Abstract

Title of Dissertation: VOCAL DYSFUNCTION IN YOUNG-ONSET PARKINSON'S DISEASE

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Vocal dysfunction is well established in persons with older-onset Parkinson's disease (PD), but has not been investigated in the young-onset PD (YOPD) population. Voice deficits associated with older-onset PD mirror the characteristics of vocal aging, suggesting that our current knowledge base of laryngeal dysfunction in the PD population is confounded by aging effects. The purpose of this study was threefold: (a) to examine perceptual voice characteristics and the potential impact of voice symptoms on quality of life; (b) to compare YOPD and healthy control (HC) speakers' performance on two routinely used clinical tasks (sustained vowel phonation and laryngeal diadochokinesis); and (c) to experimentally manipulate and compare speakers' performance in producing phonatory offset-onset gestures as reflected in four phonetic contexts (each eliciting a different mechanism) across three speaking modes. Twelve YOPD speakers and twelve healthy control (HC) speakers participated. YOPD speakers reported voice symptoms of hypophonia, tremor, hoarseness, monotone, and

impaired speech intelligibility. They demonstrated a mild to moderate voice handicap. Findings revealed no speaker group differences for speech intensity on sustained vowel phonation and reading tasks. YOPD speakers demonstrated a significantly decreased rate of syllable repetition and used a significantly greater number of pauses during production of one of two laryngeal diadochokinetic tasks. Acoustic measures associated with mechanisms of phonatory offset-onset demonstrated trends of speaker group differences, suggesting that YOPD speakers have impaired voicing control for mechanisms of phonatory offset-onset not associated with oral constriction. Intra-speaker group variability was observed for YOPD speakers. Inspection of speaker groups' performance across speaking modes suggested a disruption in the habitual setting of laryngeal posture in YOPD speakers; namely, they use a laryngeal postural setting that is similar to that observed in HC speakers when speaking in an aspirant or breathy voice mode. Speech masking facilitated a speaking mode change in YOPD speakers and could provide an effective and efficient treatment method for training persons with YOPD to speak in a projected mode. Vocal dysfunction is associated with YOPD and voice symptoms can appear early in the disease process, sometimes preceding onset of limb symptoms. Persons with YOPD should be routinely assessed for vocal dysfunction.

VOCAL DYSFUNCTION IN YOUNG-ONSET PARKINSON'S DISEASE

by

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Introduction

The word *parkinsonism* refers not to a particular disease but to a commonly recognized neurological condition marked by a characteristic set of features that include a resting tremor of the hands and legs, muscular stiffness (e.g., rigidity), and slowness of body movement (i.e. bradykinesia) (Duvosin & Sage, 1996). The most prevalent type of parkinsonism is known as idiopathic Parkinson's disease (PD) meaning the cause is unknown. Typically the onset of PD occurs in adult life, having a peak incidence between 50-69 years of age with an average onset in the sixth decade (Hoehn & Yahr, 1967; Zetusky, Jankovic, & Pirozzolo, 1985). It is, therefore, considered a disease of the elderly. Giovannini et al. (1991) speculated that the concept of PD as a disease of the elderly was so widely accepted by physicians that patients younger than 50 years who presented with parkinsonian features were diagnosed with a *parkinsonian-like* syndrome, suggesting that it was a different disease. Recent advancements in research have led to the diagnosis of PD in patients who develop parkinsonian symptoms or signs in young adulthood or prior to the age of 50 years. These young patients have arbitrarily been diagnosed with *early-onset Parkinson's disease* (Arevalo, Jorge, Garcia, Scipioni, & Gershanki, 1997; Boxall, 1994; Giovannini et al., 1991; Muthane, Satishchandra, & Subhash, 1993) or *young-onset Parkinson's disease* (Gibb & Lees, 1988; Pantelatos & Fornadi, 1993; Quinn, Critchley & Marsden, 1987; Schrag, Ben-Shlomo, Brown, Marsden & Quinn, 1999; Stern et al., 1991). Clinical studies differ in their definition of the age span inclusion of young-onset Parkinson's disease (YOPD). Some have defined their cohort to include patients

whose initial symptoms developed prior to the age of 40 years (Gibb & Lee, 1988; Pantelatos & Fornadi, 1993; Quinn, Critchley & Marsden, 1987; Schrag et al., 1999; Stern et al., 1991), whereas others have investigated young onset and early-onset using an upper age limit of prior to age of 50 years (Watts, 1997). Those who have symptoms appearing earlier than age of 50 years are referred to as *juvenile parkinsonism;* and when compared with other forms of parkinsonism, juvenile parkinsonism may represent a different clinical entity, presenting with dystonia (Muthane et al., 1994) and a higher familial incidence in comparison with idiopathic PD (Quinn et al., 1987).

The majority of individuals with Parkinson's disease develop voice and speech abnormalities during the course of their disease progression. The reported incidence of voice and speech disorders in PD ranges from 73% to 89% (Hirose, Kiritani, Ushijima, Yoshioka, & Sawashima, 1981; Logemann, Fisher, Boshes, & Blonsky, 1978; Ramig, Countryman, Thompson, & Horii, 1995). Logemann and colleagues studied 200 nonmedicated individuals with PD who were mostly of idiopathic onset but included some post encephalitic cases who developed PD subsequent to a viral flu epidemic (Logemann, Blonsky, & Boshes, 1973; Logemann, Boshes, Blonskey, & Fisher, 1977; Logemann, Fisher, Boshes, & Blonsky, 1978). The cohort represented all five stages of disease progression (Hoehn & Yahr, 1967). The investigators noted a high incidence (89%) of early onset of voice disorders in individuals with PD, while only forty-five percent had abnormal articulation. Other speech abnormalities such as hypernasality and fluency disorders (e.g., repetition of syllables, abnormally long pauses, and

abnormally long syllables) were present but less frequent. They did not measure prosodic disturbances involving intonation nor did they indicate the age of the individuals studied. Although published studies do not report quantitative data to estimate the functional impact of these PD disorders, anecdotal accounts suggest that these voice and speech deficits have adverse effects on the individual's communicative effectiveness, as well as economic, social, and psychological well-being (Barbeau, Dushay & Spiegel, 1965; Habermann, 1996; Marr, 1991; MacCarthy, & Brown, 1989; Mutch, Strudwick, Roy & Downie, 1986; Ramig, Countryman, O'Brien, Hoehn, & Thompson, 1996; Yorkston, Bombardier, & Hammen, 1994).

Other studies have corroborated the high incidence of voice disorders and early appearance of voice disorders. Stewart et al. (1995) reported that 12 persons, assessed in the early stages of the disease, had at least two characteristics of a voice disorder. Interestingly, 8 of the 12 individuals were not aware of their vocal dysfunction. Hanson, Gerratt, and Ward (1984) reported that 30 of their 32 PD participants had vocal dysfunction (described as *weakness*), and that the degree of articulatory dysfunction did not correlate with voice abnormalities. Furthermore, the occurrence of voice symptoms preceded the onset of limb symptoms in some individuals (Hanson et al, 1984; Tetrud, 1991). In fact, Tetrud (1991, p. 70) reported, "long before the disease is diagnosed, family members and friends of a prospective patient may notice the voice has become softer or slightly hoarse." He advocated the development of an automated voice and speech analysis technique to provide a method of identifying individuals with pre-clinical disease.

"This would permit the institution of protective therapy before the disease becomes clinically manifest. " (Tetrud, 1991, p. 71). In summary, voice disorders are a frequently occurring problem in PD. The effects of age on PD-related voice disorders are uncertain.

Typical vocal manifestations of PD include (a) breathy or rough voice quality (Logemann et al., 1978; Hanson, Gerratt, & Ward, 1984); (b) reduced loudness (*hypophonia*) (Ramig et al.); (c) voicing control deficits that include difficulty producing rapid phonatory offset-onset during speech (Ackermann & Ziegler, 1991; Canter, 1965; Forrest et al., 1989; Kent & Finley Kent, 2000); and (d) reduced pitch inflection and reduced control in altering pitch during speech segments (*hypoprosodia*) (Caekebeke, Jenneknes -Schinkel, van der Linden, Buruma & Roos, 1991; Robertson & Thompson, 1984; Scott & Caird, 1984). Speech timing deficits involving speech rate disturbances and the use of longer and more frequent pauses have been reported by several investigators (Hammen & Yorkston, 1996; Metter & Hanson, 1986; Pitcairn, Clemie, Gray, & Pentland, 1990; Solomon & Hixon, 1993) and are most likely attributed to phonatory deficits. Vocal tremor may also occur but is frequently not audible (Gath & Yair, 1988). Taken together, investigations of speech deficits in PD suggest that a large portion of these deficits involve the phonatory system.

Voice deficits associated with PD markedly mirror the characteristics of vocal aging, suggesting that our current knowledge base of laryngeal dysfunction in the PD population is confounded by aging effects (Hori & Ryan, 1981; Huntley, Hollien, & Shipp, 1987; Linville & Korabic, 1986; Ptacek et al., 1966). Perceptual

characteristics of the aged voice have included (a) inappropriate pitch levels, (b) hoarseness/roughness, (c) vocal tremor and increased phonatory breaks, (d) breathiness, and (e) reduced loudness (Linville, 1996; Mysak & Hanley, 1958; Shipp & Hollien, 1969). These perceptual features have been further documented with parallel acoustic observations of (a) changes in fundamental frequency (F_o), whereby males show an increase in F_o and females show a decrease (Higgins & Saxman, 1991; Hollien & Shipp, 1972; Linville, 1987, 1992; Mueller, Sweeney, & Baribeau, 1984; Ramig & Ringel, 1983; Stoicheff, 1981); (b) increased short-term acoustic perturbations of jitter and shimmer (Brown, Morris, & Michel, 1989; Decoster & Debruyne, 1997; Ramig, 1983; Ramig & Ringel, 1983); (c) increased phonatory instability or long-term acoustic perturbations (i.e. tremor, subharmonics) (Decoster & Debruyne, 1997; Linville, 1996; Mysak & Hanley, 1958; Ramig & Shipp, 1987; Shipp & Hollien, 1969); (d) increased spectral noise (Decoster and Debruyne, 1997); and (e) reduced vocal intensity (Baker, Ramig, Sapir, Luschei, & Smith, 2001; Stathopoulos & Sapienza, 1993). Although respiratory and supraglottal systems contribute to these changes, tissue changes in the composition of the extracellular matrix, the muscular changes in the larynx, and a loss of neural control are believed to be the primary contributors to vocal aging (Hoit & Hixon, 1987; Sonies, Stone, & Shauwker, 1984). Therefore, separating disease-related versus age-related changes in phonatory function is difficult when one studies only an older-onset cohort of PD persons.

Need for Study

Studies of vocal dysfunction associated with PD have included only aged persons or have grouped data for both aged individuals and persons with young-onset Parkinson's disease (YOPD). There are no published studies reporting vocal dysfunction in a cohort limited only to individuals with YOPD who are non-geriatric (under the age of 60 years). Therefore, it is uncertain if the vocal dysfunction observed in PD is attributable solely to the disease process or is confounded by aging effects.

It is important to assess the functional impact of vocal dysfunction in persons with YOPD to determine the need for voice treatment. Previous studies have not employed sensitive psychometric tools to probe psychosocial consequences of voice disorders, such as emotional, functional, and physical restrictions placed on daily living. In comparison with an older-onset cohort, it is likely that this YOPD cohort views their vocal dysfunction as having greater negative impact on their quality of life, given the economic need of the younger person to maintain employment.

Exploration of hypotheses regarding underlying mechanisms that may influence the vocal dysfunction in PD is critical to the development of effective treatment programs. One hypothesis is that the multidimensional changes of breathy voice quality and hypophonia reported in PD speakers suggests a change in the speaker's voice mode or habitual *setting* of the laryngeal posture. Recent evidence suggests that sensory processing deficits associated with the neural degenerative process in PD may disrupt the internal regulation of vocal loudness in

PD participants (Ho, Bradshaw, Iansek, & Alfredson, 1999; Ho, Iansek, & Bradshaw, 1999). That is, people with PD perceive their normal conversational voice as loud when it actually is soft because of a deficit in their internal regulation of speech volume. As such, their resulting speech intensity is insufficient for effective communication. Furthermore, evidence suggests that PD speakers use greater respiratory drive and oral constriction to compensate for glottal insufficiency. This results in a greater effort than that used by healthy speakers (Baker et al., 2001). To avoid the sense of a greater effort, speakers with PD may adjust their habitual laryngeal setting or mode to one that healthy speakers use when they are talking in a quiet or confidential voice (e.g., talking in a library or telling a secret so as not to be overheard by others).

Recent behavioral treatments for PD-related voice disorders have focused on altering voice mode by increasing loudness or physical effort when talking (Ramig, 1995a, 1995b; Ramig, 1997, 1999; Ramig, Bonitati, Lemke, & Horri, 1994; Ramig, Countryman, O'Brien, Hoehn, & Thompson, 1996; Ramig, Countryman, Thompson, & Horri, 1995; Ramig & Dromey, 1996; Ramig & Verdolini, 1998). In the Lee Silverman Voice Treatment™ (LSVT™), the clinician cues the patient to “think loud.” However, external cueing like “think loud” may impose a cognitive load that is difficult for the person with PD to remember. Although the efficacy data in one study comparing pre- to post-treatment changes in vocal intensity showed a significant increase for sustained phonation (14 dB) and reading (12 dB), changes in conversational speech (although statistically significant) were substantially less (5 dB) (Ramig et al., 1995). These results and

anecdotal comments from persons who are treated with the LSVT™, and have stated they have difficulty generalizing their 'loud speech' outside of the clinical setting, suggests a need for alternative cueing mechanisms to facilitate normal loudness when speaking. Speech masking also serves as an external cueing technique for increased vocal effort in PD, and may impose fewer cognitive constraints (Adams & Lang, 1992). However, research evidence pertaining to the use of masking noise has been limited, and the efficacy of masking noise in treating vocal dysfunction in speakers with PD is uncertain because prior studies have been confounded by aging and hearing loss (Adams & Lang, 1992; Ho, Bradshaw et al., 1999). Therefore, further studies are needed to determine the effectiveness of speech masking in speakers with PD.

Statement of Purpose

Vocal dysfunction has not been investigated in a cohort of YOPD individuals. Therefore, the main purpose of this study was to delineate the multidimensional changes in voice characteristics in these individuals, who represent a form of PD uncontaminated by aging effects, and to determine the impact of such dysfunction on quality of life. Additionally, this study sought to identify underlying vocal deficits as evidenced in traditional clinical tasks of sustained phonation and laryngeal diadochokinesis (L-DDK). Finally, this study manipulated speaking contexts by comparing a speaker's habitual or conversational voice to use of (a) a *confidential voice* elicited by external verbal cueing to speak in a breathy, aspirant voice, and (b) a *projected voice* elicited by external cueing with speech masking (to elicit changes in vocal effort associated

with speaking in a loud, projected voice). The purpose of this experimental manipulation was to determine the effects of voice mode manipulation on speech intensity and phonatory offset-onset control.

Literature Review

Prevalence and Incidence

Reported incidence and prevalence figures for PD are discrepant and varied. Incidence is the ratio of the number of new cases that arise during a time period (usually one year) to the number of people in the whole population, whereas prevalence is the ratio of the number of cumulative existing cases to the number of people in the whole population (Morton, Hebel, & McCarter, 1996). Total U.S. prevalence estimates for Parkinson's disease are wide-ranging, and vary from 500,000 to 1.5 million with the incidence being 1.2 – 1.5 times greater in males (Duvoisin & Sage, 1996; Koller, 1992; Lieberman & Williams, 1993; Wooten, Currie & Bovbjerg, 2004)). According to Rajput and Rajput (2002), 3% of the US population over the age of 65 and 10% of the US population over the age of 80 has PD.

Although the prevalence of PD is uncertain, reported figures suggest that the disease occurs with substantial frequency. Further, prevalence will most likely triple over the next 50 years with the aging of the population (Goldman &Tanner, 1998) and increasing longevity of the PD population due to improved pharmacological and medical management. The prevalence of YOPD is more uncertain and has been reported to be as high as 10% of the total Parkinson's disease population (Koller, 1992). Given the range in prevalence figures, this would represent 50,000 to 150,000 people in the U.S.

Etiology and Risk Factors

In chemical terms, parkinsonism is defined as a state of brain dopamine reduction or depletion (Weiner, Schulman, & Lang, 2001). A deficiency in dopamine can occur in a variety of ways. The nerve cells of the substantia nigra can be injured by tumors, stroke, environmental toxins, drugs, or encephalitic viruses (Goldman & Tanner, 1998). Additionally, neural cell loss is associated with the aging process (Koller et al., 1986). The exact cause of substantia nigra degeneration in PD is unknown. Theories regarding etiology have been proposed; however, none are universally accepted.

Aging, genetic, and environmental factors have been implicated as causes of PD (Duvosin & Sage, 1996; Goldman & Tanner, 1998; Lieberman & Williams, 1993). With respect to aging, some investigators have predicted that anyone over the age of 120 years would develop Parkinson's disease (Lieberman & Williams, 1993). This contention has been supported by the increased incidence of PD in people over the age of 60 years. Development of PD prior to age fifty, long before aging can be invoked as an explanation, suggests that the aging process cannot solely account for the occurrence of PD. Genetic factors have also been investigated. Investigators of familial studies have reported PD occurring in as many as five or six generations in several kindreds worldwide (as reviewed in Goldman & Tanner, 1998). Autosomal dominant and autosomal recessive inheritance patterns have been described in a small percentage of the PD population (Gasser, 1998). Also, environmental causes of PD have been suspected.

