/, the lips are closed, the vocal cords are open, and subglottic pressure and intra oral pressures become equal (figure 1). Thus the intraoral pressure (IOP) is equivalent to the subglottic pressure (SGP) during the labial occlusion of phoneme "p". The intra oral pressure can be measured by an oral pressure sensor with an intra-oral tube attached to it. Therefore, the subglottic pressure can be estimated from the measured intra oral pressure which is relatively non-invasive and

comfortable to the participant being tested. The pressure thus measured is called as estimated subglottic pressure (ESGP).

To attain a normal speech production there is need for the coordination of the major subsystem. The coordination between respiratory and laryngeal subsystems is called pneumophonic coordination. The subglottic pressure measurement is a good indicator of this pneumophonic coordination because it depends on the expiratory airflow and laryngeal resistance. In other words, SGP results from a dynamic conflict between air thrust forces and laryngeal resistance, so the evaluation of its trend in a group of breath can give a powerful index of the speaker pneumophonic coordination(Teston, 2007).

Pressures in the oral cavity vary from 3-8 cms of H_2O for the production of non-nasal consonants in normal conversation. It may be little higher for voiceless consonants as the need for aerodynamic energy required for such voiceless productions are higher.An estimated subglottic pressure range from 5 to 10 cm H_2O has been reported by Holmberg, Hillman, and Perkell (1988, 1989), in their study of normal adults (25males in the age range of 17 to 30 years and 20 females in the age range of 18 to 36 years).

Patients with PD have been reported to show reduced values on many measures of aerodynamic function. Reduced intra-oral pressure during consonant-vowel productions have been reported by Marquardt (1973), Mueller (1971), Solomon and Hixon (1993), Sarr et al., (2009). Mueller attributes these findings to the rapid, slurred articulatory and imprecise articulatory valving noted in their subjects.Sarr et al., (2009) attributes this decrease of IOP in patients to inherent dopamine deficiency in PD. Dopamine deficiency induces a dysfunction of the respiratory muscles that is partly responsible for the dysarthria (Murdoch et al., 1989). In addition there is an overall poor control of

expiratory airflow, an alteration of the air quantity needed for the vibration of vocal cords (Jiang et al., 1999a; Solomon &Hixon, 1993).

In the contrary Jiang et al., (1999) reported increased values of mean ESGP in their participants with stage 4 of disease severity. They used external rapid acting valve to interrupt the phonation of /a/ vowel. The task was carried out 3 increasing intensity levels. Also the ESGP values increased with intensity. These findings are attributed to the compensation mechanism used by these individuals to overcome their inability to increase their loudness or in completely adducting the vocal folds. The higher value of mean ESGP implies a significant increase in Laryngeal resistance (LAR) during phonation, which can theoretically be related to the phonation using a small glottal aperture or vocal fold deformation noted in patients with PD. In the same study he also found an increase of mean ESGP and MAFR values as the intensity of sound production was increased. Fox and Ramig (1997) report a decreased mean value of SPL in their participants with PD (mean= 69 dBSPL) as compared to control group (73 dBSPL)

2.5.2 Airflow measures

Airflow measurements may be carried out the nasal and oral levels. Nasal airflow measures have been frequently used in assessing velopharyngeal competence while the oral airflow measures are more related to laryngeal aerodynamics. Major oral airflow parameters studied include the average airflow/mean airflow rate/mean oral airflow and maximum flow declination rate. In order to acquire an airflow signal a pressure transducer is required. Rothenberg (1973) introduced the circumferentially vented pneumotachograph facemask for the same. Criticism evolved regarding the use of such mask, as it could interfere airflow and voice production.However study by Huber et al

(2003) proved that there was no such influence of the Rothenberg mask in airflow signal acquisition during voice production.

Bless et al (1993) reported an average airflow ranging from 40 to 32 cc/sec in adult men (mean of 119 cc/sec) and 50 to 220 cc/sec in adult women (mean of 115 cc/sec), in a healthy laryngeal condition. As it gives only a general idea regarding laryngeal function but not about the flow modulated at the level of glottis, use of the same singly is not advocated. However, it has been used as an indicator of pre and post-surgical treatment outcome(Bielmowicz, 1995; Kimura et al., 2008).

The findings of MAFR are varied in persons with PD. Reduced values of MAFR has been reported by some authors (Jiang and Tao, 2007; Sarr et al., 2009). Sarr et al., (2009) used 'airway interruption method' to estimate ESGP, MAFR and LAR, in persons with mean duration of disease being 9 years. They used sentence production task. 6 points of measurements within the sentence were taken. The values of MAFR were reduced and significantly different from the control group only during the course of sentence production and not at the start and end of the sentence, indicating that both start at the same rate of uttering the sentence. Under constant values of Laryngeal airway resistance, the fall of MAFR can be related to the noted reduction in ESGP. But LAR is also observed to be variable in the study. Jiang and Tao (2007) relate this fluctuating fall of MAFR to the tissue properties, configuration of theglottis and impedance of the vocal apparatus. It is reported more generally in extrapyramidal syndromes including movement disorders. Vincken et al., (1984) indicates that the severe airflow decrement noted could be due the reason that the movement disorders completely or partially obstruct the upper respiratory airway system.

Jiang et al., (1999) in their study of persons with stage 4 of disease severity, reported that mean ESGP value was higher and mean MAFR was lower in persons with PD, but the MAFR valueswere not statistically different between the groups. They used external very rapidly acting valve to interrupt the sustained phonation of /a/ across increasing intensity levels. And as expected the ESGP and MAFR values increased as the intensity increased.

Frequent impairments in the ability to efficiently make a transition from the plosive production to the subsequent voiced segment (as in /pa/) have been reported in PD (Ludlow and Bassich, 1984; Barlow et al., 2003). For example patients with PD often exhibit loss of air when attempting to phonate. Thus there is a need to assess the physiologic function of how the vocal folds engage in phonation in such patients.

Patients with PD have been reported to show a reduction in total amount of air expended during maximum phonation tasks (Mueller,1971). The reasons for this reduction may be numerous. Failure to generate sufficient aerodynamic energy due to weakness and rigidity of respiratory muscles (Mueller, 1971), variations in airflow resistance caused by abnormal movements of glottis and supraglottic structures leading to abnormal airflow patterns and increased aerodynamic variability as indicated by Vincken et al.,(1984) are some of the research findings. Murdoch et al., (1989) related this to highly unusual chest wall kinematics including marked irregularities in rate and amplitude of individual respiratory excursions. Solomon and Hixon (1993) found that at the initiation of speech breath groups, patients with PD had smaller ribcage volumes than normal. He attributed

this as compensation by the abdominal volume in response to the reduced rib cage compliance.

2.5.3 Laryngeal Airway Resistance

It is the resistance offered by the vocal folds to the airflow at the glottic level. It is the ratio of ESGP to the MAFR (Smitheran and Hixon, 1981), thus making it as a derived parameter. It plays a role in aerodynamic analysis of voice by estimating the efficiency of vocal fold valving. Thus it assists in verifying whether the vocal folds are hyper or hypofunctional(Hoit and Hixon, 1992).

Being a derived parameter, its value depends on the independent values and variations in ESGP and MAFR. Stathopoulos et al., (1993), in their study of normal adults, reported the value of LAR to be 50.43 cm H_2O/L/s in males and 40.62 cm H_2O/L/s in females. Goozee et al. (1998) reported values of 30.58 cm H_2O/L/s in males and 26.4cm H_2O/L/s in females in the age range of 20-30 years. In young Indian adults, the LAR values were found to be 22.56 cm H_2O/L/s in males and 26.53 cm H_2O/L/s in females in the age range of 18-25 years (Gopikishore, Pushpavathi and Sheela, 2012).

Study by Sarr et al., (2009) indicates instability in LAR in persons with PD, while their control subjects exhibit a stable LAR values. The method of the study was already discussed above. The standard deviation of LAR in the PD group was significantly higher. Constancy of laryngeal resistance in an individual indicates his/ability to master the two aerodynamic parameters (ESGP and MAFR).Smitheran and Hixon (1981) reported relatively constant LAR values in their normal control subjects. Smitheran and Hixon measurements were performed to compare laryngeal resistance values in non-invasive technique ofmeasurement with those of invasive procedures. The mean

laryngeal resistance in their patients was 35 cm H_2O/L/s. Blosseret al., (1992) reported similar values with a mean of 38.4 cm H_2O/L/s. In addition laryngeal resistances reflect the laryngeal subject behavior. Evidence comes from the animal studies done by Nasri et al., (1994). They found that increasing the recurrent laryngeal nerve stimulation resulted in rise in LAR, while paralysis of the same nerve induced a significant drop of laryngeal resistance. The assumption is that the instability of laryngeal resistance in OFF DOPA patients reflects a more variable behavior of their larynx and thus a greater fluctuation inESGP and MOAF. Lack of pneumophonic coordination is the probable reason behind this high laryngeal resistance seen.In short, it reflects a lack ofcoordination between the larynx and articulatory organs (Forrest et al. 1989; Lieberman etal., 1992).