Investigators of ecologic factors have reported a greater incidence of PD in industrialized countries. Goldman and Tanner (1998) identified rural living, farming, gardening, pesticide use, and well water drinking as risk factors. They also cited the possible role of diet. A major criticism of the environmental hypotheses of PD etiology is the rarity of clusters. Taken together, data suggest that the etiology of PD is multi-factorial.

In summary, multiple etiologies for PD appear likely and may involve a combination of both a genetic susceptibility and an exposure to toxic agents or viruses (Lieberman & Williams, 1993). Alternatively, a genetic predisposition may exist that produces a cascade of cellular reactions leading to the destruction of the dopamine-making cells of the substantia nigra (Youdim & Riederer, 1997). Evidence suggests substantia nigra neurons die as a result of an excessive accumulation of highly reactive molecules (i.e., free radicals) and in a subsequent process referred to as *oxidation*. As found in both animal and human studies, neuroprotective therapy (NPT), involving the use of drugs such as selegine and vitamin E, may protect against the oxidation process and subsequent substantia nigra cell loss (Lieberman, 1992; Mizuno, Mori & Kondo, 1994; Youdim & Riederer, 1997).

Pathophysiology of Parkinson's Signs

The complex of signs referred to as *parkinsonian features*reflects the dysfunction of a small subcortical region of the brain known as the basal ganglia, which includes the neostriatum (caudate and putamen), the ventral striatum, the external and internal segments of the globus pallidus, the subthalamic nucleus, and

the substantia nigra pars reticulata and pars compacta. (Wichman & DeLong, 2003). As stated by Murdoch, (2001), " information originating in the cerebral cortex passes through the basal ganglia and returns via the thalamus to specific areas of the frontal lobe, this feedback circuit often being referred to as the cortico-striato-pallido-thalamo-cortical loop" (p. 234). Alexander et al. (1986) identified five separate, functionally segregated circuits according to the specific regions of the frontal lobe that serves as a target for their thalamocortical projections. The cortical targeted areas serve motor regions (skeletomotor areas, oculomotor, cognitive regions (Areas 46, 12, 24) and limbic structures (Area 13). Strick et al. (1995) identified three additional loops serving supplementary motor, ventral premotor, and primary motor regions. This anatomical arrangement, whereby the output from the basal ganglia gains access to multiple areas of the frontal, including non-motor areas, has profound consequences for the possible function roles of the basal ganglia. The basal ganglia influences not only speech and limb movements, but cognition as well as language.

In PD, the hypokinetic movement disorders result from increased inhibition of thalamocortical neurons that results in cortical deficits involved in the initiation movement (Murdoch, 2001). Specifically in PD, neurons within the dopaminergic nigrostriatal pathway of the basal ganglia die which results in the depletion of the neurotransmitter dopamine (Alexander, Delong, & Strick, 1986; Delong & Georgopoulos, 1981; Evarts, Kimura, Wurtz, & Hikosaka, 1984; Wichman & DeLong, 2003). This decline in dopamine results in the following chain of events in the basal ganglia loops: (a) An excessive inhibition of the external globus

palladal output; (b) disinhibition of the output of the substantia nigra; (c) excessive excitatory drive to the output structures (internal globus pallidus and substantia nigra pars reticulata; and (d) thalamic inhibition. The thalamic inhibition renders the cortical projection areas less responsive to other inputs normally involved in initiating movement (Murdock). This is believed to result in bradykinesia and akinesia (Fahn, 1989; Marsden & Obeso, 1994; Murdoch, 2001; Wichmann & DeLong, 2003;Youdim & Riederer, 1997). It is important to note that these eight loops are "segregated in nature with specific independent circuits subserving specific anatomical independent circuits subserving specific anatomical structures such as the orofacial structures versus the limb muscles" (Murdock, p. 242) and there is a somatopic organization that is preserved in the loops.

Hypotheses regarding the dysfunctions characterizing PD are based on both human participant research and animal studies. Human participant research has focused on pharmacological studies and the effects of surgically induced lesions, such as pallidotomy (Schulz, 2000; Schulz & Grant, 2000; Solomon, McKee et al., 2000), as well as deep brain electrical stimulation (DBS) (Jahanshahi et al., 2000; Montgomery, 1999; Montgomery & Baker, 2000; Pinter et al., 1999; Sandyk, 1997; Schulz & Grant, 2000; Siegfried & Lippitz, 1994; Solomon, McKee et al., 2000). Neurochemical and neurophysiologic studies performed on normal primates and those rendered parkinsonian by the drug N-methyl-4-phenyl-1, 2,3,6-tetrahydropyridine (MPTP) have also contributed to the knowledge base of pathophysiology (Burns et al., 1983; Schneider, Unguez, Yuwiler, Berg, & Markham, 1988; Schultz, 1988; Willis, Savulescu, Horne, & Smith, 1987;

Zigmond & Stricker, 1989) and the development of theoretical models of basal ganglia function (Connor & Abbs, 1990; Delong & Georgopoulos, 1981; Denny-Brown & Yanagisawa, 1976; Hayes, Davidson, Keele, & Rafal, 1998; Miller & Delong, 1988; Obeso, Rodriguez, & DeLong, 1997; Wichmann & DeLong, 2003).

Diagnostic Criteria and Cardinal Motor Signs

Regardless of etiology, substantia nigra cell degeneration produces a distinct set of clinical signs (Fahn, 1989; Miller & DeLong, 1988; Marsden, 1984). Because there are no confirmatory laboratory procedures, the neurologist's diagnosis of PD is based on the presence of *bradykinesia* and either *rest tremor* or *rigidity* (Schneider, Diamond, & Markham, 1986) having no secondary etiologies (e.g., drug-induced, multiple infarcts, or presence of a metabolic disorder).

Bradykinesia.

Bradykinesia, from the Latin *brady* (slow) and kinesia *(movement),* is described as slow performance of voluntary movements and is measured in limb and speech movement studies by increased movement times or durations (Delwaide & Gonce, 1998). Hallett and Khosbin (1980) noted that bradykinesia is the predominant feature of the motor disability of PD persons. During single joint arm movements in healthy individuals, the pattern of electromyography (EMG) activity at both moving and stabilizing joints varies as a function of the rate of movement. When a rapid movement is made around a joint such as the elbow, a characteristic triphasic pattern of EMG activity occurs in the muscles acting across that joint. In a study of arm movements, Hallett and Khosbin (1980) demonstrated that persons with PD were unable to generate the appropriate triphasic pattern of

EMG activity within the set time frame of ballistic movement burst. The triphasic reciprocal pattern normally seen with rapid movements was absent, and it was replaced by simultaneous activity that persisted throughout the movement in both the agonist and antagonist muscle. A lack of reciprocal EMG pattern is indicative of bradykinesia. Specific limb motor deficits that are attributable to bradykinesia include the following: (a) Delayed motor initiation, reflected by prolonged reaction time; (b) hypokinesia, defined as difficulty reaching a target with a single continuous movement, whereby the movement must stop and then resume in order to reach the target; (c) rapid fatigue with repetitive movement, as demonstrated by progressively reduced movement amplitudes throughout a movement sequence, (d) an inability to execute sequential actions; and (e) loss of automatic movements, such as arm swinging while walking, loss of facial expression (hypomimia), reduced eye blinking, and reduced swallowing of saliva (Delwaide & Gonce, 1998).

Resting Tremor.

Tremor at rest (i.e. when body parts are relaxed) is often a patient's first complaint (Duvoisin & Sage, 1996; Koller & Huber, 1989) and is initially unilateral, involving a hand (most common), foot, or leg. As the disease progresses, there is bilateral limb involvement. Kinetic or intention tremor, occurring during the movement of a body part toward a goal, has not been reported in early disease progression (Logemann et al., 1978). However, postural or static tremor, occurring when a limb is outstretched, is common (Gresty & Findley, 1984). The reported tremor in speech muscles has a frequency rate of 5 Hz

(Hunker & Abbs, 1990; Ludlow, Bassich, Connor, & Coulter, 1986; Perez, Ramig, Smith, & Dromey, 1996; Philippbar, Robin, & Luschei, 1989), although the frequency may vary from muscle to muscle. Electromyography studies of tremor describe rhythmic activity alternating in antagonistic speech muscles at frequencies between 4 and 6 Hz (Hunker & Abbs, 1990; Philippbar, Robin, & Luschei, 1989). This rhythmic activity differs from the normal or physiologic rest tremor of 8 – 12 Hz that is barely visible in amplitude (Ludlow, Bassich, Connor, & Coulter, 1986).

Also, the occurrence of tremor within a particular individual is variable. The tremor is typically suppressed by willed activity, sleep, and complete relaxation of the axial postural muscles. The amplitude of the limb tremor increases with stress, anxiety, or isopropernol administration. Pathologic rest tremor of the jaw, lips, and tongue is less frequent than limb tremors (Hunker & Abbs, 1990; Hanson, Gerratt, & Ward, 1984) and head tremors are rare (Duvoisin & Sage, 1996). Vocal tremor has also been noted using acoustic or cinegraphic techniques but is usually not detected by perceptual-auditory judgment (Hanson, Gerratt, & Ward, 1984; Jiang, Lin, & Hanson, 2000; Ludlow et al., 1986; Perez et al., 1996; Stelzig, Hochhaus, Gall, & Henneberg, 1999; Zwirner & Barnes, 1992). Specifically, vocal or laryngeal tremor has been quantified in small subsets of persons with PD using spectrographic (Stewart et al., 1995) and cinegraphic visualization (Hanson et al., 1984). However, the laryngeal tremor can manifest in early stages of Parkinson's disease, appearing under endoscopic observation as vertical movement of the larynx (Perez et al., 1996; Stewart et al., 1995).

Rigidity.

Persons with PD complain of a feeling of stiffness, which may be their subjective appreciation of rigidity. Rigidity is not a symptom but a subjective sign observed during physical examination, when the clinician takes the patient's arm and gently bends and straightens it a number of times while asking the patient to relax. The clinician is palpating for a resistance to passive motion around the elbow or wrist joint. Additionally, similar resistance to passive motion at other joints such as the knee or ankle may exist. The resistance to passive motion associated with PD has a characteristic feel involving a regular, jerky quality described as a "ratchet gear" or "cogged wheel" movement across the joint being manipulated. This is referred to as *cogwheel rigidity*. The rigidity associated with PD is also distinct from the stiffness associated with spasticity, which is likened to a consistent resistance such as that associated with trying to bend a lead pipe (Duvoisin & Sage, 1996). The subjective impression of muscular rigidity can be likened to a tensed state of sustained muscle contraction observed when a limb is at rest or being passively moved. Palpation of normal musculature should reveal a relative *softness* during rest. Under conditions of rigidity there is a palpable tightness. Persons who have rigidity typically complain of a sense of stiffness, a tired or aching feeling, persistent soreness, pain, or a cramp.

Miller and Delong (1988) described two related, but dissociable disorders of muscle activity in PD. First, there is an increase in the resting level of EMG activity, with a corresponding difficulty in achieving complete muscle relaxation. Second, there is documentation of abnormally long latency reflexes (LLR) in PD

participants (Bergui et al., 1992; Meara & Cody, 1992). Miller and Delong (1988) stated:

> The LLR occurs approximately 40-100 ms following the passive stretch of a muscle and is distinguishable from the short latency spinal reflex. In normal individuals, the magnitude of the LLR is dependent upon several factors, including instructions to respond to the perturbation, stretch velocity, magnitude, and duration. However, in parkinsonian subjects, the LLR appears relatively independent of instruction and is nearly maximal at relatively small stretch parameters; hence, this indicates that the gain is relatively fixed and is abnormally high (p. 293).

Although these dissociable disorders of muscle activity have been documented, their relationship to the motor disorder associated with PD is not certain.

Electromyographic (EMG) investigations have quantified the subjective impression of rigidity in limb muscles of PD participants (Bergui et al., 1992; Meara & Cody, 1992). Electromyographic investigations of the speech musculature of PD participants have been reported for the lips (Caligiuri, 1987; Hunker, Abbs, & Barlow, 1982; Leanderson, Meyerson, & Persson, 1972; Nakano, Zubick, & Tyler, 1973; Pellat et al., 1983), the jaw (Moore & Scudder, 1989), and the larynx (Baker, Ramig, Luschei, & Smith, 1998; Guindi, Bannister, Gibson, & Payne, 1981; Hirose & Joshita, 1987; Kleu & Sram, 1969; Ludlow, Gallena, Lou, & Mennendez, 1995; Luschei, Ramig, Baker, & Smith, 1999). Muscle rigidity, consistent with findings reported in limb studies, has been documented only for lip muscles (Hunker et al., 1982; Pellat et al., 1983). Hunker et al. (1982) quantified labial rigidity in parkinsonian participants by applying known forces and observing the resultant displacements. They determined labial stiffness coefficient and measured EMG activity associated with lip displacement.

Lower lip, but not upper lip rigidity, was observed in all four persons with PD. Labial EMG responses to displacement for the parkinsonian participants involved higher-amplitude resting activity that increased markedly as the lower lip was displaced. There was no indication of a displacement-induced increase in the EMG recordings for controls.

Quantitative EMG measurements for laryngeal muscles are sparse and many of the early studies are confounded with artifact due to the type and placement of electrodes. Evidence of rigidity in laryngeal muscles in PD is speculative. Aronson (1990) attributed the characteristic parkinsonian voice to rigidity of the laryngeal muscles. He based this premise on a generalization that the laryngeal muscles would behave similarly to the limb musculature. However, conclusive evidence to support laryngeal rigidity does not exist (Jiang, Lin, Sheynin, & Hanson, 1999). In fact, Jiang et al., (1999) reported that EMG measurements suggested a lack of rigidity in the laryngeal muscles. Baker et al. (1998) reported reduced thyroarytenoid (TA) EMG amplitude in PD participants; and Luschei et al. (1999) observed that the firing rate of the TA muscle was decreased in male participants with PD. Such findings are contradictory to rigidity. Therefore, evidence to support the existence of laryngeal muscle rigidity in PD remains elusive.

The exact contribution of rigidity to the overall parkinsonian handicap is not well documented. According to Delwaide and Gonce (1998), some researchers believe that rigidity impedes voluntary movements, specifically speed. However, the observation that persons with severe rigidity demonstrate normal movement

velocities contradicts this view (Caligiuri, 1987; Hunker et al., 1982; Nakano et al., 1973). Therefore, the relationship between rigidity and slow movement is uncertain.

Secondary Signs

In addition to the cardinal motor signs, there are additional motor, sensory, autonomic, neuropsychological, and cognitive signs in PD. The motor signs include postural instability and other gait disturbances, freezing phenomena, and problems with dexterity and fine motor coordination. Sensory impairments including pain (Demirci, Grill, McShane, & Hallett, 1995; Jobst, Melnick, Byl, Dowling, & Aminoff, 1997; Koller, 1984; Solomon, Robin, & Luschei, 2000), impaired complex sensorimotor integration (Abbs & Connor, 1991; Connor & Abbs, 1991; Flint, Black, Campbell-Taylor, Gailey, & Levinton, 1993; Harrington & Haaland, 1991; Montgomery & Nuessen, 1990; Pascual-Leone, Valls-Sole, Brasil-Neto, Cohen, & Hallett, 1994; Pastor, Jahanshahi, Artieda, & Obeso, 1992; Playford et al., 1992; Schneider, Diamond, & Markham, 1986), and sensory processing deficits (Liotti, Ramig, Vogel, et al., 2003) are reported. Neuropsychiatric disturbances such as depression and hallucination may be observed in patients with PD (Marjama-Lyons and Koller (2001). In addition, neuropsychological (cognitive) deficits are associated with PD, presumably from disruptions or disconnections to other brain regions (Brown & Marsden, 1990; Delong & Georgopoulos, 1981; Marsden & Obeso; 1994; Youdim & Riederer, 1997). These motor, sensory, neuropsychiatric, and neuropsychological deficits play a role in the voice and speech disorder observed in individuals with PD.

Secondary Motor Signs.

One secondary motor sign affecting speech movements is freezing. The freezing phenomenon develops in the mid to latter stages of PD and is attributable to bradykinesia. Freezing is a sudden, unforeseen state of immobility, and frequently presents in connection with walking, speech, and hand movements (Ackermann, Grone, Hoch, & Schonle, 1993; Fahn, 1995; Logigian, Hefter, Reiners, & Freund, 1991). Freezing can occur in the absence of levodopa therapy or when the levodopa is temporarily not effective (e.g. off-freezing). The most common form of freezing, *start-hesitation*, is seen as a person initiates walking. The foot becomes immobile while attempting to lift it to step forward. The harder one tries to lift a foot, the more it is immobilized, with agonists and antagonists in the leg simultaneously contracting (Fahn, 1995). Freezing can also occur while speaking, resulting in a latency in the initiation of speech (Ackermann et al., 1993). However, speech freezing is less common than gait freezing. A form of *palilalia* (e.g. the repetition of sounds) is common in late stages of parkinsonism and may be related to the freezing phenomena (Fahn, 1995). This type of palilalia resembles stuttering, but stuttering is more severe in that the inhibitory blocks are of longer duration, and the blockages can occur at other segments during speech versus primarily the first syllable (Ackermann et al., 1993). Fahn (1995) also attributes the parkinsonism feature of *tachyphemia* (i.e. speaking rapidly) as a precursor of parkinsonian palilalia, while Darley and colleagues (Darley, Aronson, & Brown, 1969a) used the term *accelerating speech* referring to a rapid and accelerating movement pattern of the articulators. Darley et al. noted that this

extremely rapid pattern of speech production results in an unintelligible murmur, particularly toward the end of sentences. This symptom was observed in 4 of the 32 (13%) participants studied. A related symptom, *short rushes of speech* (i.e. the production of short rushes of connected speech during conversation separated by pauses) was observed in 19 of 32 (59%) of the persons with PD (Darley et al.).