The study by Sarr et al., (2009) thus confirms the presence of impairment in the pneumophonic co-ordination, which are evidenced by the fall in IOP and that of MOAF in patients compared with controlsubjects. The alteration of these two parameters leads to a greater instability oflaryngeal resistance as well. However it should be kept in mind that these parameters are closely relatedfunctionally, and that any change in one inevitably has effect on the other two.

2.6 Factors affecting the above discussed aerodynamic measures of voice

Since ESGP, MAFR AND LAR are combined and related measures they are in general affected by few factors. Few of them include age, cognitive ability issues, cooperation, physical ability, vocal fold tension, viscosity; frequency and intensity of phonation, mask seal, and laryngeal surgeries.

Anatomy and physiology of respiratory and laryngeal system vary with age and thus aerodynamic parameters as well vary as a function of age. The sizes of the

anatomical structures are small in children as compared to adults. Hirano, Kurita, and Nakashima, (1981) and Tang and Stathopoulos, (1995) stated that this smaller size limits glottal area and vocal fold vibrational amplitude and henceforth affects the aerodynamics to acoustic energy conversion. Glottal area and vibratory pattern differences are documented between men and women as well (Titze,1989)

Gopikishore, Pushpavathi, and Sheela (2012) obtained aerodynamic measures of ESGP, MAFR, and LAR, using Aeroview 1.4.4 version. The participants included 85 normal adults (54 males and 31 females in the age range of 18 to 40 year). The participants were divided further into two age groups of 18-25 years and 26-40 years. The results revealed no statistically significant effect of gender on any of these aerodynamic measures. However for the ESGP and LAR, age related changes were noted. The older adult group showed more values than the younger adult group. MAFR and LAR were found to be higher in females than males. However, the ESGP values were found to be independent of participant's age. Age-related anatomical and physiological changes in the respiratory and laryngeal system were assumed to be the probable reasons.

As the age increases there could be anatomical degenerations which would influence the mechanical function of the larynx and thus affect the aerodynamic aspects of voice. Kahane (1987) is of the opinion that the age related changes influence the laryngeal valving of airstream during phonation. Sapienza and Dutka (1996) studied 60 women for aerodynamic parameters. Results showed that 20 to 70 years found increased glottal air flow produced during speech for 70 years old group.

Role of intensity in relation to ESGP and MAFR has also been studied. Studies by Jiang et al., (1999), Holmberg E.B, Hilman R. E, Perkell J. S., (1988) and Ishiki, N.,

(1964) have reported increase of ESGP and MAFR with an increase in intensity of sound production.

2.7Phonatory system

It includes the larynx with its intrinsic and extrinsic musculature and is involved in the function of voice production. Larynx is made up of 6 cartilages, 3 of which are paired and 3 are unpaired. Vocal folds are paired muscular structures located within the larynx. It is connected to the angle of the thyroid lamina anteriorly and to the vocal process of the paired arytenoid posteriorly. Vibrations of the vocal folds help in voice production. It also determines the pitch of a person in specific.

Assessment of voice can be subjective/perceptual and objective/instrumental. Perceptual assessment of the voice includes use of appropriate rating scales. Dimensions such as harsh voice, breathy voice, and strained-strangled voice are perceptual impressions of voice quality figured prominently by Darley et al., (1975) in assessing the voice of persons with motor speech disorders.

Objective assessment includes use of instruments to record and analyze the voice. Acoustic analysis of voice with respect to motor speech disorder usually include parameters such as fundamental frequency (F_0) and its variation, standard deviation of F_0 intensity level, Maximum phonation Duration(MPD),jitter and shimmer, signal to noise ratio, and vocal tremor. Other appropriate parameters may also be used.

Most of the perceptual studies of voice in persons with Parkinson's disease indicatemonopitch, monoloudness, reduced stress as common symptoms of speech (Darley et al., 1969a,b, 1975; Ludlow et al., 1984). Hypophonia was considered as a distinctive feature (Darley et al., 1969), but however, it does not seem to be seen in all

patients with PD (Ludlow et al., 1984; Gambao et al., 1997). Voice quality changes were also reported by several others. Breathiness, hoarseness, roughness and tremor were among the most frequent symptoms (Logemann et al., 1973, 1978). However the results seem to be inconsistent, with degree of occurrence of disordered quality being different in different studies.

Certain features have been hypothesized to explain the underlying cause of voice disorder in individuals with IPD (Ho et al., 1999; Sapir et al., 2008). These features include: a reduction in amplitude of neural impulses reaching the muscles of the speech mechanism, that may result in reduced movement and monotonous and soft voice; a problem in sensory feedback mechanism that doesn't allow the person with PD from accurately monitoring his/her physical and vocal output and the individual's difficulty in independently generating (internal cueing/scaling) the right amount of effort to produce adequate loudness.

2.7.1 Fundamental Frequency

Monopitch is a feature of the dysarthria in PD speech. Several studies indicate higher fundamental frequency values than in normal speakers (Canter, 1963; Doyle, Raade, St. Pierre, and Desai, 1995; Kent, Vorperian, Kent, and Duffy, 2003). Few studies reported no differences (Zwirner, Murry, and Woodson, 1991). Certain studies indicate effect of levodopa treatment to the f0 increment (Sanabria et al., 2001) and also the clinical severity of PD (Metter and Hanson, 1986). Patients with PD have been found to have reduced f0 range in connected speech (Kent et al., 2003) and also as the disease severity increases this range has been noted to be reduced (Metter and Hanson, 1986). Few studies demonstrate no strong correlation between F0 variability and perception of

monopitch (Adams, Reynoe-Briscoe, and Hutchinson, 1998; Ludlow and Bassich, 1984). However very few studies has demonstrated a strong correlation (Adams et al, 1998).

Patients with PD are perceived as speaking with decreased loudness. However, acoustic correlates of these perceptual features have been difficult to find. Various authors report no differences between PD and normal on average peak speech intensity and range (Canter, 1963) but several studies document reduced speech intensity in PD (Adams, Haralabous, Dykstra, Abrams and Jog, 2005: Fox and Ramig, 1997) The type of speech task can have effects on speech intensity. For example, Moon, Adams, and Jog, (2006) found that subjects with PD had greater reduction in speech intensity for conversational speech.

2.7.2 Maximum Phonation Duration

Maximum phonation Duration(MPD) is the maximum length of time a person can phonate a vowel after a maximal inspiration. Mean value of 15.7 seconds has been reported in normal adults by Jayakumar, T., and Savithri, S.R, (2012). Studies with respect to MPD in PD have been inconsistent. Some studies have indicated reduced MPD's (Boshes, 1966; Yuceturk, Yilmaz, Egrilmez, and Karaca, 2002). While others have suggested MPD values similar to those of age equivalent normal subjects (Fox and Ramig, 1997; Ho et al, 2001). Previous reports suggest that MPD values are highly variable and influenced by testing procedures and the amount of practice the subjects received (Kent, Kent &Rosenbek, 1987). However it gives a good estimate of the coordination between respiratory and phonatory system

2.7.3 Jitter

Jitter is the term used to define variability occurring in time, wherein successive glottal periods differ from cycle to cycle.This acoustic measure reflects cycle to cycle variation in the duration.Fullness or richness is perceived in a normal voice quality when there is certain amount of this variability. But too much of a jitter results in - perception of a noisy voice quality.Patients with PD have been found to have significantly higher and more variable (across patients) jitter values than normal speakers (Gamboa et al, 1997; Kent et al., 2003).Higher values of jitter in persons with PD as compared with normal subjects have also been reported by Rahn, A et al., (2007).The authors suggest that the abnormal vocal fold rigidity and vocal fold stiffness, which characterized the persons with PD, could be the probable reasons.

Zwirner et al., (1991) studied certain acoustic parameters of voice in three neuropathological groups including PD and compared it with normal subjects. They found that neuropathologic voices show a considerably higher variability of SD F0, jitter, and shimmer compared to normal control voices. This observation is consistent with studies by other investigators. Ludlow et al., (1986) and Ramig et al., (1998) found that the Standard Deviation (SD) of jitter of all neurologic patients were twice that of normal voices. Also, certain studies indicate no significant relationships between acoustic parameters, degree of dysphonia, and level of disease severity for the PD patients (Metter and Hanson, 1986). Also, the variability of speech features in PD in clinical as well acoustical measures noted by these authors was independent of the severity of the disease.

2.7.4 Dysphonia Severity Index

Several studies have been done to find the correlation between perceptual and instrumental assessment procedures in voice analysis. Most of them investigated the quantitative correlation between isolated acoustic variables with the perceptual judgment (Hillenbrand, Cleveland, & Erickson, 1994; Kreiman et al., 1994; Crevier-Buchman, 1998). Studies have been done to investigate the relationship between combinations of acoustic variables and the perceptual ratings as well (Eskenazi, Childers,& Hicks, 1990; Wolfe, Fitch, & Cornell, 1995). However, Wolfe et al. (1995) indicated that none of the acoustic variables neither their combinations were strongly correlated with the dysphonia ratings and prediction of dysphonia.