Investigators have studied performance of rapid syllable repetition tasks (i.e. diadochokinesis tasks) that are speech correlates to the limb studies that have used finger tapping tasks (Ackermann et al., 1993; Ackermann, Hertrich, & Hehr, 1995; Ackermann, Konczak, & Hertrich, 1997; Canter, 1965; Kojima, Shimoyama, Ninchoji, & Uemura, 1989; Kreul, 1972; Mueller, 1971; Svensson, Henningson, & Karlsson, 1993). Diadochokinetic tasks require alternating articulatory movements (i.e., reiteration of a given syllable as fast as possible on a single breath). A widely used clinical tool, diadochokinetic tasks are useful for determining the speed and regularity of reciprocal movements of the speech articulators (Duffy, 1995). Renout and colleagues (Renout, Leeper, Bandur, & Hudson, 1995, p. 74) described diadochokinesis "as the action of arresting one motor impulse and substituting in its place a movement that is diametrically opposed." Oral diadochokinetic tasks involve consonant vowel repetitions (i.e. "pa pa pa …", "ta ta ta …" or "ka ka ka …"), and they assess functional capacities of the oral or supraglottal articulatory system. Laryngeal diadochokinetic (L-DDK) tasks assess the capability of the vocal folds to either (a) adduct- abduct rapidly to offset and onset phonation (i.e. "uh uh uh …"); or (b) abduct-adduct rapidly for phonatory offset and onset (i.e. "huh huh huh") (Ptacek, Sander, Maloney, &

Jackson, 1966; Renout et al., 1995; Verdolini & Palmer, 1997). Findings have been consistent across studies, indicating impaired performance for both oral and laryngeal diadochokinetic tasks in PD participants (Ackermann et al., 1995; Ackermann et al., 1997; Boshes, 1966; Canter, 1965; Kreul, 1972; Mueller, 1971). For oral tasks, performance is often normal if persons are verbally cued to speak at a slow or normal rate, or visually or auditory paced to repeat syllables at a rate of 4-6 Hz. However, speech freezing occurs in persons with PD if they are not externally cued or if paced at a rate above 6 Hz.

Ackermann and colleagues (Ackermann et al., 1993) described freezing periods associated with oral diadochokinetic tasks as characterized by production of a sustained /a/ instead of the required consonant-vowel sequences. The underlying articulatory trajectories were reduced in amplitude, with repetition rates in the range of 8-10 Hz (e.g., faster than normal rate of 5-6 Hz.). The articulatory undershoot (i.e., reduced movement amplitude) failed to establish a sufficient occlusion of the vocal tract, giving rise to the perceived speech freezing. Similar to finger tapping studies (Nakamura, Nagasaki, & Narabayashi, 1976; Narabayashi, 1995), oral diadochokinesis was preserved at lower rates (e.g. 4-6 Hz) while freezing occurred at faster rates. Reduced range of movement and freezing, occurring only for the faster oral diadochokinesis task in persons with PD, was also observed during repetitions of the syllable /va/ (Caliguiri, 1989). Logigian et al. (1991) asked PD participants to perform an oral diadochokinetic task cued (auditory-paced) at rates ranging from 1-10 Hz. Five of seven PD participants exhibited a progressive increase in repetition rate that the investigators

referred to as vocal *hastening.* Logigian et al. suggested that vocal hastening could be a result of an interaction of the articulators with the resting tremor. That is, "the influence of parkinsonian tremor on repetitive, voluntary motor behavior may derive from synchronization of central pattern generators, which generate voluntary rhythmic movements, by neural oscillators, which generate pathological tremor" (p. 178). This view has also been supported by Nakamura, Nagasaki and associates, who have investigated the hastening phenomena in finger tapping tasks (Nakamura, Nagasaki, & Narabayashi, 1976; Narabayashi, 1995).

A third secondary sign in PD involves difficulty with dexterity and fine motor coordination. Similar to limb findings, EMG descriptions of *coordinative breakdown* in parkinsonian speech have consistently reported the loss of reciprocity in activation of agonist and antagonist muscles associated with rapid lip (Caligiuri, 1987; Nakano et al., 1973; Pellat et al., 1983), jaw (Moore & Scudder, 1989) and laryngeal movements (Baker et al., 1998; Guindi et al., 1981; Hirose & Joshita, 1987; Kleu & Sram, 1969). Moore et al. (1989, p. 158) described a "less rigid coupling" of activity between jaw muscles irrespective of speech and nonspeech movements. In comparison to healthy controls who exhibited a clearly defined, reciprocal pattern of activity between jaw agonists and antagonists, persons with PD produced a poorly defined pattern of muscle activation. The relative timing of EMG activity in these participants was distinguished by tonic, low-level activation of the antagonistic muscle (i.e., diagastric) on which was superimposed appropriately timed modulation of higher-level activity. Furthermore, correlation coefficients between antagonistic pairs (i.e., diagastric

with each of the jaw elevating muscles) were high for controls (ranging from –0.81 to –0.86) and low for the PD participants (from 0.05 to 0.10). Likewise, correlations between agonist pairs showed the same trend (i.e., high correlations for controls and low for PD participants). Consequently, these reports contribute to the view that coordination of movement is impaired in PD.

Sensory Signs and Proprioceptive Disturbances.

Although the symptomatic manifestations of PD are predominantly motor (Marsden, 1984), sensory disturbances are well documented; it is apparent that they contribute to the voice and speech deficits. In fact, some investigators (Connor & Abbs, 1990; Fox, Morrison, Ramig, & Sapir, 2003) have suggested that the dysarthria associated with PD should be viewed as a sensory-motor disorder, and not a pure speech-motor control deficit. Fox, Morrison, Ramig, and Sapir (2002) cited two consistent and frustrating challenges to treating individuals with PD: (a) A failure of the PD patient to recognize that loudness is reduced, and (b) a persistent resistance to increase loudness to a normal, conversational level. For example, soft-speaking individuas with PD will report that they are not speaking softly; rather their spouse needs a hearing aid (Fox & Ramig, 1997; Marsden, 1982). When listening to an audio recording while speaking with increased loudness, persons with PD can easily recognize that their voice sounds within normal limits, despite the feeling of talking in a loud or projected manner (Fox et al., 2002). Fox et al. have speculated that "receptive listening is not impaired; rather, the breakdown may be in online feedback (auditory and proprioceptive) while speaking" (p. 115).

Using the published investigations of sensory function in PD, one can cite compelling evidence of visual, auditory, olfactory, tactile, and kinesthetic deficits associated with the neural degenerative process in PD. Primary sensory symptoms of burning, pain, tingling, or numbing were found in 43 out of 101 (43%) (Snider, Fahn, & Isgreen, 1976) and in 19 of 50 (38%) PD participants (Koller, 1984). Disturbance of olfactory function in persons with PD has also been documented (Quinn, Rossor, & Marsden, 1987; Ward, Hess, & Calne, 1983). These abnormal sensory deficits may be the result of a "release" of sensory centers from an inhibitory control normally mediated by the basal ganglia (Koller, 1984). Neural evidence in monkeys rendered parkinsonian included a documented loss in selectivity of firing of globus pallidus neurons to passive sensory stimulation (Filion, Tremblay, & Bedared, 1988). A decreased threshold of sensory-triggered jaw reflexes in cats with bilateral globus pallidus lesions was reported by Schneider (1987). Decreased sensory-evoked brain activation in persons with PD has been reported, as measured by Positron Emission Tomography (PET) scan data, in cortical parietal, frontal, and temporal areas (Boecker et al., 1999; Liotti et al, 2003).

Proprioceptive deficits are well documented in persons with PD for both limb and speech movements (Connor & Abbs, 1991; Ho, Bradshaw, Iansek, & Alfredson, 1999; Ho, Iansek, & Bradshaw, 1999; Hovestadt, de Jong, & Meerwaldt, 1987; Jobst, Melnick, Byl, Dowling, & Aminoff, 1997; Odlozinski, 1998; Schneider, Diamond, & Markham, 1986; Solomon, Robin, Lorell, Rodnitzky, & Luschei, 1994; Solomon, Robin et al., 2000). Jobst et al. (1997)

compared performance of PD participants and controls on tasks of sensory integration and praxis, finger identification, graphesthesia, localization of tactile stimuli, and kinesthesia. Without visual guidance (e.g., using a task requiring reliance on proprioceptive feedback), PD participants had more difficulty in perceiving the extent of a movement made to a target away from the body. Hovestadt et al. (1987) tested spatial orientation in PD participants and healthy controls, also using a task that relied on proprioceptive feedback. They reported impaired spatial perception occurring in the early stages of the disease. Similarly, Connor and Abbs (1991) compared performance of nonspeech jaw movements performed with and without visual guidance. When PD participants were asked to produce visually-guided nonspeech jaw movements, their jaw movements were significantly slower than those of controls.

Schneider et al. (1986) reported proprioceptive deficits in orofacial function in PD. Such observed deficits included (a) impaired jaw proprioception; (b) deficits in tactile localization for the tongue, gums, lips, and teeth; and (c) inability to accurately perform head tracking movements using perioral somatosensory feedback.

Soloman and colleagues (Solomon & Hixon., 1993; Solomon et al., 1994; Solomon, Robin et al., 2000) reported proprioceptive deficits in a task whereby PD participants were asked to produce tongue pressures in 10% increments from 10% to 100% of their maximum tongue pressure. The PD participants demonstrated difficulty in scaling their actual production of tongue pressure with their perception of effort. For example, one participant with PD consistently

underestimated her sense of effort, even though she had normal tongue strength and endurance levels. Although she was physically able to exert higher tongue pressures, she demonstrated an abnormal perception of effort and produced tongue pressures that were in the lower end of the total range available when instructed to exert high levels of effort.

Odlozinski (1998) reported deficits in scaling speech rate in PD participants. In a series of experiments, she manipulated speech rate control in three conditions that involved rate changes with external cueing (e.g. DAF, metronome pacing, and visual feedback) and one condition of internal cueing (magnitude production, a proprioceptive task whereby participants were asked to increase and decrease their sentence rate relative to a target phrase by a factor of 2 and 4). The PD participants were able to produce a faster rate across all four conditions, which was similar to the rate produced by the controls. Also, they were able to use a slower rate (e.g. 2 or 4 times slower than the habitual rate) comparable to the controls for the three external cueing conditions. However, during the internally cued proprioceptive task that required production of the target phrase at a rate four times as slow as their habitual rate, the PD participants used a faster speech rate than that used by controls, even though they were able to physically produce a slower rate (similar to that used by controls) in the externally cued conditions.

Ho and colleagues (Ho, Bradshaw et al., 1999; Ho, Iansek et al., 1999) investigated perception and production of vocal loudness in persons with PD. Ho, Iansek et al. (1999) assessed volume perception using a listening task. Participants

with PD and healthy controls listened to 15 seconds of taped speech at varying distances and matched the volume of the speech heard by adjusting a volume control slide. Results indicated that both PD participants and healthy controls perceived a decline in speech volume as distance between experimenter and participant increased. However, the participant groups behaved differently with respect to increasing distance. PD participants perceived speech to be louder than healthy controls at increased distances. That is, there was minimal difference between patients' and controls' perception at 1 m. In contrast, as distance increased, PD participants increasingly overestimated volume relative to controls.

While it is evident that persons with PD have a deficit in their capacity to use kinesthetic or proprioceptive feedback to guide limb and speech movements, the mechanism underlying the proprioceptive disorder is not clear. Schnieder (1987) suggested that the basal ganglia may filter sensory information related to movement. In persons with PD, the basal ganglia may no longer be able to effectively filter nonrelevant sensory information (Markham, 1987). Furthermore, timing among relatively remote elements in speech motor sequences is influenced by ongoing sensory input (Abbs & Connor, 1991).

Current models of basal ganglia function hypothesize that the basal ganglia do not appear to have a direct influence on the activity of motor neurons; instead, they may *gate* or regulate the access of sensory information to these motor neurons (Schneider, 1984; Schneider, Watts, Gearing, Brewer, & Mirra, 1997; Schneider, 1987). When the basal ganglia are diseased, certain abnormalities of movement may result from a disturbance of the sensory gating systems of the basal ganglia.

This, in turn, results in abnormal sensory input to motor areas (Boecker et al., 1999; Playford et al., 1992). A defect in sensorimotor integration thus may result in abnormal movements. In the case of PD, loss of nigrostriatal dopamine enhances the inhibitory sensory gating function of the basal ganglia. As a result, there is greater than normal inhibition of the access of sensory information to relevant motor areas (i.e., increased gating), resulting in a net motor hypoexcitability. It is possible that in PD the gating process may shut down and appropriate sensory signals do not gain access to effector regions. With this disruption of normal sensory gating mechanisms, sensory information may not gain access to critical brain areas, resulting in inappropriate responses.

In summary, deficits in sensory and sensorimotor integration are well documented for limb, oral facial, and speech movements. It is evident that when relying on internal cues (e.g. proprioception) and sensory processes to guide their speech motor output, persons with PD misjudge the habitual gain setting and produce a speech volume gain that is too low (Ho, Bradshaw et al., 1999; Ho, Iansek et al., 1999) and a speech rate gain that is too fast (Odlozinski, 1998). Additionally persons with PD are unable to modulate speech volume, rate, or movement amplitude and scale the motor output to achieve normal loudness, rate, or articulatory movement (Odlozinski, 1998; Ramig, Countryman, O'Brien, Hoehn, & Thompson, 1996; Weismer, Kimellman, & Gorman, 1985). The sensory mismatch between the perceived vocal effort and vocal output can present a significant barrier to generalization and maintenance of treatment effects over time (Fox et al, 2002). However, external cues, such as verbal instructions, delayed

auditory feedback (DAF), metronome pacing, and visual feedback improve speech motor performance (Ho, Bradshaw et al., 1999; Odlozinski, 1998. Ramig et al., 1996).

Neuropsychiatric Deficits.

Psychological reactions to PD and psychiatric complications are well documented. Psychological reactions range widely from no response to nuisance, grief reaction, demoralization, discouraged/depressed, and anxiety. Psychiatric complications include depressive syndromes, anxiety syndromes, and medication-related moods and other changes (Starkstein, Berthier, Bolduc, Preziosi, & Robinson, 1989). The reported occurrence of depression or a related mood disorder in PD ranges from 40% to 50% (Zesiewicz, Gold, Chari, & Hauser, 1999). Such mood disorders stem from pre-morbid reaction (Winokur, Black, & Nasrallah, 1988) or be endogenous, which means they are related to the neurogenerative disease process (Fetoni, Soliveri, Monsa, Testa, & Girotti, 1999). Mood disorders are known to affect voice and speech language functions. The loss of prosody in PD could reflect the flat monotonous speech of depression rather than a motor-sensory speech disorder (Darkins, Fromkin, & Benson, 1988). In turn, others have suggested that the mood disorders can be negatively affected by the speech deficits (Murry, Canito, & Woodson, 1990; Sapir & Aronson, 1990). However, Sapir et al. (2001) investigated the relationship between depression and the prevalence and number of voice and speech abnormalities in 42 persons with PD. They reported no significant relationship.

Neuropsychological Deficits.

According to Scharre and Cummings (1994), 40%-50% of individuals with PD experience decreased cognitive function. In some cases these deficits are part of a global process of dementia and are associated with geriatric PD persons (Agid, Ruberg, Dubois, & Pillon, 1987). A wide range of cognitive impairments has been documented in persons with PD who have roughly intact mental status or no obvious evidence of dementia (Gotham et al., 1988). A close parallel exists between these deficits in persons with PD and those found in persons who have damage to the prefrontal cortex. The deficits have been reported in the areas of (a) language (Hanley, Dewick, Davies, Playfer, & Turnbull, 1990; Illes, Metter, Hanson, & Iritani, 1988; Ingvar, 1983; McNamara, Obler, Au, Durso, & Albert, 1992; Natsopoulos, Katsarou et al., 1991; Natsopoulos, Mentenopoulos et al., 1991); (b) memory (Harrington, Haaland, Yeo, & Marder, 1990; Tweedy, Langer, & McDowell, 1982); and (c) concept formation and behavioral regulation (Bowen, Kamienny, Burns, & Yahr, 1975; Cools, Van den Berken, Horstink, van Spaendonck, & Berger, 1984; Flowers & Robertson, 1985; Lees & Smith, 1983).

With respect to language, frontal-type expressive and receptive language deficits include (a) impaired verbal fluency (Hanley et al., 1990; Lees & Smith, 1983); (b) anomia (Matison, Mayeux, Rosen, & Fahn, 1982); (c) hesitant, aprosodic non-fluent speech, similar to that observed in Broca's aphasia (Illes et al., 1988); (d) impaired self-monitoring of speech errors (McNamara et al., 1992); and (e) impaired syntactic comprehension (Lieberman et al., 1992), particularly for comprehension of relative clauses (Natsopoulos, Mentenopoulos et al., 1991) and

before and *after* temporal constructions displaying complex grammar (Natsopoulos, Katsarou et al., 1991).