Heylen et al., 1998 suggests that multivariate techniques prove to be useful in voice research. Voice Range Profile Index (VRPI) is one such example. The voice range profile (VRP) uses a two-dimensional representation of frequency and intensity that cannot be trivially expressed by a single value. Due to its limitation in clinical use as there was a need to interpret the overall shape to assess the performance of a patient, VRPI was developed - which still seems to be not adequate enough in voice assessment. Piccirillo, Painter, Fuller, Haiduk and Fredrickson (1998) constructed an index based on voice range profile and aerodynamic measurements. The authors state that their index is preferentially a classification tool rather than a predictive or treatment effectiveness index. The large variability of most characteristics is probably one of the major reasons why most measurements fail to quantify overall voice quality(Van de Heyning et al., 1996). For example, suppose the range of normative for MPD is 6.7 to 37 s, which

indicates that a person performs poorer than normal only if his MPD is below 6.7s which is very low indeed.

Thus the need to construct a multidimensional measure that reflects the overall vocalquality based on an integration of voice-range profile, aerodynamic, and acoustic measurements led to the finding of an index called the Dysphonia severity Index (DSI). It isan objective and quantitative correlate of the perceived vocal quality (Wuytset. al., 2000). The DSI is based on the weighted combination of selected set of voice measurements such as: highest frequency (F_{hi}in Hz), lowest intensity (I-Low in dB), maximum phonation time (MPT in seconds), and jitter (%).Wuyts et al., (2000) indicates a normal range of 0 to = +5, while a negative value indicates dysphonia. Thus if the DSI value is more negative it is indicative of severe voice problem and more positive better the voice quality. Ethnic and geographical variation has been noted with a reduced DSI values (mean of 3.47) in Indian population (Jayakumar and Savithri,2012) when compared to the European population (mean of 5.0) as reported by Wuyts et al., (2000). This measure is recently being used as objective voice measure in various disorders of voice. As most of the parameters of voice are affected in persons with PD, we predict it would be better measure in evaluating the quality of voice in Parkinsonian speech.

CHAPTER III

METHOD

The aim of the current study was to obtain a few aerodynamic and acoustic parameters of voice of patients with Parkinson's disease and compare the measures with mean age- matched control group. In order to obtain the measures, the following methodology was adopted.

3.1 Participants

Two group of participants were considered for this study. The first group comprised of ten individuals with Idiopathic Parkinson's disease (IPD). The age range was 65-80 years (mean age=73.3) and they were considered as the clinical group. The second group comprised of 15 typically aging individuals with no voice complaints or laryngeal disorders, in the age range of 60-80 (mean age=70) and they would considered as the control group. Table 2 shows the details of the persons with Parkinson's disease.

3.2 Selection criteria

a. *Selection criteria for participants in the clinical group*

- The participants should be clinically diagnosed as having Idiopathic Parkinson's disease by a neurologist
- Linguistic, cognitive and hearing abilities should be adequate enough so as to understand the procedural instructions.

b. ***Selection criteria for participants in the control group***

- All the individuals should be in the ages of 60 and above
- They should not be having any linguistic, cognitive and speech deficits
- They should not be having any neurological and psychological problems

Table 2
Details of the persons with Parkinson's disease and control groups

Sl.No	Participants	Age/ gender	Stage of the disease	Onset of the disease
1	PD1	68 years/Male	EARLY	Since 5 years
2	PD2	73 years/Male	EARLY	Since 1 year
3	PD3	75 years/Male	EARLY	Since 2 years
4	PD4	70 years/Male	EARLY	Since 2 years
5	PD5	76 years/Male	EARLY	Since 2 years
6	PD6	79 years/Male	MIDDLE	Since 6 years
7	PD7	65 years/Male	MIDDLE	Since 6 years
8	PD8	80 years/Male	MIDDLE	Since 7 years
9	Control group (15 number)	Mean age=70 years	-	-

3.3 Ethical standards

Participants were thoroughly informed about the purpose and procedure of study. An informed consent was obtained from each of the individuals using AIISH ethical guidelines for bio-behavioral research.

3.4Materials

The materials used for the study include:

a) General information sheet

b) Checklist to identify the stage of IPD based on speech, swallowing and motor symptoms by Amulya and Swapna 2012)

The checklist has a variety of features generally observed in IPD and it categorizes the stage of PD into early, middle and advanced based upon the presence of salient features delineated. These features helps in differentiating the stages. The early stage mentioned in the checklist is inclusive of stage 1 and 2 of HoehnandYahr's motor staging, while the middle stage is inclusive of stage 3 and 4 and advanced stage is equivalent to the stage 5.

3.5 Instrumentation

The Aeroview 1.5 version (Glottal Enterprises Inc, Syracuse, NY) was used to measure ESGP, MAFR LAR and LAC. The Aeroview is a computer based portable system consisting of a circumferentially vented (CV) pneumotachograph mask coupled to PT-25B air pressure transducer with an intraoral tube attached to it and PT-2E wideband model airflow transducer. Low pass filtering for airflow was set at 500 Hz as per the manufacturer recommendation. Window length of 5.12 seconds was selected for recording as it was the maximum limit.Lingwaves software (WEVOSYS, German) was used to derive the Dysphonia severity Index (DSI) values. This instrument has facility to measure the acoustic signal from sound pressure level meter (SLM). Hence the acoustic parameters can be measure accurately including intensity measure with the sampling rate of 44 KHz and 16 bit rate. Phonotogram VDC was used to estimate the DSI which is the sub module present in Lingwaves software.

3.6 Procedure

All the recordings for the participants with PD were done at the time, where the effect of medication was least or in the OFF DOPA condition. OFF DOPA condition would be the time wherein the person may exhibit certain Parkinsonian symptoms like

resting tremor that would suggest that the time for the next medication was near. For most of the patients half an hour before their second medication was noted to be the OFF DOPA condition. One participant didn't report or observe this ON-OFF phenomenon and thus all the recordings were done uniformly before half an hour prior to their second medication.

Before each recording in the aeroview instrument the surface of the pneumotachograph mask and intraoral tube was cleaned with antiseptic liquid. The instruments were calibrated as per the instructions in the manual and checked for proper measurements.

3.6.1 Aerodynamic measures

Before each recording in the aeroview instrument the surface of the pneumotachograph mask and intraoral tube was cleaned with antiseptic liquid. The instruments were calibrated as per the instructions in the manual and checked for proper measurements.

The participants were made to sit straight and comfortably on a chair. They were given with the instructions about the instrument and the recording procedure in detail. The examiner also demonstrated the way of holding the mask and the rate of syllable production (2-4 syllables/sec). For the clinical group, the examiner held the pnematachograph mask firmly without interfering the comfort of the participants, so as to avoid leakage of air during recording. This was done owing to the difficulty in holding the mask due to the tremors of hand. For the control groupthe usual procedure was followed. The participants were asked to place the intraoral tube within the oral cavity (with closed lips and without the tongue being occluded). They were made to utter

syllable /pa/ 5 times at comfortable pitch and loudness. Two practice trails were done prior to the actual recording. Three recordings were done for each participant and the data was stored for analysis.

Rothenberg (1973) suggests the use of /pa/ syllable, because the distance between the fundamental frequency and first formant of the vowel facilitates the inverse filtering procedure. When F0 and F1 are closely spaced, it can be difficult to adequately filter out the effects of vocal tract resonances in order to obtain a glottal flow waveform. Hence the /pa/ syllable was selected for obtaining the mentioned aerodynamic parameters.

3.6.2 Acoustic measures

Lingwaves software - Phonotogram VDC was used to estimate the DSI was used for DSI calculation. Theparameters required were recorded as follows:

a. Maximum phonation time (MPD)

The participants were instructed to inhale deeply, and produce and sustain vowel |a| for as long as possible at a comfortable pitch and loudness. The phonation will be recorded using Lingwaves software. Phonation time was measured as the time duration between the onset and offset of regular waveform. The longest of the three measured MPTs was be used for further analysis.

b. Frequency and Intensity (High-F0 – Hz & low-In -dBSPL)

The SLMwas placed at a distance of 10 cm with 30 degree angle from the mouth of the participants. Participants were instructed to phonate vowel /a/ as softly as possible at a comfortable pitch. After this, they were instructed to phonate vowel /a/, starting at a comfortable pitch going up to the highest and down to the lowest pitch. The clinician prompted and modelled the subject to achieve the highest possible pitch. The highest F_0 and the lowest intensity wasnoted for further analysis.

c. Jitter (%)

Participants were asked to sustain phonation of the vowel /a/ at a comfortable pitch and loudness for 5 seconds and it was recorded thrice. Percent jitter was calculated for a sample of 4 second duration. The first and last half-second of the sample was eliminated for the analysis.The lowest of the three calculations was used for DSI calculation.

The software calculates the DSI using the following algorithm:

DSI = 0.13(MPT) + 0.0053 (High-F0) – 0.026(low-In) – 1.18(jitter %) + 12.4

3.7 Statistical analysis

The obtained aerodynamic and acoustic parameters were analyzed using SPSS 17. For the reliability measures Cronbach's alpha was used. To compare the PD and Control group MANOVA was used. Mann whirney U test was used to compare the stages within PD and each stage with the control.

CHAPTER IV

RESULTS AND DISCUSSION

The present study was aimed to obtain few aerodynamic and acoustic parameters of voice in persons with Parkinson's disease and to compare them with mean age matched control group. Total of eight participants in the clinical group (PD) and 15 participants in the control group were included in the present study. The participants in the PD group were categorized into early and middle stages using the checklist developed by Amulya&Swapna (2012).

The aerodynamic and acoustic data were obtained using Aeroview 1.5 version and Ling wave (WEVOSYS) software as per the procedure described in the method. The data was analyzed using SPSS 17 version with the following statistical methods.

a) Cronbach's alpha to determine the reliability between the trails
b) Descriptive statistics (Mean & SD) of aerodynamic and acoustic parameters
c) MANOVA was used to compare the PD and Control group
d) Mann-Whitney 'U' test used to compare the two stages of PD group (early Vs middle), early PD Vs control group and middle Vs control group.

The results of the study was presented and discussed under following headings

1. Reliability measures
2. Kolmogorov –Smirnov test for normality
3. Comparison of mean and SD of aerodynamic parameters between PD and the control group
4. Comparison of aerodynamic voice parameters between the stages of PD
5. Comparison of acoustic measures of voice between PD and the control group

6. Comparison of acoustic measures of voice between the stages of PD

4.1 Reliability of parameters

The inter-trial reliability for aerodynamic parameters were estimated using Cronbach's alpha test. Cronbach's Alpha values was greater than 0.9 for all the aerodynamic parameters, which indicates good reliability between the trails. Table 3 shows the inter -trial Cronbach's Alpha values for aerodynamic parameters.

Table 3
Inter- trial Cronbach's Alpha values for aerodynamic parameters.

Parameters	Cronbach's Alpha Value
ESGP	0.942
MAFR	0.945
LAR	0.953
LAC	0.955
SPL	0.984
Pitch	0.955

4.2 Kolmogorov –Smirnov test for normality

Kolmogorov–Smirnov test was performed to check the normality of the data. The test was performed separately for PD group and the control group and result showed that both the group data are from the normal distribution. Hence the parametric test was done for the comparison between the groups.

4.3 Comparison of aerodynamic parameters between the PD and control group

a. PD group (both stages) Vs Control group

The mean and SD for the aerodynamic parameters were obtained using descriptive statistics. MANOVA was done to compare both the groups. Results showed that only SPL showed significant difference between PD and the control group (figure 1). However the descriptive data showed that increased, MAFR, LAC, and Pitch in PD than

control group. The ESGP, SPL and LAC was reduced in PD than control group. Table 4 shows the mean, SD, F and *p value* of the MANOVA result.

Table 4
Mean, standard deviation, (SD), F and /p/ values of aerodynamic parameters for PD and the control group

	PD		**Control**		**MANOVA results**	
	Mean	**SD**	**Mean**	**SD**	**F**	***/p/***
ESGP	6.47	1.41	7.47	1.54	2.325	0.142
MAFR	269	115	258	128	.048	0.829
LAR	0.029	0.0138	0.041	0.020	1.105	0.305
LAC	39.90	25.35	35.59	18.24	0.223	0.642
SPL	69.5	10.4	75.4	3.2	4.178	0.054*
Pitch	141	32	136	21	0.185	0.671

*P ≤0.05

Overall reduction in ESGP and increased MAFR may be due to the incomplete closure of the vocal folds in persons with PD. Mean LAR is noted to be reduced in the PD group. Vocal fold bowing is a feature mostly associated with the disease which may be due to muscle weakness and rigidity and thus causing increased MAFR. The increased MAFR is indicative of the reduced LAR. However the vocal fold bowing and muscle weakness and rigidity has to be confirmed using EGG and EMG respectively.The reduced ESGP value in PD is in concordance with the most of the studies (Marquardt, 1973; Mueller, 1971, Solomon and Hixon, 1993; Sarr et al., 2009) conducted in this area. The increased MAFR found in this study is contradictory to most of the studies which indicate a reduced MAFR (Mueller, 1971; Solomon and Hixon, 1993; Sarr et al., 2009). Reduced SPL values noted in the study is in support for the hypophonia which is a an important feature reported to be observed in PD

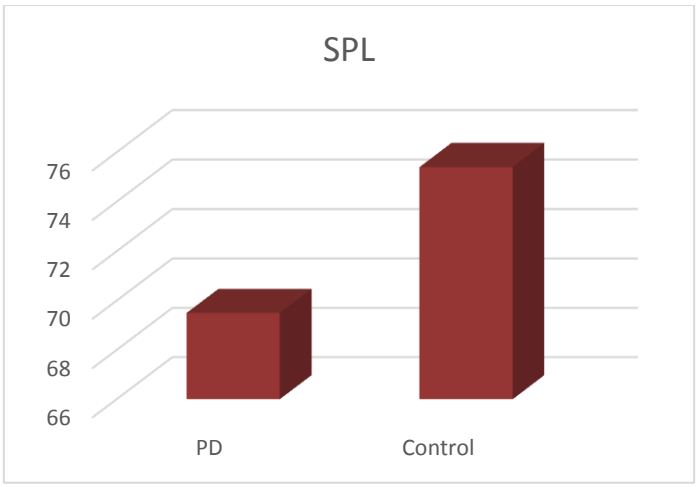

Figure 1: *Comparison of mean SPL between PD and control group.*

b. Early stage of PD Vs control group

Mean and SD were obtained for the early stage PD and the control group using descriptive statistics. They were subjected to Mann-whitney test to test for any significant differences among the parameters. Table 5 shows the mean, SD values and /z/ values of Mann-Whitney U test.

Table 5
Mean Standard Deviation and /z/ value for early stage PD and Control group

Parameter	**Early PD**		**Control**		
	Mean	**SD**	**Mean**	**SD**	**|Z|**
ESGP	6.68	1.32	7.47	1.54	0.830
MAFR	279	81	258	128	0.218
LAR	0.027	0.130	0.041	0.030	0.742
LAC	38.30	25.09	35.59	18.24	0.131
SPL	69	10	75.4	3.2	0.830
Pitch	121	15	136	21	1.441

Note: P ≤0.05

The mean values for ESGP, MAFR, LAR for the early stage are 6.68 cm H_20, 279 mL/s and 0.027 cm H_20/mL/s respectively and for the control group it was 7.47 cm H_20, 258 /mL/s and 0.030 cm H_2O/mL/s respectively. However Mann whitney test revealed no significant differences of the parameters between the group.

c. Middle stage of PD VsControl group

The mean and standard deviation values for the aerodynamic parameters were obtained using descriptive statistics and Mann-whitney test was used to test if any significant differences existed between the parameter among the middle stage PD and Control group. Table 5 shows the mean, SD values and /z/ values of Mann-whitney U test.

Table 6
Mean, Standard deviation, and /z/ values for Middle PD and control group
.

Parameter	**Middle PD**		**Control**		**Mann-Whitney U test**
	Mean	**SD**	**Mean**	**SD**	**|Z|**
ESGP	6.12	1.79	7.47	1.54	1.126
MAFR	253	181	258	128	0.059
LAR	0.032	0.016	0.041	0.030	0.415
LAC	42.57	31.18	35.59	18.24	0.415
SPL	70	13	75.4	3.2	0.299
Pitch	173	27	136	21	**2.074***

Note: *P ≤0.05

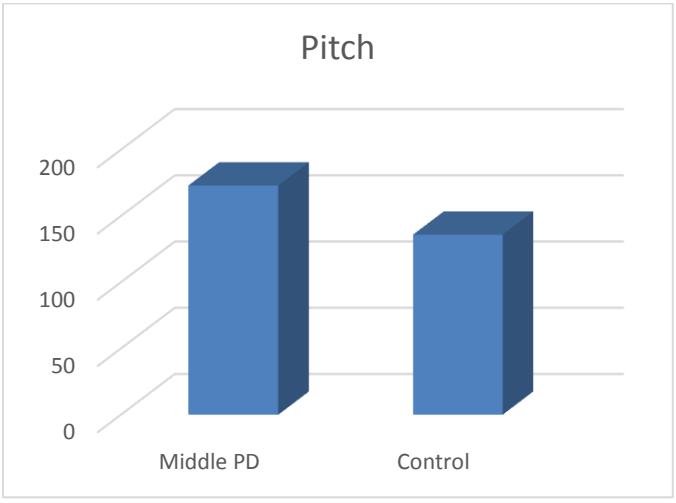

Figure 2: *Comparison of mean pitch between middle stage PD and control group.*

The mean values for ESGP, MAFR, LAR for the middle stage are 6.12 cm H_20, 253 mL/s and 0.032 cm H_20/mL/s respectively and for the control group it was 7.47 cm H_20, 258 /mL/s and 0.030 cm H_2O/mL/s respectively. Thus the mean values of ESGP and MAFR was found to be lower in the middle PD as compared to the control group, while the LAR was found to be higher for PD group. However Mann Whitney test revealed no significant difference of the parameters between both the groups, except for the pitch which was higher in middle PD. vocal fold rigidity, stiffness and bowing of the PD may be the possible reason for high pitch. Abnormal vocal fold rigidity, stiffness and bowing have been attributed to co contraction of opposing thyroarytenoid and cricothyroid muscles Increased fundamental frequency has been reported in PD by Canter, 1963; Doyle, Raade, St. Pierre, and Desai, 1995; Kent, Vorperian, Kent, and Duffy, 2003.