Memory deficits associated with PD include problems in procedural memory (Harrington, Haaland, Yeo, & Marsden, 1990; Tweedy, Langer, & McDowell, 1982) and reduced working memory capacity, also described as short-term memory (Dalrymple, Kalders, Jones, & Watson, 1994). The range of neuropsychological changes associated with PD are numerous and include the following: (a) Bradyphrenia or slow thinking and slowed cognitive processing speed (Lees, 1994); (b) slow learning (Tweedy, Langer, & McDowell, 1982); (c) problems shifting cognitive sets (Cools, Van Den Bercken, Horstink, Van Spaendonck, & Berger, 1984); and (d) problems internally cueing (Brown & Marsden, 1988, 1990). Lees and Smith (1983) commented that spouses and those closest to the person with PD notice subtle behavioral changes within the first few years of the illness. Such changes can include a slight lack of spontaneity, diminished imagination, lack of initiation, passivity, and a tendency towards repetition. Persons with PD sometimes complain of forgetfulness and occasionally of losing their way in unfamiliar surroundings. These cognitive deficits in non-demented individuals with PD, though apparent in formal neuropsychological testing, may be subtle or undetectable in daily interactions. Fox et al., (2002) stated that, "Although neuropsychological impairment does not necessarily play a role in the voice and speech disorder observed in individuals with PD, cognitive functioning may affect an individual's ability to benefit from speech treatment" (p. 116).

The pattern of cognitive impairment observed in persons with PD is linked to the close anatomical associations between the striatum and the frontal cortex (Brunner, Kornhuber, Seemuller, Suger, & Wallesch, 1982; Ingvar, 1983). That is, the apparent frontal deficits found in PD may be attributed to disruption within the complex loop between prefrontal areas and the basal ganglia (Gotham et al., 1988). However, caution is necessary in distinguishing a true cognitive impairment from poor performance on neuropsychological tests. In PD, factors such as nonspecific age-related decline, impaired motor speech and dexterity, mental and physical fatigability, depressed mood and drug-related confusional states are all potential causes of poor performance on tests.

In summary, there is a high occurrence of neuropsychological deficits in persons with PD that can range from subtle cognitive changes, only detected with neuropsychological testing, to apparent dementia. However, dementia is primarily associated with elderly PD patients and those in the later stages of the disease process. Subtle cognitive deficits may not play a direct role in the voice and speech deficits associated with PD; however, they can affect an individual's ability to benefit from speech treatment.

Young-onset Parkinson's Disease

The terms *young-onset* and *early-onset* PD did not appear in the literature until approximately 15 years ago, and both terms have been used interchangeably. The term young-onset is less ambiguous than early-onset because the latter can refer to persons (irrespective of age) in their early stages of Parkinson's disease (Stewart et al., 1995). To avoid ambiguity, the term *young-onset Parkinson's*

disease (YOPD) is used in the present study to refer to persons who experienced onset of idiopathic parkinsonian symptoms between 21 to 50 years of age.

Young-onset Parkinson's disease appears to be the same nosologic entity as older-onset PD (Golbe, 1991). However, studies comparing the clinical, pathological, and pharmacological differences in young-onset versus late-onset groups have reported differences between these two groups in terms of (a) clinical features (Bostantjopoulou, Logothetis, Katsarou, & Mentenopoulos, 1991; Friedman, 1994; Gibb & Lees, 1988; Rajput, Pahwa, Pahwa, & Rajput, 1993); (b) drug-related response fluctuations (Arevalo, Jorge, Garcia, Scipioni, & Gershanki, 1997; Bostantjopoulou et al., 1991; Friedman, 1994; Giovannini et al., 1991; Golbe, 1991; Kostic, Przedborski, Flaster, & Sternic, 1991; Quinn, Critchley, & Marsden, 1987); (c) disease progression (Gibb & Lees, 1988; Golbe, 1991; Jankovic et al., 1990; Narabayashi, 1995; Rajput, Uitti, Stern, & Laverty, 1986); (d) depression (Jankovic et al., 1990; Kostic et al., 1991; Starkstein, Berthier, Bolduc, Preziosi, & Robinson, 1989; Wagner, Fedak, Sage, & Mark, 1996); (e) dementia (Gibb & Lees, 1988; Giovannini et al., 1991; Quinn, Critchley et al., 1987); and (f) risk-factors (Arevalo et al., 1997; Giovannini et al., 1991; Ludin & Ludin, 1989; Quinn, Critchley et al., 1987; Sandy et al., 1996; Stern et al., 1991).

With respect to clinical factors, persons with YOPD present with rigidity and bradykinesia as the predominant features at onset. In contrast, older-onset persons exhibit more tremor at the beginning, and later in the course they develop the full triad of symptoms (i.e. tremor-rigidity-bradykinesia) (Bostantjopoulou et al., 1991; Friedman, 1994; Gibb & Lees, 1988; Rajput et al., 1993). Among

persons with YOPD, bradykinesia is often the dominant clinical feature (Friedman, 1994). Many have hypothesized the existence of clinical subtypes of PD, including one group presenting with bradykinesia, postural instability, and gait difficulty (PIGD) and another tremor-dominant PD (Bostantjopoulou et al., 1991; Jankovic et al., 1990; Rajput et al., 1993). Therefore, there may be two different clinical syndromes within the YOPD population.

Pharmacological response differences in young-onset versus late onset groups are consistently reported (Arevalo et al., 1997; Bostantjopoulou et al., 1991; Friedman, 1994; Giovannini et al., 1991; Golbe, 1991; Kostic et al., 1991; Quinn, Critchley et al., 1987). Persons with YOPD have a significantly higher frequency of both levodopa-induced dyskinesias and fluctuations, and they develop these symptoms earlier in the disease process than persons with older-onset PD. Because of drug intolerance and increased sensitivity over time, there is a trend to delay the initiation of levodopa treatment for as long as possible in the young-onset patient. Persons with PD build up a tolerance to levodopa over time, forcing increased dosages to receive benefit. However, higher dosages produce greater side effects such as dyskinesia (Arevalo et al.). Also, some people develop an increased sensitivity to the medication, causing side effects to occur with even the smallest dosages (Lieberman & Williams, 1993). It has been argued that some differences found between the YOPD and older-onset group could be related to the delay in treatment and the use of levodopa-sparing strategies (Arevalo et al., 1997).

Persons with YOPD have a more gradual progression of parkinsonian signs and symptoms in comparison with the older-onset group (Gibb & Lees, 1988; Golbe, 1991; Jankovic et al., 1990; Narabayashi, 1995; Rajput et al., 1986). Jankovic et al. (1990) reported that persons with YOPD reached the same level of motor disability as the late-onset PD group but at a significantly slower rate. When older-onset and young-onset groups were matched for disease duration, the younger group demonstrated a significantly higher frequency of depression (Starkstein et al., 1989). Stern et al. (1994) reported differences in frequency of depression of 73% versus 37% in the younger-onset and older onset groups, respectively.

Studies have consistently reported the absence of dementia in persons with YOPD, even when participants from both onset groups were matched for disease duration (Gibb & Lees, 1988; Giovannini et al., 1991; Quinn, Critchley et al., 1987). Ludin and Ludin (1989) reported that YOPD participants performed better on mental testing despite longer disease duration. These investigators also noted that the younger cohort was taking less dopaminergic medication. Therefore, some of the cognitive deficits associated with PD may be secondary to drug effects. However, Giovannini et al. (1991) reported that attention processes are selectively impaired in YOPD persons. Furthermore, impaired performance for finger tapping was noted in this study. Visual and auditory brainstem evoked potential abnormalities also occur in persons with YOPD (Muthane, Satishchandra, & Subhash, 1993). Therefore, not all of the sensorimotor and cognitive disorders associated with older-onset PD are attributable to aging effects.

It is uncertain whether risk factors differ between the young-onset and older-onset groups (Arevalo et al., 1997; Giovannini et al., 1991; Ludin & Ludin, 1989; Quinn, Critchley et al., 1987; Sandy et al., 1996; Stern et al., 1991). Because PD is thought to develop as a result of interactions between genetic susceptibility factors and environmental exposures to agricultural chemicals similar to MPTP (Sandy et al., 1996), investigators have compared familial history of PD and exposure to environmental toxins in young-onset and older onset groups. However, epidemiological and clinical evidence does not suggest that persons with PD have experienced increased exposure to environmental toxins (Arevalo et al., 1997; Giovannini et al., 1991; Stern et al., 1991). Therefore, genetic risk factors may influence disease onset (Sandy et al., 1996), and clinical differences between young and older-onset cohorts may be accounted for by age-related physiologic changes in the basal ganglia.

In summary, literature supports the view that investigations of the signs and symptoms of older-onset PD are confounded by aging and medication effects. Clear differences in the disease process have been noted, particularly with respect to pharmacological response and lack of dementia in persons with YOPD who have had the disease for more than 20 years. Furthermore, reports of a greater frequency of depression associated with YOPD provide evidence that the disability associated with PD has a greater impact on the quality of life for younger individuals (Starkstein et al., 1989).

Speech Disorders.

Although motor, sensory, neuropsychiatric, and neuropsychological deficits negatively impact speech, voice, and swallowing in PD, the neural mechanisms underlying these deficits are not well understood (Fox et al., 2002). Logemann et al. (1978) hypothesized a progression of dysfunction in parkinsonism, beginning with the laryngeal system and subsequently involving the posterior tongue, more anterior portion of the tongue, and finally the labial articulators. They suggested that this progression is related to a predictable pattern of neural degeneration in the somatotopic representation of the speech articulators in the basal ganglia. This hypothesis was further supported by the observation that significant dysphagia (e.g. swallowing impairment requiring dietary changes, gastrostomy, or tracheostomy) has been associated only with the older-onset PD cohort or those who are in the later stages of the disease. Dysphagia in PD is associated with impairment initially involving the pharyngeal stage and followed by the oral phase (Bird, Woodward, Gibson, Phyland, & Fonda, 1992; Bushmann, Dobmeyer, Leeker, & Perlmutter, 1989; Clarke, Gullaksen, Macdonald, & Lowe, 1998; Hartelius & Svensson, 1994; Logemann, Boshes, Blonsky, & Fisher, 1977; Stroudley & Walsh, 1991; Wintzen et al., 1994). Disorders of resonance are uncommon in hypokinetic dysarthria. Hypernasality was identified in only eight of the 32 participants (25%) in the Mayo Clinic study (Darley et al., 1969a) and 20 of 200 participants studied by Logemann et al. (1978). Although hypernasality due to inadequate velopharyngeal closure has been perceived in some persons with

PD (Hoodin & Gilbert, 1989), it is not considered a prominent feature of the speech disorder.

Some investigators have reported respiratory deficits as evident in kinematic observations of speech breathing in PD (de la Torre, Mier, & Boshes, 1960; Murdoch, Chenery, Bowler, & Ingram, 1989; Solomon & Hixon, 1993). Solomon and Hixon (1993) reported that PD participants (a) had smaller rib cage volumes but larger abdominal volumes when initiating breath groups, (b) produced fewer within breath group pauses, and (c) spoke for less time per breath group than healthy controls. Low oral air pressure has been consistently reported in persons with PD by investigators who employed aerodynamic measures (Mueller, 1971; Netsell, Daniel, & Celesia, 1975; Solomon & Hixon, 1993). However, one might interpret these findings as indicative of inadequate valving of the air stream (either phonatory or articulatory) rather than a direct respiratory dysfunction.

There are many reports of imprecise consonant articulation in some but not all persons with PD that primarily appears to affect stop consonants (Ackermann & Ziegler, 1991; Canter, 1965; Connor, Ludlow, & Schulz, 1989; Forrest, Weismer, & Turner, 1989), affricates (Logemann, Blonsky, & Boshes, 1973), and fricatives (Weismer, 1984b). An early report of imprecise consonant articulation in persons with PD noted that stops appeared to be weak and were produced as fricatives (Cramer, 1940, as reported by Canter, 1965). Logemann and Fisher (1981) reported that 45% of PD participants exhibited misarticulations. These investigators provided a detailed description of the speech articulation errors of PD

participants, based on phonetic transcriptions of 90 participants' speech. Their analysis revealed that stops, affricates, and fricatives were often distorted, and that these distortions appeared to be the result of an inadequate narrowing of the oral constriction. That is, stops and affricatives became fricative-like, while fricatives showed a general reduction in frication energy.

Acoustic observations have also provided information regarding the potential acoustic correlates of imprecise consonant articulation in PD speech. Two such acoustic correlates are *spirantization* and *spectral tilt*. Spirantization is the presence of fricative-like, aperiodic noise during stop closures. PD speakers produce an abnormal amount of spirantization, particularly during bilabial stops (Weismer & Fennell, 1985). Spectral tilt refers to the relative distribution of energy (e.g., high versus low frequencies) in the spectra of stop and fricative consonants. PD speakers appear to produce fricatives and affricates such as /s/ and 'sh' with an abnormal distribution of spectral energy. In particular, the fricative spectra obtained from PD speakers show an overall tilt toward the lower frequencies. However, one could argue that these acoustic correlates of imprecise consonant articulation are related to deficits in phonatory control (e.g., inadequate laryngeal valving resulting in a reduced glottal noise source) rather than orofacial impairment.

Several investigators have described abnormalities in the lip, jaw, and tongue movements of PD participants during speech and nonspeech tasks (Caligiuri, 1987; Caligiuri, 1989; Connor & Abbs, 1991; Forrest et al., 1989; Hirose, Kiritani, & Sawashima, 1982; Hirose, Kiritani, Ushijima, Yoshioka, &

Sawashima, 1981; Hunker et al., 1982; Solomon et al., 1994; Solomon, Robin et al., 2000). Kinematic observations have indicated that persons with PD show reduction in the amplitude and peak velocity of jaw and lip movements during speech, relative to healthy controls (Connor, Abbs, Cole, & Gracco, 1989; Forrest et al., 1989). Forrest et al. (1989) indicated that jaw movements for PD participants were, on average, approximately one-half the size of the jaw movements observed in controls. Interestingly, the durations of jaw movements during speech produced by PD participants and healthy controls were not significantly different.

Examination of strength in the orofacial system of people with PD has been reported in a few studies. Observations of electromyographic activity and assessment of maximum force generally have reported reduced strength of the lip (Netsell et al., 1975; Wood, Hughes, Hayes, & Wolfe, 1992) and tongue (Solomon et al., 1994; Solomon, Robin et al., 2000). Although the physiological mechanisms responsible for these speech problems are not known, the strength and endurance abnormalities in the orofacial musculature may contribute to articulatory impairment associated with PD.

Vocal Dysfunction.

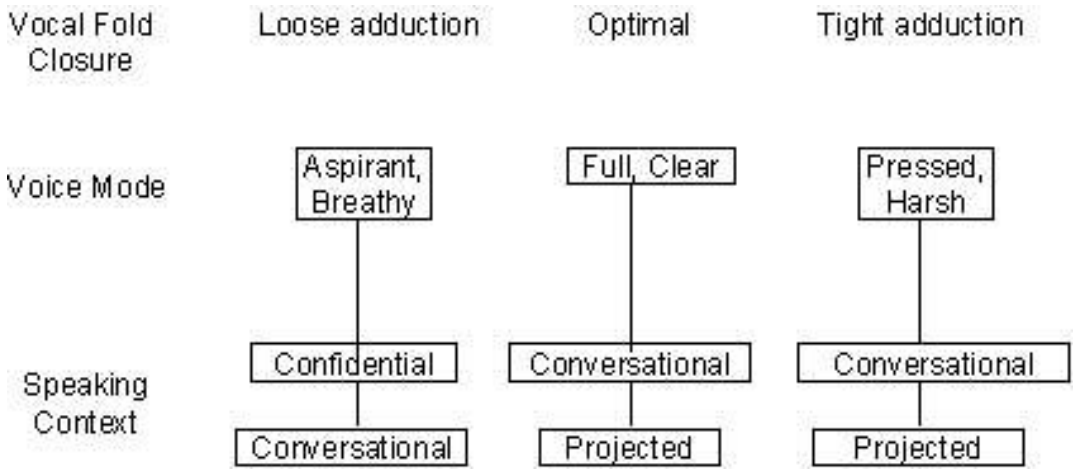
Logemann and colleagues hypothesized that voice is the most prominent disorder and the first to appear in PD disease progression, and this has been corroborated by others (Aronson, 1990; Hartelius & Svensson, 1994; Sapir et al, 2001). Sapir et al (2001) suggested that one explanation for the early appearance of voice disorders in PD might be that the neural mechanisms underlying disorders

of vocalization are different from those underlying articulation, fluency, and prosody. Specifically, vocalization is a phylogenetically older system involving limbic (e.g., anterior cingulate cortex), brainstem (e.g., periaquaductal gray) and other subcortical systems (Holstege & Ehling, 1998), whereas articulation, fluency, and prosody are more related to linguistic and cognitive functions, that are subserved by neocortical mechanisms (Brown, 1975; Dingwall, 1979; Murdock, 2001). The progression of voice and speech deficits may reflect the progression of the neurodegenerative disease process of the initial disturbance occurring in subcortical regions, with later progress to pre-frontal and high level mechanisms. Typical vocal manifestations include (a) *breathy* or *rough voice quality* (Logemann et al.; Hanson, Gerratt, & Ward, 1984); (b) reduced loudness (*hypophonia*) (Ramig et al., 1996); (c) *voicing control deficits* that include difficulty producing rapid phonatory offset-onset during speech (Ackermann & Ziegler, 1991; Canter, 1965; Forrest et al., 1989; Kent & Finley Kent, 2000); and (d) reduced pitch inflection and reduced control in altering the duration of speech segments (*hypoprosodia*) (Caekebe ke, Jenneknes-Schinkel, van der Linden, Buruma & Roos, 1991; Robertson & Thompson, 1984; Scott & Caird, 1984). Speech timing deficits involving speech rate disturbances and the use of longer and more frequent pauses have been reported by several investigators (Hammen & Yorkston, 1996; Metter & Hanson, 1986; Pitcairn, Clemie, Gray, & Pentland, 1990; Solomon & Hixon, 1993), and they are most likely related to phonatory deficits. Vocal tremor may also occur but is frequently not audible (Gath & Yair, 1988).