4.4 Comparison of aerodynamic parameters within the stages PD

The mean and standard deviation values for the aerodynamic parameters were obtained using descriptive statistics and Mann-Whitney U test was used to test if any significant differences existed between the parameters across both the early and the middle stages of PD and the Control group. The values are depicted in the table 7.

Table 7
Mean, SD and /z/ values for Early and Middle PD

Parameter	Early stage		Middle stage		
	Mean	**SD**	**Mean**	**SD**	**|Z|**
ESGP	6.68	1.32	6.12	1.79	0.149
MAFR	279	81	253	181	0.447
LAR	0.027	0.130	0.032	0.016	0.149
LAC	38.30	25.09	42.57	31.18	0.447
SPL	69	10	70	13	0.300
Pitch	121	15	173	27	1.978

The mean values for ESGP, MAFR, and LAR for the early PD are 6.68 cm H_20, 279 mL/s and 0.027 cm H20/mL/s respectively and for the middle PD it was 6.12 cm H20, 253 /mL/s and 0.032 cm H_2O/mL/s respectively. However Mann Whitney test revealed no significant differences of the parameters between the groups. However, if hypo-adduction of VF is presumed to be the reason, the middle stage PD is expected to have increased MAFR values than early PD. But a higher SD value of MAFR in the middle PD indicates higher variability of this parameter within that group. If Minimum and maximum values of MAFR are considered it has been found that the maximum MAFR value of 460 mL/s is noted in one of the participant in the Middle PD (PD8).

4.5 Comparison of Mean and SD of acoustic measures of voice between the PD and control group

a. Between the PD group (both stages) and control group

The mean and SD for the acoustic parameters were obtained using descriptive statistics and then MANOVA was done to know if there exists any significant differences among parameters between the both groups. The mean, SD, F and *p value* are depicted in the table 8.

Table 8
Mean standard deviation (SD), F and /p/ values of acoustic parameters for PD and the control group

Parameters	**PD**		**Control**			
	Mean	**SD**	**Mean**	**SD**	**F-value**	***p-value***
MPD (sec)	8.6	3.6	12.6	3.3	7.297	**0.013***
High-F_0 (Hz)	256	54	316	81	3.449	0.077
Low –In dBSPL)	48.7	3.8	47.7	4.3	0.298	0.591
Jitter (%)	0.295	0.125	0.218	0.076	3.326	0.082
DSI	1.838	1.470	3.053	1.387	3.84	0.063

*P ≤0.05

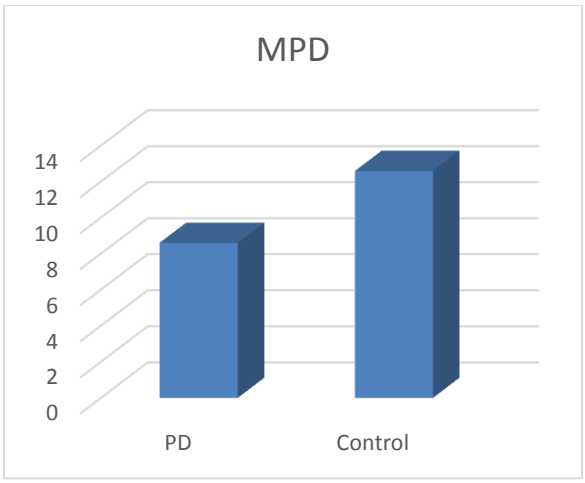

Figure 3: *Comparison of mean MPD between PD and control*

The mean values of MPD, High F0, Low Intensity, Jitter and DSI for PD group has been found to be 8.6 s, 256 Hz, 48.7 dBSPL, 0.295 % and 1.838 respectively, while for the control group it was found to be 12.6 s, 316 Hz, 47.7 dBPL, 0.076% and 3.053 respectively. All of the measures were found to be lower in the PD group except for the lowest intensity and jitter value which had a mean value higher than that of the control group. MANOVA was carried to test if there existed any significant differences among the measures and it indicated a reduced value of MPD in the PD group. All the other measures did not reveal a statistically significant difference. The reduced MPD could be due to the loss of air available while phonating. Supporting evidences for reduced MPD comes from Boshes (1966), Canter (1965), Yuceturk et al., (2002). There could be respiratory capacity inadequacies due to muscle weakness and rigidity leading to the reduced availability of expiratory air for phonating as long as possible.

b. Early stage Vs control group

Mean and SD were obtained for the early stage PD and the control group using descriptive statistics. They were subjected to Mann-Whitney test to test for any

significant differences among the parameters. The mean, SD values and /z/ values are presented in table 9.

Table 9
Mean, Standard Deviation, and /z/ value for the acoustic measures of voice for early stage PD and Control group

Parameter	Early PD		Control		
	Mean	SD	Mean	SD	|Z|
MPD (sec)	10.5	2.9	12.6	3.3	1.267
High-F_0 (Hz)	275	42	316	81	1.049
Low -In (dBSPL)	48.6	2.9	47.7	4.3	0.699
Jitter (%)	0.280	0.126	0.218	0.0763	0.831
DSI	2.221	1.116	3.053	1.387	1.004

The mean value of MPD and highest F_0 were lower in the early stage PD as compared to the control group while the jitter was found to be higher. Mean DSI value was also found to be lower. However there was no statistically significant difference between the mean values shown above.

c. Middle PD Vs control group

Mean and SD were obtained for the middle stage PD and the control group using descriptive statistics. They were subjected to Mann-Whitney U test to test for any significant differences among the parameters. The mean, SD values and /z/ values are depicted in table 10.

Table 10
Mean, Standard Deviation and /z/ value for middle stage PD and control group for acoustic measures

Parameter	**Middle PDControl**				
	Mean	**SD**	**Mean**	**SD**	**|Z|**
MPD (sec)	**5.4**	2.1	12.6	3.3	**2.618***
High-F_0 (Hz)	**223**	63	316	81	**2.075***
Low -In (dBSPL)	**48.9**	5.9	47.7	4.3	0.416
Jitter (%)	**0.320**	0.143	0.218	0.0763	1.118
DSI	**1.201**	2.025	3.053	1.387	1.719

*P ≤0.05

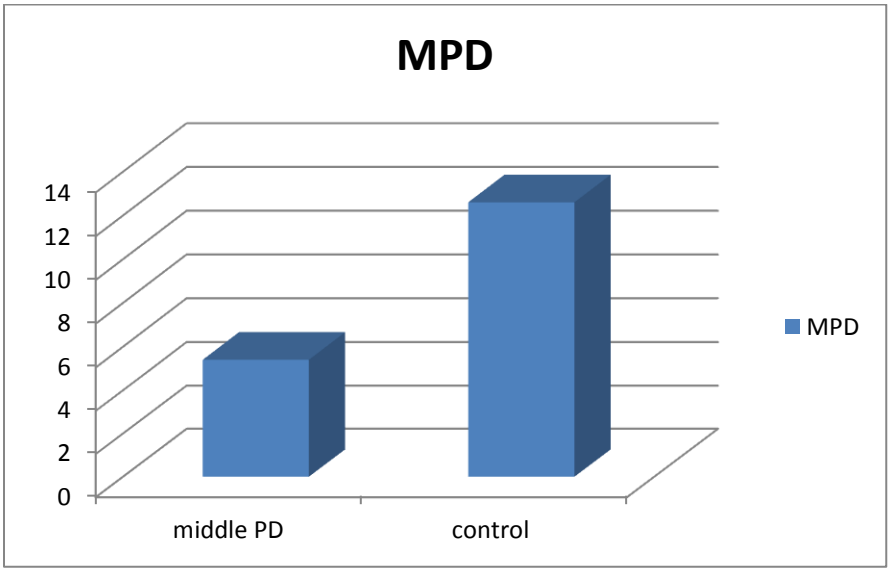

Figure 4: *Comparison of mean MPD between middle PD and control group*

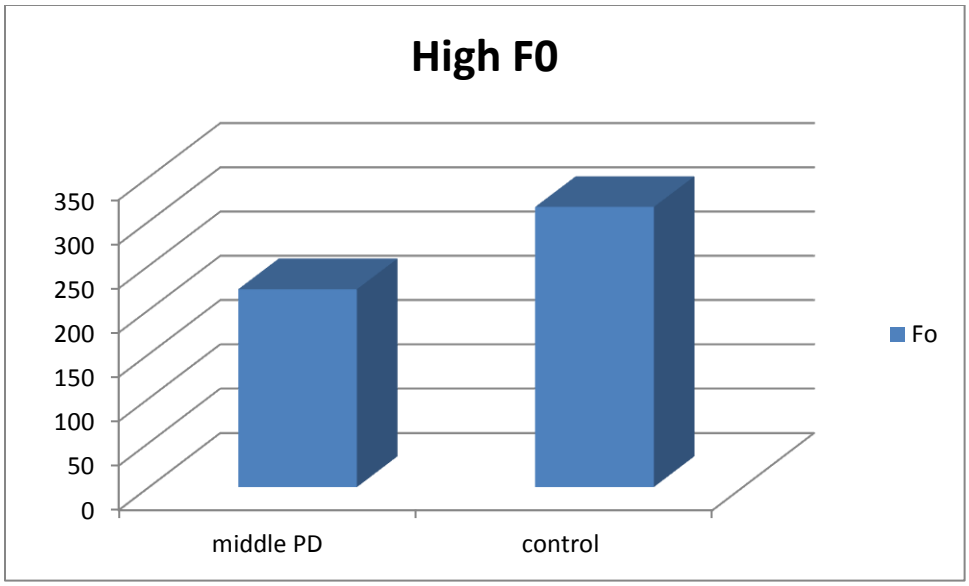

Figure 5: *Comparison of mean highest F0 between middle PD and control group*

The mean values of MPD, high F_0 were found to be lower in the PD group as compared to the control group. The lowest intensity and jitter values were higher for the PD group. Mann-Whitney test revealed a statistically significant difference between the MPD and high F0 values between both the groups. The reduced MPD could be attributed to the strooped posture observed in all the three participants of the PD group (PD6, PD7 and PD8). The abnormal posture may have led to the reduced respiratory capacity in these participants of the middle stage of disease severity. The reduced ability to increase or phonate at the maximum pitch level possible may be attributed to the rigidity that may be present in the vocalis muscle.This might have led to their inability to alter the visco-elastic properties of the vocal folds. Thus the ability to increase the pitch may be affected. DSI values were reduced in the middle PD group indicating reduced voice quality in the PD group than the control group. Only PD8 had a negative DSI value (-0.983) indicating more disturbed voice quality as compared to others.

4.6 Comparison of early Vs middle stage of PD

Mean and SD were obtained for the early stage PD and the control group using descriptive statistics. They were subjected to Mann-Whitney U test to test for any significant differences among the parameters. The mean, SD values and /z/ values are depicted in table 11.

Table 11
Mean, Standard Deviation, and /z/ value for early PD and middle PD group for acoustic measures

Parameter	**Early PD**		**Middle PD**		
	Mean	**SD**	**Mean**	**SD**	**|Z|**
MPD (sec)	10.5	2.9	5.4	2.1	1.938
High-F_0 (Hz)	275	42	223	63	1.342
Low -In (dBSPL)	48.6	2.9	48.9	5.9	0.149
Jitter (%)	0.280	0.126	0.320	0.143	0.600
DSI	2.221	1.116	1.201	2.025	0.745

The mean values of MPD, high Fo were found to be lower in the middle PD group as compared to early PD group. Jitter values were higher in the middle PD indicating more variation. However there were no statistically significant differences among the values.

CHAPTER V

SUMMARY AND CONCLUSION

The present study was aimed at obtaining few aerodynamic parameters and acoustic measures of voice in persons with Parkinson's disease and compares it with those mean age matched individuals.A total of 8 participants constituted the clinical group (PD). They were categorized into early and middle stages using a Checklist for identifying the stage of Idiopathic PD based on speech, swallowing and motor symptoms (Amulya and Swapna, 2013). There were 5 participants in the early stage and 3 participants in the middle stage. 15 mean age matched individuals with no voice or laryngeal and respiratory disorder served the control group.

The ESGP, MAFR, LAR, LAC, SPL, Pitch were the aerodynamic parameters and MPD, high-F0, low-intensity, jitter were the acoustic measures of voice were obtained. Dysphonia Severity Index (DSI) was derived from the weighted combination of the acoustic measures obtained. It is an objective measure of voice quality. Aeroview 1.5 version (Glottal Enterprises Inc, Syracuse, NY)was used to obtain the aerodynamic parameters and Lingwave software (WEVOSYS, German) was used to obtain the acoustic measures of voice from which DSI was derived by using an algorithm.

The data obtained from both the groups were subjected to data analysis using SPSS 17. In general, among the aerodynamic parameters obtained, there was a decrease of ESGP, LAR, SPL in the PD group with SPL showing a significance. The SPL was reduced in the PD group. However between the early and the control group there was no significant difference for SPL. Between the middle stage of PD and control group, pitch

showed significance with middle PD exhibiting higher mean value. Among the acoustic measures MPD and high F_0 showed a significant difference. However when early PD and control group were compared, there was no significant difference of any of the acoustic measures studied. MPD was significantly reduced at the middle stage of the disease (mean = 5.4 s). Thus MPD is very much associated with the stage of the disease. The ability to produce high pitch was also significantly reduced in the middle stage of the disease. They were unable to produce highest pitch possible as efficiently as the control group could produce. The values of DSI were lower in the PD group indicating poorer voice quality as compared to the control. Among the 3 participants in the middle stage of the disease, PD8 exhibited negative value of DSI indicating severe voice problem/dysphonia. It also indicates that as the disease progresses the voice quality may be severely impaired.

To conclude it has been found that aerodynamic and acoustic measures of voice is affected in persons with PD, even though only few of them achieved statistical significance. It has been observed that as the disease progresses the aerodynamic and acoustic parameters of voice also worsen.

5.1 Clinical implications:

a) Aerodynamic and acoustic parameters can be useful to supplement the identification of Stages of the PD.

b) Efficacy of treatment can be documented and thus in pre-post treatment comparison can be made.

5.2 Limitations of the study:

a) Sample size is not adequate enough to generalize the findings to the PD population

b) Perceptual evaluations could have been incorporated to increase the reliability of objective measures obtained.

5.3 Future Directions:

a) Since this is a preliminary attempt to determine the aerodynamic and acoustic measures of voice in patients with Parkinson's Disease in the Indian population, only a limited sample was undertaken and hence further attempts should be made to test for the same parameters on a larger sample to obtain more reliable outcomes.

b) Attempts can be made to study the correlation between the respiratory capacities and the aerodynamic measures of voice.

References

Adams, S.G., Haralabous, O., Dykstra, A., Abrams, K., & Jog, M. S. (2005). Effects of multi-talker background noise on the intensity of spoken sentences in Parkinson's disease. *Canadian Acoustics*, *Vol 33,* 94-95.

Adams, S.G., Reyno-Briscoe, K., & Hutchinson, L. (1998). Acoustic correlates of monotone speech in Parkinson's disease. *Canadian Acoustics, Vol 26*, 86-87.

Amulya, P, R. & Swapna, N. (2012). Speech Rhythm in Reading in Persons with Parkinson's Disease. Unpublished Dissertation submitted to University of Mysore.

Baken R. J. & Orlikoff R. F. (2000).*Clinical measurement of speech and voice* (2nd ed.), Singular Thomson Learning: San Diego

Barlow, S.M.,&Abbs, J.H. (1983). Force transducers for the evaluation of labial, lingual, and mandibular motor impairments. *Journal of Speech and Hearing Research, Vol 26*, 616-621.

Blosser S, Wigley F, M., Wise, R, A. (1992).Increase in translaryngeal resistance during phonation inrheumatoid arthritis.*Chest*, *Vol 102, 2,* 387-90.

Braak, H., & Braak, E. (2000).Pathoanatomy of Parkinson's disease.*Journal of Neurosciences,247*, 3-10.

Braak, H., Del Tredici, K., Rüb, U., Vos RAI de., Jansen Steur, E. N. H., & Braak, E. (2003a). Staging of brain pathology related to sporadic Parkinson's disease. *Neurology of Aging, Vol 24,* 197- 211.

Bielamowicz, S., Berke, G. S., & Gerratt, B. R. (1995).A comparison of type I thyroplasty and arytenoids adduction.*Journal of Voice, Vol 9,4,* 466 – 472.

Bless, D. M., Glaze, L. E., Biever – Lowry, D., Campos, G., & Peppard, R. C. (1993). In I. R. Titze (Ed.), *Stroboscopic, acoustic, aerodynamic and perceptual attributed of voice production in normal speaking adults.* 121 - 134. Iowa: National Center for voice and speech

Canter, G. J. (1963). Speech characteristics of patients with Parkinson's disease: Intensity, pitch and duration. *Journal of Speech and Hearing Disorders*, *Vol30*, 44-49

Canter, G. J. (1965a). Speech characteristics of patients with Parkinson's disease: Physiological support for speech. *Journal of Speech and Hearing Disorders, Vol 30*, 44-49.