Voice Quality Deficits.

Voice quality is not easily defined (Kent & Finley Kent, 2000). The term usually denotes the auditory impression that a listener perceives upon hearing the speech of another talker (Childers & Woo, 1990). Speakers use a variety of voice qualities, and each speaker can change his or her voice quality (Ananthapadmanabha, 1995). An important determinant of voice quality is the *setting* of laryngeal posture. All speakers use various settings of laryngeal posture, biasing the posture of the larynx to a given *mode* of vocal fold vibration (Laver, 2000; Laver & Hanson, 1981; Sundberg, 1994). The speaker develops his or her settings. That is, habits of particular muscular tensions are acquired and used automatically (Abercromie, 1967). Figure 1 illustrates the range of settings of laryngeal postures (for modal phonation) that can vary from incomplete vocal fold closure during phonation (loose adduction) to an overly tight medial compression (tight adduction), as adapted from a model proposed by Colton and Casper, 1996. The optimal setting involves a balance, whereby the vocal folds are barely touching during the closed phase of the glottal cycle. This setting provides for optimal glottal source power, resulting in maximal glottal efficiency (Titze, 1994; Verdolini , Druker, Palmer, & Samawi, 1998). Titze (1994, p. 227) states, “In the continuum of pressed voice (tight voice) to breathy voice (loose adduction), the optimal adjustment for vocal power seems to be near the center, but barely toward the breathy side.”

Figure 1. Range of laryngeal postures.



As illustrated in Figure 1, the degree of vocal fold closure also determines the type of voice mode produced. Incomplete closure or loose adduction results in an excessive flow of air though the glottis and is perceived as a *breathy* or *aspirant* voice. Tight adduction results in a *pressed* or *harsh* voice. Pressing the vocal folds together too hard or separating them too widely will limit their effectiveness as an acoustic power source. Ultimately vocal fold vibration ceases (devoicing) at both extremes because it cannot be sustained if the vocal folds are either too widely or too tightly approximated (Verdolini et al., 1998).

Speakers use different laryngeal settings to meet varying speaking situations or contexts. Laver (2000) defines the *neutral phonatory setting* as a theoretical construct. The neutral setting is not necessarily normal or a rest position of the larynx. Rather, it represents the setting where there is average or optimal degree of muscular tension throughout the larynx. Healthy speakers use a

predominately neutral setting; however, they vary their phonatory setting according to the communication need of the speaking context (Ohala, 1981). The neutral, optimal setting results in a *full, clear voice* that is used for conversational speaking situations and for projecting the voice (e.g., public speaking, or calling someone from a distance). Healthy speakers use an *aspirant* or *breathy* voice in situations when they wish to speak quietly (but not whisper) in settings such as libraries or private conversations. This voice is referred to as the *confidential voice* (Casper, 2000; Casper, Colton, & Brewer, 1989; Casper, Colton, & Woo, 1990; Colton & Casper, 1996). Also, some healthy speakers use a habitually breathy conversational voice as a social marker of female gender among English speakers (Hillenbrand, Cleveland, & Erickson, 1994; Klatt & Klatt, 1990). A small posterior opening or *glottal chink* is commonly noted in normal female speakers undergoing laryngeal stroboscopic examination.

The *pressed*, *harsh* voice mode can be observed occasionally in healthy speakers who are speaking in a conversational context or projecting their voice and when speaking under the influence of emotional situations of anger, fear, stress, or anxiety (Colton & Casper, 1996). A pressed voice refers to an impression of a tight, strained voice. A harsh voice is considered a deviant voice quality. Consistent use of the pressed, harsh mode is viewed as a hyperfunctional voice disorder, resulting in the perception of a voice that sounds rough, grating, unpleasant, and harsh.

Measures of voice quality include subjective perceptual judgments and objective, corroborative acoustic measures. The latter are referred to as voice

spectra measures (Kent & Finley Kent, 2000; Kent, Kent, Duffy, & Weismer, 1998; Kent, Vorperian, & Duffy, 1999). Darley, Aronson, and Brown (1969) developed a perceptual classification system to describe the deviant voice quality characteristics associated with neurological speech disorders (such as *dysarthria*). The deviant characteristics associated with Parkinson's disease are *breathiness* and *harsh*/*roughness*.

The reported occurrence of breathiness in the voice of PD speakers is similar across studies, occurring at a rate of 15-17% of participants tested, regardless of medicated or nonmedicated testing status (Logemann et al., 1978; Ludlow & Bassich, 1984). It is not certain if breathiness associated with vocal dysfunction is similar or different from normal breathiness (Alku & Vilkman, 1996; de Krom, 1994; Eskenazi, Childers, & Hicks, 1990; Javkin & Kaun, 1991; Shoji, Regenbogen, Yu, & Blaugrund, 1992).

In contrast to the consistent occurrence of breathiness observed in PD speakers, the occurrence of harsh/roughness varies across studies, possibly due to differences in the definition of this perceptual attribute. Logemann et al. (1978) reported hoarseness in 45% and roughness in 29% of their participants, while Ludlow and Bassich (1984) found that 83% of their participants had abnormally harsh voices. However, if the hoarseness and roughness data are combined in the Logemann et al. (1978) study, the occurrence of rough voice is consistent with that noted by Ludlow et al. That is, roughness occurs in at least 74% of persons with PD. Importantly, these studies have used a variety of speech tasks to judge the perceptual voice characteristics. The tasks have included sustained phonation,

diadochokinesis, counting, and reading. Conversational speech samples have typically not been used for rating perceptual dimensions. It is possible that task differences may account for the discrepant findings of harshness across studies. Nevertheless, roughness, harshness, and hoarseness are prominent features in the voices of persons with PD.

Voice spectra measures that correlate with perceptual dimensions can only be derived using sustained vowel phonation tasks (Kent et al., 1999), and there are a number of software systems available for voice spectra analyses (Kent & Finley Kent, 2000). Spectral measures of *harmonic to noise ratio* (HNR) and *spectral tilt* correlate with breathiness. Air leakage through the glottis creates turbulence, which results in aspiration noise perceived as breathiness. Acoustic studies have demonstrated that the amplitude of the first harmonic correlates moderately with breathiness ratings (Hillenbrand, Cleveland, & Erickson, 1994). Also, breathiness is associated with reduced harmonic energy in high frequencies, referred to as increased spectral tilt (Hillenbrand et al.; Klatt & Klatt, 1990). de Krom (1995) reported that the harmonics-to-noise ratio was the best single predictor of breathiness. Again, task differences (e.g., sustained vowel productions versus connected speech) may account for the perceptual differences reported across studies.

Measures of short-term acoustic perturbations (jitter and shimmer) correlate with harshness (Takahashi & Koike, 1975; Yumoto, Gould, & Baer, 1982). These measures reflect a short-term instability or variability in the cycle-to-cycle period length (jitter) or amplitude (shimmer). Perturbation measures are

consistently identified as deviant in persons with PD (Gamboa et al., 1997; Kent & Finley Kent, 2000; Kent et al., 1999; Larson, Ramig, & Scherer, 1994; Ramig et al., 1996), whereas measures of noise-to-harmonic ratio are not consistently noted as deviant (Kent et al., 1999). This correlates with the observation in perceptual studies that roughness is observed in a larger percentage of the PD participants than is breathiness.

In summary, breathiness is a phonatory mode used in specific settings by healthy speakers, and it is also a voice quality associated with the voice of PD speakers. There are no studies comparing the similarities of breathy voice of healthy speakers with the breathy voice quality observed in speakers with PD. Possibly, breathy voice associated with PD speakers represents a change in the habitual mode or setting of the phonatory system. That is, PD speakers consistently adjust their habitual setting or mode to one that healthy speakers use when they are using a quiet or confidential voice. This hypothesis is further supported by the observation reported by Ramig and colleagues (Fox & Ramig, 1997; Ramig, Bonitati, Lemke, & Horii, 1994) that unlike the typical person with a hypophonia secondary to vocal fold paralyses or paresis, PD speakers can increase vocal intensity rather quickly once verbally cued to "Think loud!"

Hypophonia.

Reduced loudness or hypophonia is generally recognized as one of the predominant voice symptoms in PD (Canter, 1963; Darley et al., 1969a; Ramig et al., 2002) and occurs in the early stages of the disease (Stewart et al., 1995; Tetrud, 1991). While researchers who have completed perceptual studies have typically

described parkinsonian speech as being reduced in volume (Ackermann & Ziegler, 1991; Darley, Aronson, & Brown, 1969b), not all researchers have reported reductions in vocal loudness or intensity (SPL). For reading tasks, some investigators have reported differences in vocal SPL (Fox & Ramig, 1997; Illes et al., 1988) while others have not (Boshes, 1966; Ludlow & Bassich, 1984; Metter & Hanson, 1986). Fox and Ramig (1997) reported that PD speakers exhibited significantly reduced vocal SPL (ranging from 2-4 dB SPL) in comparison with healthy controls for speech tasks of sustained phonation, reading, and conversation. Ludlow and Bassich (1984) reported that only 42% (5/12) of their PD speakers were perceived as having reduced speech loudness. However, differences in vocal loudness reported across studies may be related to task differences or instructions to the participants. That is, when persons are externally cued to speak in a mode that is different than normal, they are more attentive as to how they execute their speech, thereby increasing vocal loudness (Ho, Bradshaw et al., 1999).

Baker, Ramig, Sapir, Luschei, & Smith (2001) assessed the effects of varying loudness on laryngeal and articulatory muscle activity in two PD participants. EMG recordings were obtained from laryngeal muscles (i.e. thyroarytenoid, cricothyroid) and articulatory muscles (anterior belly of the diagastric and orbicularis oris inferior) while repeating a phrase for three conditions of vocal loudness (soft, comfortable, and loud). Vocal intensity at soft, comfortable, and loud conditions was associated with mean SPL of 60.5, 66.5, and 82.7 dB (30 cm), respectively. The difference between the loud and comfortable

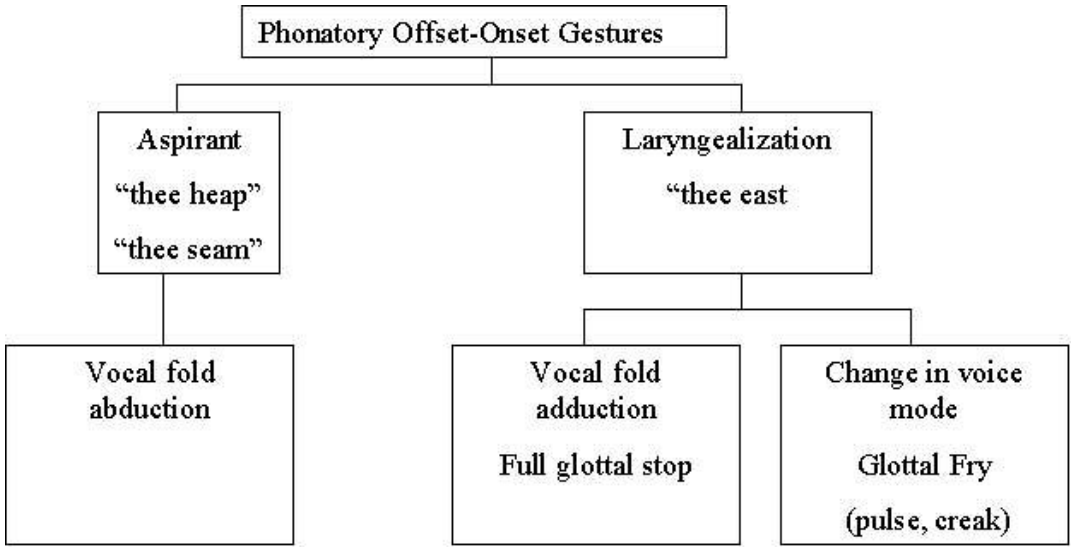
conditions was 16.2 dB SPL and between the soft and comfortable conditions was 6 dB SPL. Both of these differences were significant and indicated that persons with PD were able to increase vocal intensity for a phrase repetition task when externally cued with instructions. For both participants, loud speech was associated with a significant increase in laryngeal and articulatory muscle activity in comparison with comfortable speech; whereas soft speech resulted in no significant change in muscle activity, with no clear pattern within or across participants. Electromyographic variability was lower for all muscles in both loud and soft conditions compared to comfortable loudness. Furthermore, intentional changes in loudness were accompanied by unintentional lengthening of phrase duration. The reduction in muscle variability associated with loud speech observed in this study is congruent with previous findings, suggesting that loud phonation may help increase motor stability across the speech system. Studies reported by Dromey and his colleagues (Dromey & Ramig, 1998; Dromey, Ramig, & Johnson, 1995) found reduced variability in articulatory movements with loud speech compared to normal speech in both healthy speakers and in speakers with PD. An interesting finding of this study was the observation that the laryngeal and articulatory muscles showed either no change in activity or an increase in activity relative to the comfortable condition. One might predict that these muscles would decrease activity in the soft condition. This finding suggests a change in the habitual postural setting of the larynx rather than a single parameter of loudness.

Phonatory Offset-onset Deficits.

The production of connected speech involves a delicate balance of setting the vocal folds into vibration, momentarily interrupting the vibration, and then resuming vibration. These laryngeal articulatory gestures are referred to as phonatory *offset-onset*, and these gestures are required to produce voiceless consonants and to mark word boundaries in certain phonetic contexts. Studies have suggested that persons with PD have deficits in controlling voice-offset gestures.

Laryngeal behavior for phonatory offset-onset was described as "devoicing" in healthy speakers over 30 years ago (Collier, 1979; Hirose, 1974; Ladefoged, 1973; Lisker, 1967; Lisker, 1984; Lofqvist, 1984; Priestly, 1976) and can be achieved using two distinct laryngeal mechanisms, *aspiration* and *laryngealization* (Sawashima & Hirose (1983); Ludlow, Sedory, & Fujita, 1991).

Figure 2. Summary of phonatory offset-onset gestures used by healthy speakers.



As shown in Figure 2, voice offset is produced either by vocal fold abduction in the phonetic context of /isi/ (*aspiration)* or by adduction during the context /i?i/ (*laryngealization*, where the symbol *?* denotes a glottal stop) (Gauffin, 1977; Sawashima & Hirose, 1983; Smith, 1968). Further, the laryngealization gesture has three variants. First, it can be produced as a full glottal stop, with no vibration of the vocal folds for 25 ms to 200 ms. Second, the gesture can involve an abrupt change in voice mode referred to as glottal fry or creaky voice or pulse mode (Hollien, 1974; Stager, Bielamowicz, Regnell, Gupta, & Barkmeier, 2000). This mode change involves an abrupt shift to a super-low fundamental frequency, where vocal fold vibration momentarily slows to approximately 7-78 Hz in males and 2-78 Hz in females (Hollien, 1974). Third, Hillebrand and Houde (1996) found that a change in fundamental frequency and/or amplitude was sufficient for the perception of intervocalic glottal stops.

Aspiration Gesture.

The aspiration gesture is a well-studied mechanism involved in phonatory offset-onset and occurs frequently in the English language (Cole & Cooper, 1975; Denes, 1955; Haggard, 1978; Kim, 1970; Klatt, 1975; Stevens, Blumstein, Burton, & Kurowski, 1992). As Denes (1955) describes, most English consonants can be arranged into pairs such that one member of the pair is *voiced* while the other is *voiceless* The oral articulation of the paired sounds is the same. The two sounds are heard as voiced or voiceless "because the vocal cords do or do not vibrate during oral articulation, and that correspondingly the speech wave spectra will be

harmonic or inharmonic" (p. 761). The aspiration gesture involves a brief opening of the vocal folds from the midline position of phonation to an approximate intermediate position or partially abducted. The vocal folds are sufficiently apart to preclude vibration, therefore voicing is stopped but airflow (in relation to aspiration or frication) continues because of the partially opened glottis.

The physiological mechanisms underlying phonatory offset-onset associated with the aspiration gesture have been widely investigated in healthy speakers (Hillel, 2001; Hirose & Gay, 1973, Hutters, 1985; Kim, 1970; Lofqvist & McGarr, 1992; Lofqvist, McGarr, & Honda, 1984; Odhe, 1984). Additionally, it appears that there are two mechanisms. One involves reciprocal activity of the laryngeal adductory and abductory muscles. That is, there is a simultaneous suppression of the adductory interarytenoids (INT) and activation of the abductory posterior cricoarytenoid (PCA) muscle (Hillel, 2001; Hirose & Gay, 1973; Lofqvist & Baer.,1984; Sawashima & Hirose, 1983). A second mechanism involves an adjustment in the length and tension of the vocal folds. In an electromyography study, Lofqvist and Baer (1989) reported that the cricothyroid muscle (CT) increased activity for voiceless consonants. Hence, the aspiration gesture appears to be associated with increased longitudinal tension of the vocal folds that likely contributes to the cessation of vocal fold vibration. This laryngeal tension may be used to supplement the reciprocal activity of the INT and PCA muscles, involved in the abductory movement.

The two mechanisms involved in the aspiration gesture may also include an active mechanism related to laryngeal muscle activity. However, Stevens (1991,

1999) posited a passive mechanism of voicing control that has been corroborated by Boucher and Lamontagne (2001). In this model, intraoral air pressure (IOP) increases are created when there is a partial or complete oral articulatory constriction or closure. The increased IOP creates decreased transglottal airflow, and this drop in transglottal airflow is sufficient to account for voice stoppage. Therefore, healthy speakers may achieve phonatory offset-set for the production of voiceless obstruents (stop-plosives, fricatives, and affricates) by using (a) an active mechanism involving activity of the laryngeal musculature; or (b) a passive mechanism involving increased IOP, thereby decreasing transglottal airflow. In the production of the voiceless glottal fricative /h/, speakers must use the active mechanism. However, for voiceless alveolar fricative /s/ production, speakers can use the passive mechanism.

Laryngealization Gesture.

Like the aspiration devoicing gesture, the laryngealization devoicing gesture is used in some languages to provide phonologically contrastive words. Words can be distinguished by the absence or presence of a full glottal stop or a glottalized voice quality (Werner-Kukuk & von Leden, 1970). However in English, laryngealization occurs with regularity to provide segmentation of word boundaries (Pierrehumbert, 1995; Umeda, 1978). Speakers use this devoicing gesture as a common mark of word junction in phrases such as *known ocean*, which is perceived as *no notion* when the devoicing gesture is not produced. This devoicing gesture also marks word boundaries. For example, a glottal stop is often inserted between a final vowel sound in a word and the next word when it also

begins with a vowel, as in the utterance *she eats*. Failure to produce a full glottal stop or a laryngealization might result in perceptual confusion between the two-word phrase *she eats* and the one-word utterance*sheets* (Allen, 1970; Hillenbrand & Houde, 1996; Nakatani & Dukes, 1977; Priestly, 1976; Umeda, 1978). Pierrehumbert (1995) and Stager et al. (2000) reported the use of the laryngealization devoicing gesture when it is needed to mark word boundaries as a substitution for production of voiceless stops. For example, the contrast in allophony between "nitrate" (the first /t/ is produced as affricate) and "night-rate" (the first /t/ is glottalized), thereby losing its coronal articulation.

Umeda has noted that the use of the laryngealization devoicing gesture varies across speakers. Listeners judged those speakers who used frequent laryngealizations or glottal stops to mark word boundaries as having greater fluency and clarity in comparison with speakers who infrequently produced this devoicing gesture.

Glottal stops are also typically inserted between the syllable boundaries of repeated syllables (e.g., ee-ee-ee). In the case of a syllable chain such as "ee see ee see …", glottal stops are usually inserted between the repetitions of adjacent vowel syllables.

The mechanical activity that contributes to the production of the glottal stop is uncertain. Using flexible videoendoscopy, Stager et al. (2000) reported the co-occurrence of false vocal fold compression with the production of glottal stops, suggesting that glottal stops are associated with a quick dampening of the vibration of the true vocal folds, resulting in reduction of sound pressure level (also see

Fisher-Jorgensen, 1989). Ludlow, Sedory, and Fujita (1991) investigated adduction and abduction for phonation onset during speech using electromyography (EMG). They reported that the thyroarytenoid (TA), an adductory muscle, was active during glottal stop production, but not active for vocal fold adductory gestures involving movement from the paramedian position into the phonatory position. In summary, these studies support the view that the production of glottal stops primarily involves the use of laryngeal muscles.

Researchers have not systematically investigated production of both the aspiration and laryngealization devoicing gestures in speakers with PD. But there are anecdotal reports suggesting deficits are present for both devoicing gestures. Acoustic investigations find that PD persons have deficits in producing the aspiration devoicing gesture, as they: (a) fail to *cut-off* phonation for production of voiceless consonants in inter-vocalic contexts (Canter, 1965); (b) use continuous voicing in that voiceless segments are brief in duration or replaced by voiced segments (Ackermann, Grone, Hoch, & Schonle, 1993; Kent & Rosenbek, 1982; Kruel, 1972; Lieberman et al., 1992); (c) maintain or continu vocal fold vibration for more than 20 ms into a voiceless stop consonant constriction (Weismer, 1984); and (d) use longer voice onset times (Forrest et al., 1989). Furthermore, persons with PD have difficulty producing the laryngealization gesture. Kent and colleagues (Kent et al., 1994) investigated segmental (phonetic) aspects of laryngeal function using a word-identification task that contrasted 19 phonetic features. Participants included 22 males and 9 females with PD. All males with PD evidenced high error rate for the “at” vs. “hat” contrast. The investigators

noted low error rates for the other feature contrasts for all but two of the males. In contrast, the female group did not demonstrate a high error rate for the "at" vs. "hat" contrast. Therefore, there may be gender differences with respect to phonatory offset-onset control deficits.

Previous studies have not systematically manipulated phonetic context to determine the nature of the laryngeal voice offset control deficit in Parkinson's disease. Therefore, it is uncertain whether these persons have (a) more difficulty producing the aspiration devoicing gesture, (b) more difficulty producing the laryngealization devoicing gesture, or (c) similar difficulty producing both devoicing gestures.

Hypoprosodia.

The term prosody refers to "all the variations in time, pitch and loudness that accomplish emphasis, lend interest to speech, and characterize individual and dialectal modes of expression" (Darley et al., 1969a, p. 5). Kent and Read (1992) have noted that prosody encompasses the suprasegmental aspects of speech, indicating that the phenomena of interest are not confined to phonetic segments. Rather, they are observed over larger intervals such as syllables, words, phrases, sentences, and discourse. Prosody includes intonation (e.g., the patterns of pitch rise and fall, loudness variations, and the patterns of stress in a given language) and tempo (e.g., pause and lengthening). Ohde and Sharf (1992) note that stress, intonation, duration, word juncture, rhythm, and tempo are all features associated with prosody.

Perceptual observations of parkinsonian speech characteristics have emphasized the prominence of prosodic disturbance in PD, noting that monopitch and monoloudness ranked in the top three deviant speech characteristics of persons with PD (Darley et al., 1969b; Ludlow & Bassich, 1984). Measurements of F_o and the variability of F_o have provided acoustic corroboration of monopitch PD (Caekebeke, Jennekens-Schinkel, van der Linden, Buruma, & Roos, 1991; Darkins, Fromkin, & Benson, 1988; Hertrich & Ackermann, 1993; Le Dorze, Ryalls, Brassard, Boulanger, & Ratte, 1998; Ludlow & Bassich, 1984; Metter & Hanson, 1986). Persons with PD have been reported to have a reduced range and variability of F_o during connected speech (Canter, 1963; Ludlow & Bassich, 1984). In Canter's 1963 study, the PD participants demonstrated a smaller pitch range the age-matched control group. Similarly, Ludlow and Bassich (1984) reported limited fundamental frequency ranges in 75% (9/12) of their participants with PD during a pitch glide task. Similar reductions in F_o change were measured on imitation of sentence intonation patterns (7/12 participants) and on the use of fundamental frequency change to contrast compound nouns (e.g., bluebell) with two equally stressed words (e.g., blue bell) in 5 of 12 participants.

Acoustic measures of intensity variability for stress contrasts have indicated no significant differences between PD participants when compared with healthy controls (Canter, 1963; Metter & Hanson, 1986). However, Metter and Hanson (1986) commented that speakers with PD have reduced intensity variation with increased severity of the disorder. Age differences in intensity variability have been noted in studies that compared older and younger healthy speakers. Less

intensity variation has been observed in older healthy speakers. Therefore, the lack of intensity variation differences in older PD persons may be attributable to aging (Brown, Morris, Hicks, & Howell, 1993; Morris & Brown, 1994; Ptacek et al., 1966).

Temporal aspects of speech are impaired in PD participants. Persons with PD produce fewer words and spend less time producing speech per breath group, (Hammen & Yorkston, 1996; Hammen, Yorkston, & Beukelman, 1989; Metter & Hanson, 1986; Pitcairn et al., 1990; Solomon & Hixon, 1993; Tjaden, 2000; Volkmann, Hefter, Lange, & Freund, 1992). Pause characteristics have been reported as deviant and highly variable across PD participants. Acoustic measures of pause durations indicate that PD participants use more frequent and shorter pauses that occur most often at syntactically inappropriate locations, in comparison to speech pauses produced by healthy controls (Darkins et al., 1988; Hammen & Yorkston, 1996). Speakers with PD who have difficulty with rapid phonatory offset-onset control may compensate by using a greater number of pauses (Boshes, 1966; Hammen et al., 1989; Illes et al., 1988; Pitcairn et al., 1990). Pitcairn, Clemic, Gray, and Pentland (1990) found that persons with PD used six times as many pauses in comparison with healthy controls during conversation. Boshes (1966) reported that during a reading passage of 99 words, the speakers with PD used an average of 15 pauses, whereas healthy controls averaged 9 pauses.

Speaking rate is a measure of the amount of speech produced per unit time and frequently is expressed in syllables per second or words per minute (Tjaden, 2000). Rate is determined by both the amount of time a person spends speaking as

well as the amount of pause time. A pause is defined as some minimum time period in the speech stream that is silent (unfilled pause) or contains nonspeech noise, such as inhalation or fillers (filled pause) (Tjaden, 2000). Pause time, a function of the duration and frequency of pauses, is typically computed by summing the durations of the pauses in a speech sample (Hammen & Yorkston, 1996; Miller, Grosjean, & Lomanto, 1984; Tsao & Weismer, 1997). Perceptually, persons with PD give listeners the impression that they are speaking at an uncontrollably fast rate referred to as short rushes (Darley et al., 1969a). However, even persons who are not speaking in short rushes give listeners the impression of a faster speech rate (Kent & Rosenbek, 1982; Weismer, 1984b). Yet, quantitative studies using acoustic or physiologic measures of duration have reported variable results. Speech rate is reported as (a) reduced (Weismer, 1984a), (b) more variable than normal, (Canter, 1963), (c) faster than normal (Illes et al., 1988), or (d) essentially similar to that of healthy speakers (Ackermann et al., 1995; Hertrich & Ackermann, 1993). The discrepancies observed within these quantitative studies could be attributed to the particular speech samples analyzed or the method of calculating speech rate. Investigators reporting slower speech rates for PD participants (Weismer, 1984a) included pause time in their rate measure, whereas studies that have reported the speech rate as fast (Illes, 1989) have excluded pause time. For example, Weismer (1984a), who reported slow rate, used a sentence repetition task, whereas Illes (1989) reported a faster rate using a conversational task. Therefore, the perceptual impression of speech rate in persons with PD is multidimensional in that it is influenced by segment duration cues as well as

pauses. Tjaden (2000) has suggested that perceptual impressions of articulatory rate in persons with PD may overestimate the actual physical rate.

The mismatch between the perceptual impression of accelerated speech and acoustic findings of normal or slowed rate may also be attributable to factors other than the temporal properties that influence a listener's perception of speech rate. For example, speech utterances produced with a small amount of fundamental frequency (F_o) variation (i.e. monotonous speech) tend to be perceived as more rapid than speech produced with a relatively large amount of F_o variation (den Os, 1985). Second, acoustic measures of formant transition rates showed a reduction in PD speaker's rate in both isolated and repeated syllables, suggesting that movement accuracy is reduced (Connor, Ludlow et al., 1989). Furthermore, physiologic measures of movement duration and amplitude suggested that articulator movement durations are normal or slow, however the amplitude of movement is reduced, resulting in articulatory undershoot. The decreased movement accuracy during speech production may blur acoustic contrasts, giving the impression of an abnormally fast rate (Kent & Rosenbek, 1982). In summary, there are conflicting results in the literature regarding the subjective judgment of a speech rate in PD speakers and the objective physiologic and acoustic studies, suggesting that physical articulatory rate may be slow.

Researchers have questioned if there is an underlying disorder of processing emotional information (e.g. detecting facial expressions or voice attributes that signal angry, happiness, sadness, etc.) that might account for the prosodic disturbances noted in PD speakers (Benke, Bosch, & Andree, 1998;

Blonder, Gur, & Gur, 1989; Borod et al., 1989; Caekebeke et al., 1991; Dalby, 1994; Darkins et al., 1988; Flint, Black, Campbell-Taylor, Gailey, & Levinton, 1992; Scott & Caird, 1984; Scott, Caird, & Williams, 1984). However, there is no evidence of impaired emotional processing in persons with PD. Investigators have reported that PD participants displayed normal emotional receptive processing abilities. In comparison with healthy controls, PD participants (a) appreciated vocal and facial expression (Benke et al., 1998; Caekebeke et al., 1991); (b) comprehended linguistic stress contrast (Darkins et al., 1988); and (c) experienced similar emotional valence and arousal to that of controls when confronted with emotional visual stimuli (Dalby, 1994).

Although comprehension of linguistic stress and appreciation of vocal and facial expression appears intact in most persons with PD, performance for expressive correlates to these tasks (e.g. production of vocal and facial expressions to portray anger, sadness, happiness, etc.) is consistently reported by investigators as impaired (Benke et al., 1998; Blonder et al., 1989; Borod et al., 1989; Caekebeke et al., 1991; Dalby, 1994; Darkins et al., 1988; Flint et al., 1992; Scott & Caird, 1984; Scott et al., 1984).

In summary, hypoprosodia does not appear to be related to a disorder of processing emotional information nor a lack of knowledge of linguistic rules necessary to differentiate syntactic structures (e.g., to distinguish between a noun phrase *green house* and a noun compound *greenhouse*). The underlying deficits seem to involve a difficulty in maintaining the rhythmicity of speech or reduced

ability to control or manipulate the duration of speech events so as to effect rate and stress differences (Ludlow, Connor, & Bassich, 1987).

Pathophysiology of Laryngeal Function

The pathophysiology of laryngeal function that is responsible for the phonatory abnormalities of hypophonia, voice quality deficits, rapid phonatory offset-onset deficits, and hypoprosody associated with PD has not been adequately described (Baker, Ramig, Sapir, Luschei, & Smith, 1998; Hanson et al., 1984). Importantly, the vocal dysfunction may be explained by abnormalities observed in the phonatory posture of the laryngeal structures. As observed under laryngoscopy, glottal incompetence with vocal fold bowing is often found in some persons with PD and would account for breathy voice quality, due to increased escape of air though the glottic gap (Hanson et al.). Smith, Ramig, Dromey, Perez and Samandari (1995) noted bowing in 12 of 21 (57%) PD participants. Hanson et al., (1984) reported bowing in 30 of 32 (94%) patients, and the two patients without bowing were without voice complaint and were judged clinically as having normal voices. Vocal fold bowing is described as a tight approximation of the vocal processes during vibration and a significant, visible, posterior glottic gap. Hanson et al. also noted a "crossing of the vocal processes" (i.e., one vocal process moved under the other in some patients) when using increased intensity. Hanson et al., (1984) noted that the appearance of the bowed vocal folds in PD patients was not typical of that seen with paralysis subsequent to laryngeal nerve damage. There are some reports of bilateral vocal cord paralysis in Parkinson's disease (Corbin & Williams, 1987; Isozaki et al., 1995; Plasse, Abraham, & Lieberman, 1981; Read

& Young, 1983; Sanders & Thomsen, 1995) leading to stridor and upper airway obstruction. However, the occurrence of stridor and upper airway obstruction is rare and appears to be a manifestation of advanced disease and perhaps aging (Plasse et al., 1981).

Although the mechanism of bowing associated with Parkinson's disease is uncertain, one could speculate that it is related to reduced TA activity reported in recent EMG studies of PD participants (Baker et al., 1998; Luschei et al., 1999). Tanaka, Hirano, and Chuiwa (1994) reported that the occurrence of vocal fold bowing in 127 patients with vocal fold paralysis, sulcus vocalis, or laser surgery was closely related to reduced activity of the TA muscle.

Supraglottic constriction (involving a squeezing of the ventricular folds) has been noted in some PD speakers who were observed to have bowed vocal folds (Countryman, Hicks, Ramig, & Smith, 1997; Hanson et al., 1984). Countryman et al. (1997) suggested that the supraglottal constriction was due to a compensatory behavior resulting from the bowing of the true vocal folds.

Aberrant vocal fold vibration and reduced laryngeal reaction time have been reported for PD speakers. As noted in glottographic observations, abnormalities in the vibratory behavior of the vocal folds (Hanson, Gerratt, & Ward, 1983; Lin, Jiang, & Hanson, 1999) have included an increased time for vocal fold opening relative to vocal fold closing, whereas reported videostroboscopic observations have described an abnormal phase closure and phase asymmetry (Perez et al., 1996). Jiang, et al., (1999) found that laryngeal reaction time (i.e., the time between stimulus and onset of phonation) was

significantly slower in comparison with healthy age-gender matched controls. Jiang et al. also developed a vocal equivalent of movement time (i.e., time from voice onset to reaching a target for frequency and intensity) that was found to be significantly slower in the PD group.