Crevier-Buchman L. (2007). Modélisation du fonctionnement laryngé. In Auzou P, Monnoury-RollandV, Pinto S, Özsancak C (eds*), Les dysarthries. Solal Marseille (*pp 91-100).

Cramer, W. (1940). De spaak bij patienten met Parkinsonisme. *Logopaedica Phoniatrica*, *Vol 22*, 17-23.

Darley, F. L., Aronson, A. E., & Brown, J. R. (1975).Motor Speech Disorders. Pheladelphia, PA: W. B. Saunders.

Darley F. L., Aronson A. E., & Brown, J. R. (1969a).Differential diagnostic patterns of dysarthria.*Journal of Speech and Hearing Research*,*Vol12*, 246-269.

Das, S. K., Misra, A. K., Ray, B. K., Hazra, A., Ghosal, M. K., Chaudhuri, A., Roy, T., Banerjee, T. K., & Raut, D. K. (2010). Epidemiology of Parkinson disease in the city of Kolkata, India.*Neurology*, *75, 15*, 1362 – 1369.

Das, S. K., & Sanyal, K. (1996).Neuroepidemiology of major neurological disorders in rural Bengal.*Neurology India, Vol 44,* 47-58.

DeLong, M. R. (1990). Primate models of movement disorders of basal ganglia origin. *Trends in Neurosciences, Vol 13,* 281-285.

Demolin D, Giovanni A, Hassid S, Heim C, Lecuit V, Socquet A (1997). Direct and indirectmeasurements of subglottic pressure.*Proc. Larynx 97*, 69-72.

Del Tredici, K., Rüb, U., Vos RAI de., Bohl, J. R. E., & Braak, H. (2002). Where does Parkinson disease pathology begin in the brain? *Journal of Neuropathology and Experimental Neurology, Vol 61,* 413-426.

Dejonckere, P. H., Remacle, M., Fresnel-Elbaz, E., Woisard, V., Crevier Buchman, L., & Millet, B. (1996). Differentiated perceptual evaluation of pathological voice quality: reliability and correlations with acoustic measurements. *Revue de Laryngology Otology Rhinology, 117,* 219 – 224.

De Pandis, M. F., Starace, A., Stefanelli, F., Maruzzo, P.,Meoli, I., De Simone & Stocchi, F (2002). Modification of respiratory function parameters in patients with severe Parkinson's disease.*Neurological Science*, *Vol 23*, 69-70.

Doyle, P.C., Raade, A. S., St. Pierre, A., & Desai, S. (1995). Fundamental frequency and acoustic variability associated with production of sustained vowels by speakers with hypokinetic dysarthria. *Journal of Medical Speech-Language Pathology, Vol 3*, 41-50.

Duffy, J. F. (2005). Hypokinetic dysarthria. In J. R. Duffy, *Motor speech disorders: Substrates, differential Diagnosis, and management* (2nd ed., pp. 187-215). St. Louis, MO: Elsevier Mosby.

Eskenazi, L., Childers, D. G., & Hicks, D. M. (1990). Acoustic correlates of vocal quality. *Journal of Speech andHearing Research, Vol 33*, 298–306.

Fahn, S. (1986).Parkinson's disease and other basal ganglion disorders. In A. K. Abury, G. M. McKhann, & W. I. McDonald (Eds.), *Diseases of the nervous system: Clinical neurobiology* (pp. 1217-1228). Philadelphia, PA: Ardmore Medical books.

Fahn, S. (1989).The history of Parkinsonism.*Movement Disorders*, *Vol 4*,*1,*2-10.

Fahn, S., & Przedborski, S. (2005).Parkinsonism. In L. P. Rowland (Ed.), *Merritt's Neurology*, (11th ed., pp. 828-846), Philadelphia: Lippincott Williams & Wilkins.

Forrest, K., & Weismer, G. (1995).Dynamic aspects of lower lip movement in Parkinsonian and neurologically normal geriatric speakers' production of stress.*Journal of Speech and Hearing Research, Vol 38*, 260-272.

Fox, C. & Ramig, L. (1997).Vocal sound pressure level and self-perception of speech and voice in men and women with idiopathic Parkinson disease.*American Journal of Speech-Language Pathology, Vol 6,* 85-94.

Gamboa, J., Jimenez-Jimenez, F. J., Nieto, A., Montojo, J., Orti-Pareja, M., Molina, J. A., Garcia-Albea, E., & Cobeta, I. (1997). Acoustic voice analysis in patients with Parkinson's disease treated with dopaminergic drugs. *Journal of Voice, Vol 11*, 314-320.

Gouri Devi, M., Gururaj , G., Satishchandra, P., & Subbakrishna, D.K. (1999). Prevalence of Parkinson's disease in Bangalore.A door to door survey of urban and rural population.*Neurology India , Vol 47*, 73-75.

Goozee, J. V., Murdoch, B. E., Theodoros, D. J., & Thompson, E.C. (1998).The effects of age and gender on laryngeal aerodynamics.*Internal Journal of Language and Communication Disorders, Vol 33*, 221-238.

Gopikishore, P. Pushpavathi, M. and Sheela, S. (2012).Laryngeal Aerodynamic Measures in Normal Adults.*Journal of All India Institute of Speech and Hearing, Vol 31,* 56 -63.

Heylen, L., Wuyts, F. L., Mertens, F., De Bodt, M., Pattyn, J., Croux, C., & Van de Heyning, P. (1998).Evaluation of the vocal performance of children

using a voice range profile index.*Journal of Speech, Language,and Hearing Research, Vol 41*, 232–238.

Hillenbrand, J., Cleveland, R. A., & Erickson, R. L. (1994). Acoustic correlates of breathy vocal quality. *Journal of Speech and Hearing Research, Vol 37*, 769–778.

Ho, A. K., Iansek, R., & Bradshaw, J. L. (2001). Motor instability in parkinsonian speech intensity.*Neuropsychiatry, Neuropsychology, and Behavioural Neurology, Vol 14,* 109-116.

Hoehn, M., & Yahr, M. (1967). Parkinsonism: Onset, progression and mortality. *Neurology, 17*, 427-442.

Hoit, J.D., & Hixon, T. (1992).Age and Laryngeal airway resistance during vowel production in women.*Journal of Speech Language and Hearing Research, Vol 35,* 309-313.

Hirano, M., Kurita, S., & Nakashima, T. (1981).The structure of the vocal folds.In K. N. Stevens & M. Hirano (Eds.), *Vocal fold physiology.*(pp: 33- 43). Tokyo: University of Tokyo Press

Hoit, J. D., & Hixon, T. J. (1992).Age and laryngeal airway resistance during vowel production in women.*Journal of Speech Language and Hearing Research*, *Vol 35,* 309-313.

Holmberg EB, Hilman RE, Perkell JS (1988). Glottal airflow andtransglottal air pressure measurements for male and female speakers in soft, normal, and loud voice.*Journal of Acoustic Soc iety of America, Vol 84.* 511-529.

Holmberg, E. B., Hillman, R. E., & Perkell, J. S. (1989).Glottal airflow and transglottal air pressure measurements for male and female speakers in low, normal, and high pitch.*Journal of Voice, Vol 3*, *4,* 294-305.

Huber, J. E., Stathopolous, E. T., Ramig, L. O., & Lancaster, S. L. (2003). Respiratory function and variability in individuals with Parkinson disease: Pre and post – Lee Silverman voice treatment. *Journal of Medical Speech and Language Pathology, Vol 11,* 185-201.

Isshiki ,N. (1964). Regulatory mechanism of voice intensity variation..*Journal of Speechand HearingResearch*, *Vol* 7, 17-29.

Jankovic, J. (2008). Parkinson's disease: Clinical features and diagnosis. *Journal of Neurology, Neurosurgery, and Psychiatry, Vol 79,* 368 – 37.

Jayakumar, T., & Savithri, S. R. (2012).Effect of Geographical and Ethnic Variation on Dysphonia Severity Index: A Study of Indian Population, *Journal of Voice, Vol 26, 1*, 11-16.

Jiang, J., O'Mara, T., Chen, H. J., Stern, J. I., Vlagos, D., & Hanson, D. (1999).Aerodynamic measurements of patients with Parkinson's disease.*Journal of Voice, Vol 13, 4,* 583-591.

Jiang, J., & Tao C. (2007).The minimum glottal airflow to initiate vocal fold oscillation.*Journal of Acoustic Society of America, Vol 121, 5,* 2873-81.

Kahane, J. C. (1987). Connective tissue changes in the larynx and their effects on voice. *Journal of Voice, Vol 1,* 27–30.

Kent, R. D., Kent, J.F., & Rosenbek, J. C. (1987). Maximum performance tests of speech production. *Journal of Speech and Hearing Disorders*, *Vol 52*, 367-387

Kent, R. D., Vorperian, H. K., Kent, J. F., & Duffy, J. R. (2003). Voice dysfunction in dysarthria: application of the multidimensional voice program. *Journal of Communication Disorders, Vol 36*, 281-306.