In another experiment (Jiang, O'Mara et al., 1999), aerodynamic measures of airflow, subglottal pressure, and laryngeal resistance were obtained for a group of persons with PD who had varying voice complaints ranging from none to severe. Measures were obtained in persons with PD and healthy controls for three intensity levels of soft, regular, and loud (75-dB, 80-dB, 85-dB SPL, respectively at 6 cm). One-third of the PD participants could not produce phonation at normal and loud intensities that were comfortable for the controls. The mean subglottal pressure of PD participants who could produce 3 levels of intensity was significantly higher than the mean subglottal pressure for controls. There were no significant differences between the two groups with respect to airflow. Because the airflow used by PD participants to produce a given intensity of phonation did not vary greatly from the flow employed by controls, the higher subglottal pressure implied a significantly greater laryngeal resistance during phonation in the patient group. The authors commented:

> Theoretically, increased resistance could be caused by phonation with a smaller glottal aperture or by greater resistance to deformation of the vocal folds with decreased pulsing of airflow. However, subglottal pressure can be increased to compensate with airflow and acoustic intensity levels measured in controls. That higher pressure, and presumably respiratory effort was used by subjects with Parkinson's disease to produce similar flow and intensity of phonation confirms the impressions of our subjects with Parkinson's disease who feel that they are working hard to

> produce intensity even though their voice is not as loud as they would like (p. 589).

The interactive coordination of the laryngeal musculature in the control of normal phonatory posture is not clearly delineated; however, studies have reported differences in muscle activation patterns for speech and non-speech tasks (Ludlow & Fujita, 1988; Ludlow & Lou, 1997; Ludlow et al., 1991). The intrinsic laryngeal muscles (e.g. those having attachments between the laryngeal cartilages) are (a) the posterior cricoarytenoid (PCA); (b) the lateral cricoarytenoid (LCA); (c) the thyroarytenoid muscles (TA), including the vocalis fibers and aryepiglottic fibers of the lateral TA; (d) and the interarytenoids, both transverse and oblique portions. All of these muscles are paired, with the exception of the transverse interarytenoid. These muscles presumably act in a coordinated manner to maintain a given vocal fold position. One other paired intrinsic muscle, the cricothyroid (CT), also affects the vocal folds. Contraction of the CT acts to lengthen the vocal folds and is associated with increasing pitch.

The fine motor control of these intrinsic muscles apparently breaks down in Parkinson's disease; however, reported findings are in disagreement regarding the precise mechanism of dysfunction. Some have argued that there is increased background activity with increased co-contraction of agonist and antagonist muscles indicative of rigidity of the laryngeal muscles (Ludlow et al., 1995; Hanson et al., 1984). However, Ramig (personal correspondence) and others (Jiang, O'Mara et al., 1999) have argued there is no convincing evidence for rigidity in the phonatory muscles. Recent evidence from two studies suggests

there is reduced myoelectric activity and overall weakness in these muscles (Baker et al., 1998; Luschei et al., 1999).

Baker et al. (1998) compared TA EMG amplitude in PD patients, age-matched healthy controls, and younger individuals under conditions of known vocal loudness. Absolute and relative (compared to maximum) EMG amplitudes of the TA muscle were compared during speech and nonspeech tasks. Reduced, rather than increased TA muscle amplitudes, and low, rather than excessive levels, of tonic background TA activity were observed for PD patients. Relative TA amplitudes were generally the highest for young individuals, lowest for older individuals, and intermediate for PD patients. The finding of intermediate, rather than low, relative TA amplitudes was related to the lower TA maximums observed for the PD individuals compared with the older or age-matched controls. The reduced muscle activation ranges caused by these low TA maximums suggested that PD patients used more of the TA operational range, but very likely did so with similar or lower levels of neural drive to laryngeal motorneuron pools when compared with the older individuals. These findings support the pathophysiologic models suggesting that hypokinesia (reduced movement) and bradykinesia (slowed movement) may affect laryngeal function. The low prephonatory TA amplitudes may affect the prephonatory posturing for initiation of phonation and may impede production of rapid offset-onset of phonation for glottal stop production.

Luschei and colleagues (Luschei et al., 1999) investigated discharge characteristics of TA and CT single motor units during phonation in young and older adults and in PD patients. The firing rate of TA motor units was decreased

in both older and PD men, and there was no significant difference in TA firing rates between older and PD males. This effect was not observed in females. The firing rate of CT motor units was similar for PD patients, younger participants, and older participants. Thus, there was differential involvement of laryngeal muscles for both older males and PD patients. The CT and TA muscles are innervated by different branches of the vagus nerve. It is interesting that the CT muscle is innervated by the superior laryngeal nerve, while the TA is innervated by the recurrent larygeal nerve.

In summary, the pathophysiology of laryngeal function in PD involves bowing of the vocal folds; however, bowing is not observed in all patients (Hanson et al., 1983; Smith et al., 1995). Reduced levels of TA muscle activity may contribute to the voice disorder associated with PD (Baker et al., 1998; Luschei et al., 1999). Persons with PD may use increased effort associated with increased respiratory drive and supraglottal constriction to compensate for incomplete glottal closure during phonation in order to maintain normal airflow values (Jiang, O'Mara et al., 1999). Such increased effort may underlie the self-perception of increased vocal effort in persons with PD.

Impact of Vocal Dysfunction on Quality of Life

Quality of life (QOL) refers to an individual's sense of well-being, purpose in life, and the ability to assume valuable roles (Koplas et al., 1999). Schrag, Ben Shlomo, Brown, Marsden, and Quinn (1998) investigated differences in QOL and psychosocial function between YOPD and OOPD individuals using a questionnaire developed specifically to assess PD issues with respect to QOL.

Self-report of QOL was significantly lower for the YOPD group in comparison with the OOPD group. YOPD persons reported lower marital satisfaction, poor stigma, greater depression, a higher loss of employment, and an increased disruption of family life.

Communicative dysfunction can negatively impact QOL (Antonius, Beukelman & Reid, 1996; Jacobson et al., 1997; Yorkston, Bombardier & Hammen, 1994). Brod, Mendelsohn, and Roberts (1998) reported that persons with PD listed impaired communication skills as a major factor of disability with PD, often leading to changes in career goals and early retirement. The impact of vocal dysfunction on quality of life in persons with PD is not well understood. However, it has been suggested that the progressive deterioration of speech and an inability to communicate effectively with family, friends, and employers has a negative impact on quality of life and self-esteem (Barbeau, Dushay, & Spiegel, 1965; Haberman, 1996; MacCarthy & Brown, 1989; Mutch, Strudwick, Roy, & Downie, 1986; Ramig et al., 1996; Yorkston, Bombardier, & Hammen, 1994). Current knowledge on this topic, based on published anecdotal descriptions, suggest a substantial impairment in communication-related quality of life in persons with PD (King, Ramig, Lemke, & Horii, 1994). Impaired communication skills are listed as a major factor of disability associated with PD, often leading to a change of occupation or early retirement (Nakano et al., 1973). While the early appearing symptom of hypophonia may not reduce intelligibility, it has a subtle negative effect on communication because it requires the "listener to work hard" (Ramig, 1992). Disease progression of PD is usually slow and life expectancy is

only slightly less than normal (Diamond & Markham, 1979). Therefore, the vocal symptoms may have an even greater impact on quality of life for a person who experiences early-onset of the disease.

Coates and Bakheit (1997) assessed the frequency and severity of verbal communication disability in persons with older-onset PD to establish if these deficits were useful predictors of disability. Using a 'yes' or 'no' response format, the researchers probed the older- onset PD participants' awareness of their speech difficulties and the negative effects on their lifestyle. Of the participants with a documented speech disruption (Yorkston & Beukelman, 1984), a large majority complained of day-to-day deficits in communication, their questionnaire did not probe specific psychosocial consequences of voice disorders such as emotional, functional, and physical restrictions placed on daily living (Jacobson et al., 1997). Consequently, this study may have underestimated the negative impact of vocal dysfunction on quality of life.

Effect of Aging on Laryngeal Function and Voice

As previously mentioned, the voice deficits associated with PD markedly mirror the characteristics of vocal aging, suggesting that our current knowledge base of laryngeal dysfunction in the PD population is confounded with aging effects (see p. 4 for previous review of aging effects on laryngeal function and voice). A person's overall physical condition has significant impact on vocal function (Ramig, 1983; Ramig & Ringel, 1983). Physiologic measures such as heart rate, blood pressure, percent of body fat, and forced vital capacity can be used to predict voice changes. Ramig (1983) and Ramig and Ringel (1983)

investigated the relationship between age-related changes in body physiology and acoustic parameters of voice in 48 men, ranging in age from 25 to 74 years of age. They observed that if the data were analyzed with respect to relationship between age and the various acoustic measures, then the sole age-related change in laryngeal functions was that it increased shimmer. However, when an estimate of physiological aging (e.g. physical condition) was correlated with the acoustic measures, there were significant differences in jitter, shimmer, and phonation range observed in sustained vowels produced by men in both good and poor physical condition. That is, younger men in poor physical condition evidenced increased perturbation values and a decreased phonation range. Therefore, individuals of the same chronological age but in different physical conditions have differing voice characteristics. Hence, aging alone does not account for laryngeal changes.

Videoendoscopic observations have provided evidence for reduced vocal fold closure with age (Honjo & Isshiki, 1980; Linville, 1992; Linville, Skarin, & Fornatto, 1989; Tanaka et al., 1994). Researchers have suggested that the aging individual uses some hyperfunctional compensation involving supraglottal constriction (Close & Woodson, 1989; Melcon, Hoit, & Hixon, 1989; Mueller et al., 1984; Ryan & Burke, 1974).

Tissue changes in the larynx involve changes in the biomechanical tissue of the epithelium and lamina propria (i.e. the 4 layers of tissue that cover the vocalis muscle). While the tissue changes that occur in the vocal folds have been well documented (Gracco & Kahane, 1989; Kahane, 1987; Mueller et al., 1984) a

detailed description is beyond the scope of this review. In short, the main process of tissue aging involves a change in the collagen, elastin and proteins that comprise the extracellular matrix of the epithelium and lamina propria. These changes cause increased stiffness, decreased elasticity and decreased thickness of the lamina propria. Collectively, these changes contribute to the increasing short-term perturbations of F_o as people age and could account for the hoarseness/ roughness perceptually heard in the elderly (Gracco & Kahane, 1989).

Changes in the cricoarytenoid joint have been well documented (Kahane, 1988) and described as "roughened surface areas, reduced working surfaces caused by ossification, thickening, or raised rims of the articular facets..." (Kahane, 1987, p. 54). The functional significance of these changes involves a reduction or restriction of the arytenoid cartilages that interferes with the extent and completeness of vocal fold approximation. This, in turn, could contribute to the incomplete glottal closure with increased breathiness secondary to air leakage.

Changes in muscular morphology and physiology with aging probably occur in the larynx. In other skeletal muscle, the process of muscular atrophy and decreased neural control begins at age 25 years in humans and progresses rapidly with increasing age (Lexell, 1995). There is certainly the potential for these changes to also occur within laryngeal muscle. Age-related atrophy has been described for all laryngeal muscles innervated by the recurrent laryngeal nerve (Bach, Lederer, & Dinolt, 1941; Benjamin, 1981; Malmgren, Fisher, Bookman, & Uno, 1999; Malmgren & Ringwood, 1988; Segre, 1971). However, minimal data are available on peripheral neural pathologies that affect control of laryngeal

muscles (Shindo & Hanson, 1990). Studies of laryngeal EMG observations have reported reduced amplitude and decreased firing rates for the TA muscle in older compared with younger individuals (Baker et al., 1998; Luschei et al., 1999). Furthermore, Luschei et al. (1999) reported that aging does not affect all muscles or muscle fiber types equally. They observed greater reductions and variability in firing rates associated with the thyroarytenoid muscle (TA), a muscle used to adduct the vocal folds and influence medial compression (Ludlow et al., 1991) in comparison with the cricothyroid, a muscle used to increase Fo. Furthermore, there appear to be gender-related differences in the TA muscle. Luschei et al. (1999) reported that the firing rate of TA motor units was decreased in older men but not in older women. Relevant to the current question, there was no difference observed between the firing rate of TA motor units in healthy older men and age-matched PD participants.

It has been suggested that muscle fiber atrophy, disturbed neural drive, and cartilaginous changes reduce the completeness of glottal closure and this inadequate closure underlies the soft, breathy voice of the aged speaker (Linville et al., 1989). Also, certain aged individuals have a tremulous voice quality or experience phonatory breaks during sustained phonation. These characteristics have been associated with age-related changes in neural laryngeal control (Ramig & Ringel, 1983; Ramig & Shipp, 1987). Furthermore, the slower rate and poor devoicing associated with vocal fold diadochokinesis task performance in aged individuals (Ptacek et al., 1966) may be attributable to loss of muscle fibers in the TA and decreased firing rate (Ludlow & Lou, 1997; Luschei et al., 1999).

In summary, vocal aging characteristics parallel the hypophonia associated with PD. Studies of vocal dysfunction associated with PD have included only aged participants or they have grouped data for both the aged and young-onset individuals. Consequently, it is uncertain if the hypophonia observed in PD is attributable solely to aging effects, the disease process alone, or an interaction between aging and disease. Furthermore, given the relationship between physical condition and voice characteristics, it is likely that persons with YOPD, in poor physical condition, might evidence vocal dysfunction.

Voice and Speech Treatment Approaches

Various approaches have been used to treat voice and speech deficits in persons with PD. Treatment methods have included pharmacological, surgical, and behavioral (e.g., speech therapy). There are discrepancies in the literature regarding the efficacy of speech treatment in persons with PD. Schulz and Grant (2000) noted that such differences in treatment response may be attributable to factors such as dysarthria severity, task used to assess treatment, co-occurring conditions (including aging), and/or the specific neurological substrate involved in the disease process.

Pharmacological Methods.

Categories of drugs are available to treat PD. The preferred treatment approach utilizes the least amount of medication to control symptoms while allowing for acceptable levels of functioning. The most effective pharmacological treatment involves dopamine replacement (Lieberman, 2003; Waters, 1999; Weiner, Schulman, & Lang, 2001). First introduced in 1968, Levodopa (L-dopa),

acts to replenish dopamine levels in the brain. Combining L-dopa with carbidopa produces Sinemet, which is the principle medication for treating PD. The addition of carbidopa prevents nausea and vomiting (Weiner et al., 2001). Symptom improvement usually follows within thirty minutes after administration of L-dopa, particularly for reduction in bradykinesia, ridigity, and tremor. These benefits, however, are often accompanied by early side effects of nausea and hypotension. L-dopa is converted into dopamine by nigral neurons, replacing the diminished supply of the neurotransmitter. However, treatment over time and the extended use of L-dopa drugs can cause on-off fluctuations and dyskinesia (Marsden, 1994). The *on-off effect* refers to the drug-dose related fluctuations. Instead of experiencing sustained relief from tremor, ridigity, and bradykinesia, patients notice a marked drop in mobility and/or increase in tremor as one dose of medication "wears off" and before the next dose "kicks in" (Weiner et al., 2001). A second, common side effect of L-dopa is the appearance of various involuntary movements, termed *dyskinesia* (Duvoisin & Sage, 1996). These include twitches, jerks, nods, gestures, twisting or writhing movements, or simple restlessness. The most common dyskinetic movements (e.g., twisting or writhing movements) appear at higher doses of L-dopa and usually occur only for a brief period when the brain levels of dopamine are highest, 1 to 2 hours after the last daily dose. Some persons, particularly YOPD patients, are more susceptible to the development of dyskinesias (Arevalo et al., 1997). Unfortunately, attempts to control the on-effect by increasing the dosage of L-dopa produce severe dyskinesia. In addition to the on-off effect and dyskinesia, L-dopa can cause

confusion, dementia, hallucinations, and delusions (Marsden, 1994). Most importantly, PD patients build up sensitivity to L-dopa after 5-10 years of use, which makes the course of treatment gradually ineffective (Duvoisin & Sage, 1996; Lieberman & Williams, 1993). Recently, Catechol-O-Methyltransferase (COMT) inhibitors, have been used in combination with L-dopa (Marjama-Lyons & Koller, 2001). COMT inhibitors (i.e., entacapone [Comtan] and tolcapone [Tasmar]) inhibit levodopa metabolism and thus increase the bioavailability. With the increased bioavailability, higher concentrations of levodopa can cross the blood brain barrier, thus providing effective treatment of the end-of-dose wearing off effects (Poewe, 2004)

Studies were completed in the 1970's comparing symptoms during placebo and L-dopa treatment (Nakano, Zubick, & Tyler, 1973; Rigrodsky & Morrison, 1970). These researchers noted a general trend in improvement of speech production but more dramatic results in limb symptom improvement. This trend was observed subjectively during spontaneous speech as well as in oral reading through the evaluation of overall speech adequacy, clarity of speech, intelligibility, and temporal aspects of speech (i.e. rate, rhythm, and pauses). As summarized by Schulz and Grant (2000), current investigators continue to report the same trend. Pharmacological treatment methods work effectively to reduce limb symptoms, but these methods have small impact on voice or speech deficits. Schulz and Grant, however, suggested that speech therapy was maximized in persons who were optimally medicated. Investigations of voice deficits associated with PD have used patients who are receiving L-dopa treatment. Therefore, the results may

be confounded with the medication side effects and may not present an accurate profile of vocal symptoms associated with PD.

Based on diminishing effectiveness of dopamine replacement therapy during the course of disease progression, the initiation of levodopa drugs is commonly delayed until the middle to later stages of the disease. (Weiner et al., 2001). Therefore in early stages of PD, if Activities of Daily Living (ADLs) are not significantly limited, then either no medical treatment or pharmacological treatment with selegiline are recommended. Selegiline is a selective monoamine oxidase B (MAO-B) inhibitor that has been demonstrated to have a neuroprotective effect in slowing down the disease progression (Ahlskog, 2003). Shea, Drummond, Metzer, and Krueger (1993) investigated the effect of selegiline on speech performance in 10 persons with PD. Significant improvement was reported for only 8 of the 40 measures relating to the speech processes of respiration, phonation, resonation, articulation, and prosody. Those measures showing improvement were related to articulation and respiration. Phonatory measures did not demonstrate a significant change associated with selegiline treatment. Consequently, like L-Dopa, these medications are also effective in improving limb symptoms, but have little effect in improving speech intelligibility.

Dopamine agonist drugs are generally initiated once the disease begins to significantly impact on ADLs (Marjama-Lyons & Koller, 2001). This category of drugs include bromocriptine (Parlodel), pergolide (Permax), pramipexole (Mirapex), and most recently ropinirole (Requip). Similar to selegiline, dopamine agonist medications have neuroprotective effects in the laboratory, in vitro and in

vivo (Ahlskog, 2003). In addition, these medications delay the onset of involuntary movements (dyskinesia) commonly observed after 3-5 years of treatment with levodopa. Reportedly, dopamine agonists improve motor symptoms; however, their effects on speech have not been reported (Schulz & Grant, 2000).

Anticholinergics, used in the early stages of PD, include trihexyphenidly (Artane) and benztropine (Cogentin). These drugs block acetylcholine, thereby restoring the balance in the basal ganglia between dopamine and acetylcholine. Side effects include impaired thinking, concentration, and memory, particularly in older PD individuals (Waters, 1999).

In summary, pharmacological methods of treatment in isolation do not appear to significantly improve voice and speech function in individuals with PD. However, Schulz and Grant (2000), in their review of the efficacy literature, suggested that "…speech therapy (when persons with PD are optimally medicated) has proven to be the most efficacious therapeutic method for improving voice and speech function" (p. 61).

Surgical Methods.

Surgical intervention has become an acceptable alternative for individuals with PD once dopamine replacement is ineffective (Weiner, Shulman & Wang, 2001). Surgical treatment for PD may include ablative surgery (thalamotomy and pallidotomy), transplantation, and deep brain stimulation (DBS). Phonosurgery, involving injection of collagen into the true vocal fold, also has recently been used to improve glottal incompetency secondary to vocal fold bowing.

During ablative surgery for PD, a lesion is created in the thalamus, globus pallidus, or subthalamus. Currently, DBS has replaced ablative surgery. Researchers who investigated symptoms postoperatively after thalamotomy or pallidotomy procedures reported that although most patients were observed to have significant reduction of limb symptoms, there was no improvement or a worsening of speech symptoms (Almgren, Andersson, & Kullberg, 1972; Countryman & Ramig, 1993; Favre, Burchiel, Taha, & Hammerstad, 2000; Ghika et al., 1999; Hugdahl, Wester, & Asbjornsen, 1990; Iacono, Lonser, & Kuniyoshi, 1995; Iacono, Lonser, & Yamada, 1994; Kondziolka et al., 1999; Schrag et al., 1999; Schulz, 2000; Schulz, Peterson, Sapienza, Greer, & Friedman, 1999; Schuurman, de Bie, Speelman, & Bosch, 1997; Scott et al., 1998). In fact, unilateral operations on the thalamus in the individual's dominant hemisphere were more likely to produce new speech disturbances, such as monotonous voice, slow speech (Jenkins, 1968) and decreased vocal loudness and articulation difficulties (Allan, Turner, & Dadea-Ciria, 1966). Data regarding voice and speech function have demonstrated varied results following pallidotomy. Voice and speech function was reportedly improved in some studies (Laitinen, Bergenheim, & Hariz, 1992; Sutton, Couldwell, & Lew, 1995). Other investigators have reported the emergence of speech and/or voice symptoms following pallidotomy, including transient facial paresis (Lozano et al., 1995) and reduced labial force (Barlow, Iacono, Paseman, Biswas, & D'Antonio, 1998). However, Schulz and colleagues (Schulz et al., 2000) presented data that measured changes in vocal intensity following pallidotomy in a large sample of speakers with PD who

exhibited a range of hypokinetic dysarthria. They reported that mildly dysarthric patients had significantly greater relative increases in vocal intensity following pallidotomy than either moderately or severely dysarthric patients. Moderately or severely dysarthric patients exhibited reduced vocal intensity following pallidotomy.

Perhaps the most salient finding of the effects of pallidotomy is the case-to-case variability noted. Such variability demonstrates the outcome of the segregated nature of neuronal circuits in the basal ganglia loops, with independent circuits subserving specific anatomical structures such as the orofacial versus limb structures. Small inconsistencies in the site and volume of the lesion could variably affect the functioning of the limb and speech muscles, consistent with the case-to-case variability noted (Murdoch, 2001).

The transplantation procedure involves replacing cells (from the basal ganglia of fetal pigs) in the striatum, which is thought to stimulate dopamine production (Solomon, McKee et al., 2000). This experimental procedure is only performed at a limited number of centers. Baker and colleagues (Baker, Ramig, Johnson, & Freed, 1997) reported findings from acoustic, electroglottography, and perceptual measures that were analyzed pre- and post-surgery in five patients with PD, ranging in age from 47—67 years of age. Findings suggested that transplantation surgery did not systematically influence voice and speech production, whereas it did improve overall limb motor performance.

Deep brain stimulation (DBS) refers to the electrical stimulation of the thalamus, the subthalamic nucleus or the globus pallidus for alleviation of

parkinsonian symptoms. Electrodes are surgically implanted and the amount of electrical stimulation can be controlled through an external device worn by the patient. DBS is performed instead of pallidotomy because it offers less risk of permanent neurologic damage, and it is reversible. Solomon and colleagues (Solomon, McKee et al., 2000) examined the efficacy of DBS in three men with severe PD. Two patients had bilateral pallidal stimulation; one had unilateral stimulation contralateral to a prior pallidotomy. Measures included overall motor function, auditory perceptual speech characteristics, intelligibility, interpause speech rate, and aerodynamic measures. Researchers noted improved overall motor function (better mobility, reduced tremor and dyskinesia) in all three patients. Responses for speech varied widely. One patient developed marked hypophonia postoperatively that was not improved via deactivation of the bilateral stimulators.

Phonosurgical techniques have recently been used to target the glottal incompetence that often underlines the voice complaints of patients with PD (Berke, Gerratt, Kreiman, & Jackson, 1999). The procedure involves the percutaneous injection of bovine collagen near the vocal ligament. After injection, collagen may exist in the larynx for more than one year. Berke et al. (1999) reported favorable results from a telephone survey of 35 patients with PD who underwent collagen augmentation of the vocal folds for hypophonia associated with PD. Patient satisfaction with the procedure was reported by 75% of the respondents. However, data reporting objective outcome measures and more long-term follow-up are needed to establish the efficacy of this technique.

In summary, with the exception of percutaneous injection of bovine collagen in the vocal fold, surgical treatments do not appear to be effective in reducing voice and speech symptoms with the exception of those who present with mild symptoms. In fact, some surgical procedures may trigger the appearance of new speech disturbances such as monotonous voice, slow speech (Jenkins, 1968) and decreased vocal loudness as well as articulation difficulties (Allan, Turner, & Dadea-Ciria, 1966).

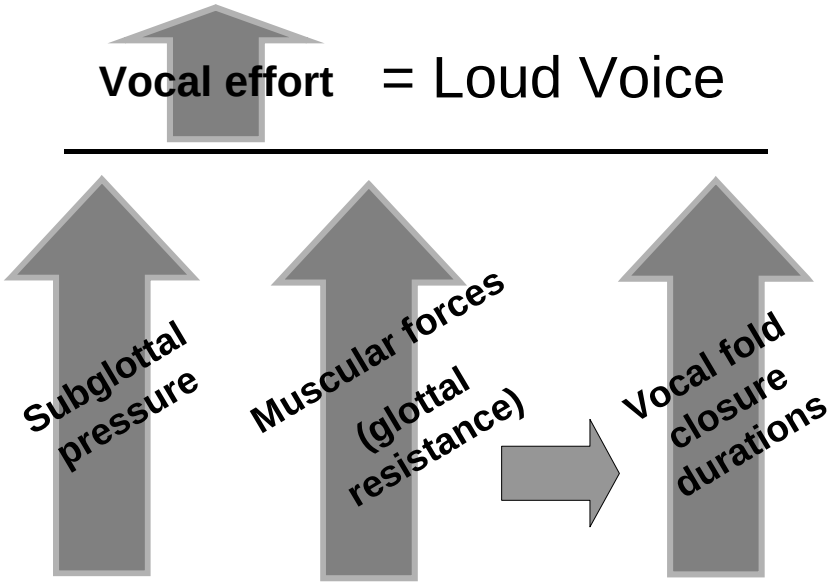
Speech Therapy.

Prior to the 1990's, opinions of the efficacy of speech therapy for PD were negative. The basis for this skepticism included the following: (a) The progressive nature of the disease and the age of many patients (Sarno, 1968); (b) the emphasis on traditional articulatory drill work (Adams, 1997); and (c) the lack of intensive treatment (Ramig, 1995a, 1995b; Ramig et al., 1996; Ramig & Dromey, 1996; Robertson & Thomson, 1984). Fox et al., (2002) eloquently summarized this issue by stating, "Individuals with [PD] have been particularly resistant to speech treatment, with the conventional wisdom being summarized by the statement that changes observed in the treatment room disappear on the way to the parking lot" (p. 111).

Over twenty years ago, Ramig and colleagues shifted the focus of treatment in PD from an articulatory to a phonatory focus. Treatment procedures targeting an increase in vocal loudness evolved into the Lee Silverman Voice Treatment or LSVT™ , a treatment approach developed by Ramig and her colleagues in an effort to improve the previously unsuccessful approaches that

focused on improving articulatory mobility (Countryman et al., 1997; Countryman et al., 1994; Countryman & Ramig, 1993; Dromey et al., 1995; King et al., 1994; Ramig et al., 2002; Ramig, 1997; Ramig et al., 1994; Ramig et al., 1996; L. O. Ramig et al., 1995; Ramig & Dromey, 1996; Ramig & Verdolini, 1998; Smith et al., 1995). The LSVT™ therapy program emphasizes high effort loud phonation (80 – 90 dB SPL at 30 cm) and loud speech (i.e. 75 – 80 dB SPL at 30 cm) to improve respiratory, laryngeal, and articulatory functions during speech. The primary focus stresses shifting the patient from a quiet mode to a normal mode by increasing vocal effort. As illustrated in Figure 3, increased vocal effort facilitates increased subglottal pressure and increased muscular forces (glottal resistance) (Isshiki, 1964). These adjustments result in increased vocal fold closure duration and increased speech intelligibility (Dromey & Ramig, 1998; Dromey et al., 1995; Isshiki, 1964; Pickett, 1956; Rostolland, 1985; Schulman, 1989; Schulz, 2000).

Figure 3. Laryngeal changes associated with increased vocal effort and loud voice, modeled after Isshiki, 1964.



In their efficacy research, Ramig and colleagues reported significant increases in maximum duration for sustained vowel phonation, maximum fundamental frequency, habitual fundamental frequency, and fundamental frequency variability in speech in patients treated with the LSVT™ program (Countryman & Ramig 1993; Countryman, Ramig, & Pawlas, 1994; Dromey, Ramig, & Johnson, 1995; Ramig, 1995; Ramig & Dromey, 1996; Ramig, Bonitati, Lemke, & Horii, 1994; Ramig, Countryman, Obrien, Hoehn, & Thompson, 1996). Perceptual clinician's ratings have corroborated positive post-treatment effects noting increases in vocal loudness, improvements in speech intelligibility, functional communication, and improved voice quality ratings in hoarseness and roughness (Baumgartner, Shapir, & Ramig, 2001; Ramig, 1992). Physiologic evidence suggested that individuals with PD treated with LSVT™ improved vocal

fold adduction and vibratory motions (Smith et al., 1995). Improvement in swallowing has also been reported following LSVT™ (El-Sharkawi et al., 2002). Long-terms effects, for up to two years post-treatment, have been documented (Ramig et al., 1996). Furthermore, research efficacy studies have provided strong evidence that the LSVT™ outcome is treatment-specific, and the observed improvement in persons treated with LSVT™ is not secondary to placebo and Hawthorne effects or improvement associated with repeated testing (e.g., familiarity with test material, test procedures, test practice) (Ramig et al., 2002).

One of the underlying principles of this therapy program is its intensive and daily schedule (e.g., 4 sessions weekly for 4 weeks). Ramig and colleagues demonstrated that favorable treatment outcomes are not likely if the sixteen treatment sessions are delivered twice weekly for 8 weeks (Ramig et al., 1995). However it is important to note the post-treatment outcome data for LSVT™ is obtained in a sound treated laboratory setting, and the patients are externally cued for task performance. Generalization of treatment outside of the laboratory and clinical settings consists of only anecdotal remarks. Ramig and colleagues have noted that patients' family members have reported less carry-over of the post-LSVT™ stronger voice outside of clinical setting. Also, the degree of carry-over varies across patients.

More recently, Liotti and colleagues provided preliminary evidence of neural correlates of improvements in hypophonia in five individuals with mild PD following LSVT™, using PET scans (Liotti et al., 2003). Prior to treatment, in comparison to matched healthy controls, PD patients had strong speech-related

activations on PET scans (e.g., regional cerebral blood flow, rCBF) in the motor and premotor cortex (M1-mouth, supplementary motor cortex), and inferior lateral premotor cortex. These areas were overall significantly reduced post- LSVT™. Also, post-LSVT™, the PD patients evidenced a shift to greater activation in the basal ganglia and anterior insula region. These observations suggested a change from an abnormally effortful volitional control (cortex) compensating for disordered voice and speech, to a more effortless and automatic implementation of speech motor actions (basal ganglia). The authors suggested the data provided preliminary evidence to link LSVT™ effects to specific neural correlates during speech-motor tasks. However, because of the small sample size, these results must be regarded as only prelimary.

Recently, de Swaart, Willemse, Maassen, and Horstink (2003) suggested that the LVST ™ treatment might have adverse effects because it raises vocal pitch and laryngeal muscle tension. That is, a greater expiratory effort associated with the LSVT™ "think loud" increases laryngeal muscle tone and vocal pitch, and can result in a high-pitched, strained, pressed or screaming sound. De Swaart et al. developed the Pitch Limiting Voice Treatment (PLVT) of "speak loud and low," and proposed that the therapy, comparable with LSVT™, prevents an increase of vocal pitch that results in increased laryngeal muscle tone and laryngeal resistance. However, there are some limitations of the de Swaart et al. study as noted by Ramig (personal correspondence, April 2003).

In summary, long-term treatment outcomes associated with LSVT™ are documented; however, the effective change or generalization of the treatment

effect outside of the clinical setting is less than optimal (Adams, 1997; Cariski & Rosenbek, 1999). Although efficacy data comparing pre- to post-treatment changes in vocal intensity revealed a significant increase for sustained phonation (14 dB) and reading (12 dB), changes in conversational speech (although statistically significant) were substantially less (5 dB) (Ramig et al., 1995).

The LSVT™ efficacy studies have either focused on the late-onset group or have collapsed the young- and older-onset groups for analyses. Ramig (personal communication, Oct. 21, 1998) has commented that young-onset patients seem to have a higher level of improvement, and they often regain normal voice characteristics following completion of the LSVT™ program. However, because the LSVT™ Program is intensive (i.e., four 60-minute treatment sessions per week over a 4 week period) and expensive, young-onset patients, who are usually engaged in full time employment, might be reluctant to pursue a time-intensive treatment program that requires daily treatments. Furthermore, it is not certain whether young-onset patients would require such intensive treatment if their vocal symptoms are not confounded by aging effects.

Biofeedback and Prosthetic Devices.

A second, less frequently used behavioral approach for treating voice and speech symptoms in individuals with PD has incorporated the use of biofeedback and prosthetic devices also aimed at increasing vocal intensity (Adams & Lang, 1992; Cariski & Rosenbek, 1999; Greene & Watson, 1968; Greene, Watson, Gay, & Townsend, 1972; Hanson & Metter, 1983; Rubow & Swift, 1985; Yorkston et al., 1988; Zicker, Tompkins, Rubow, & Abbs, 1980). Each of these devices either

compensates for decreased vocal intensity (e.g. voice amplification) or provides a mechanism that elicits increased vocal intensity using some type of external cueing.

Speech masking techniques have been used to investigate volume regulation in PD (Ho, Bradshaw et al., 1999) and to explore the therapeutic effects for increasing speech intelligibility (Adams & Lang, 1992). Presenting healthy speakers with a masking noise is known to immediately produce a consistent increase in loudness during speech, referred to as the *Lombard effect* (Lane & Tranel, 1971; Letowski, Frank, & Caravella, 1993; Summers, Johnson, Pisoni, & Bernacki, 1988). Summers et al. (1988) reported that healthy speakers modified both the prosodic and segmental acoustic-phonetic properties when talking in noise presented at 3 levels (80, 90, or 100 dB SPL). For example, in addition to increasing speech intensity by 5 to 6 dB, speakers evidenced a 28% to 46% decrease in spectral tilt, a 3 to 19 Hz increase in F_o, and a 14% to 18% increase in segment duration. Increased intelligibility was found while speaking in noise compared with quiet. Summers et al. (1988) noted that speakers automatically adjusted acoustic features to maintain intelligibility under conditions of noise