Kimura, M., Nito, T., Sakakibara, K., Tayama, N., & Nimi, S. (2008). Clinical experience with collagen injection of the vocal fold: A study of 155 patients. *Auris Nasus Larynx, 35*, *1,* 67-75.

Kreiman, J., Gerratt, B. R., & Berke, G. S. (1994).The multidimensional nature of pathologic vocal quality.*Journal of the Acoustical Society of America, Vol 96*, 1291–1301

Laszewski, Z. (1956). Role of the department of rehabilitation in preoperative evaluation of Parkinsonian patients.*Journal of the American Geriatric Society*,*Vol 4*, 1280-1284

Lieberman, P., Kako, E., Friedman, J., Tajchman G, Feldman L, S., Jiminez E, B. (1992). Speech production,syntax comprehension and cognitive deficits in Parkinson's disease. *Brain and Language*, *Vol 43*,169-189.

Logemann, J. A., Fisher, H. B., & Boshes, B. (1973). The steps in the degeneration of speech and voice control in Parkinson's disease. In J. Siegfried (Ed.), *Parkinson's disease: Rigidity, akinesia, behaviour* (pp. 101-112). Vienna, Austria: Hans Huber.

Logemann, J. A., Fisher, H. B., Boshes, B., & Blonsky, E. R. (1978).Frequency and co-occurrence of vocal tract dysfunctions in speech of large sample of Parkinson patients.*Journal of Speech and Hearing Disorders, Vol 43,* 47-57.

Ludlow, C. L. & Bassich, C. J. (1984).Relationships between perceptual ratings and acoustic measures of hypokinetic speech. In: M. R. McNeil, J. C. Rosenbek, & A. E. Aronson (Eds.), *The Dysarthrias: Physiology, Acoustics, Perception, Management* (pp. 163-192). San Diego: College Hill

Marquardt, T. P., (1973). Characteristics of speech in Parkinson's Disease: Electromyographic, Structural Movement, and Aerodynamic Measurements. Unpublished doctoral dissertation, University of Washington, Seattle.

Metter, E.J. & Hanson, W.R. (1986).Clinical and acoustic variability in hypokinetic dysarthria.*Journal of Communication Disorders, Vol 19*, 347-366

Moon, B., Adams, S.G., Jog, M. (2006). Effects of Background noise, listener context, speech task, and requests for clarification on speech intensity in Parkinson'sdisease. *Stem-Sprak-,en Taalpathologie, Vol 14*, 84

Mueller, P. B. (1971). Parkinson's disease: motor speech behaviour in a selected group of patients. *Folia Phoniatrica, Vol 23,* 333-346

Murdoch, B.E., Chenery, H.J., Bowler, S. (1989). Respiratory function in Parkinson's subjects exhibiting a perceptible speech deficit: A kinematic and spiromoetric analysis. *Journal of Speech and Hearing Disorders*, *Vol 54*, 610-626.

Nasri, S., Namazie, A., Kreiman, J., Sercarz, J, A., Gerrat, B, R., Berke, G, S. (1994)A pressure-regulated model of normal and pathologic phonation. *Otolaryngolgyand Head and Neck Surgery, Vol 111, 6,* 807-15.

Piccirillo, J. F., Painter, C., Fuller, D., & Fredrickson, J. M. (1998).Multivariate analysis of objective vocal function.*Annals of Otology Rhinology Laryngology, Vol 107,2,* 107–112.

Piccirillo, J. F., Painter, C., Fuller, D., Haiduk, A., & Fredrickson, J. M. (1998).Assessment of two objective voice function indices.*Annals of Otology RhinologyLaryngology, 107, 5,* 396–400.

Razdan, S., Kaul, R.L., Motta, A., Kaul, S., & Bhatt, R.K. (1994).Epidemiology of Neurological disorders in Kashmir.*Neuroepidemiology*,*Vol 13*, 113-119.

Rothenberg, M. (1972).A new inverse-filtering technique for deriving the glottal air flow waveform during voicing.*Journal of the Acoustic Society of America, Vol 53, 6*, 1632 – 1645.

Sanabria, J., Ruiz, P. G., Guitierrez, R., Marquez, F., Escobar, P., Gentil, M., & Cenjor, C. (2001).The effect of levodopa on vocal function in Parkinson's disease.*Clinical Neuropharmacology, Vol 24, 2*, 99-102

Sapienza, C. M., & Dutka, J. (1996). Glottal airflow characteristics of women's voice production along an aging continuum. *Journal of Speech Language and Hearing Research,Vol 39, 2,* 322-328.

Sapir, S., Ramig, L., & Fox, C. (2008).Voice, speech and swallowing disorders.In S. Factor, & W. Weiner, (Eds.).*Parkinson disease: Diagnosis and clinical management,* (pp. 77–97). New York City, New York: Demos Medical Publishing

Sarr, M. M, Pinto, S, Jankowski, L., Teston B, Purson A, Ghio A, Regis J, Peragut, J.C., Viallet, F. (2009). Contribution de la measure de la pression introrale pour la comprehension des troubles de la coordination pneumophonique dans la dysarthrie parkinsonienne. *Rev Neurol, Vol 165*, 1055-1061

Smitheran, J. R. & Hixon, T. (1981).A clinical method for estimating laryngeal airway resistance during vowel production. *Journal of Speech and Hearing Disorders, Vol 46*, 138-146.

Solomon, N. P., & Hixon, T. J. (1993).Speech breathing in Parkinson's disease.*Journal of Speech and Hearing Research*, *Vol 36*, 294-310

Stathopoulos, E. T. & Weismer, G. (1985). Oral airflow and air pressure during speech production: A comparative study of children, youths, and adults. *Folia Phoniatrica*, *Vol 37*, 152-159.

'Tang, J., & Stathopoulos, E. (1995). Vocal efficiency as a function of vocal intensity: A study of children, women, and men. *Journal of the Acoustical Society of America,Vol 97,* pp 1885 -1892.

Tanner, C, M., & Goldman, S, M. (1996). Epidemiology of Parkinson's Disease. *Neurology Clinics, Vol 14*, 317-335

Teston, B. (2007).L'étude instrumentale des gestes dans la production de la parole : Importance del'aérophonométrie. In Auzou P, Monnoury-Rolland V, Pinto S, Özsancak C (eds),*LesDysarthries*. pp 248-258.Solal.Marseille.

Titze, I. R. (1994) Principles of voice production. San Diego: College Hill Press, as cited in Christopher D., & Lorraine O. R. (1998). The effect of lung volume on selected phonatory and articulatory variables. *Journal of Speech, Language and Hearing Research, Vol 41, 3*, 491 – 510.

Uitti, R.J. & Calne, D.B. (1993).Pathogenesis of idiopathic Parkinsonism.*European Neurology, Vol 33, 1*, 6-23

Van de Heyning et al., (1996). Research work of the Belgian Study Group on Voice Disorders.*Acta Oto-Rhino-Laryngologica Belgica, Vol 50*, 321–386.

Vincken, W. G., Gauthier, S. G., & Dollfuss, R. E. (1984). Involvement of upper-airway muscles in extrapyramidal disorders. *New England Journal of Medicine*, *Vol 311*, 438-442.

Weiner, W, J., & Lang, A, E. (1989).*Movement Disorders: A comprehensive Survey*. Mount Kisko, NY: Futura.

Wolfe, V., Fitch, J., and Martin, D. (1997).Acoustic measures of dysphonic severity across and within voice types.*Folia Phoniatrica Logopaedica, Vol 49,* 292 -299.

Wolfe, V., Fitch, J., & Cornell, R. (1995).Acoustic prediction of severity on commonly occuring voice problems.*Journal of Speech and Hearing Research, Vol 38*, 273–279.

Wuyts, F. L.et al., (2000)**.** The dysphonia severity index: an objective measure of vocal quality based on a multiparameter approach**.***Journal of Speech-Language and Hearing Research, Vol 43, 3*, 796-809.

Yuceturk, A. V., Yilmaz, H., Egrilmez, M., & Karaca, S. (2002). Voice analysis and videolaryngostroboscopy in patients with Parkinson's disease.*European Archives of Otorhinolaryngology, Vol 259*, 290-293

Zwirner, P., Murry, T., & Woodson, G. E. (1991).Phonatory function of neurologically impaired patients. Journal of Communicaton Disorders, *Vol 24*, 287-300

APPENDIX

Informed Consent Form

ALL INDIA INSTITUTE OF SPEECH & HEARING

Naimisham Campus

Manasagangothri, Mysore 570 006

TITLE OF STUDY: Aerodynamics and Acoustic measures of voice in persons with Parkinson's Disease

CONSENT FORM

I have been informed about the aims, objectives and the procedure of the study. I understand that I have a right to refuse participation or withdraw my consent at any time. I have the freedom to write to head of the Institute in case of any violation of these provisions without the danger of my being denied any rights to secure the clinical services at this institute. I am interested in allowingmy child to participate for the study and hereby give my written consent for the same.

I, _____, the undersigned, give my consent to be participant of this investigation/study/program. I have no objection in participating in the program.

Signature of Participant Name and Address:

Date: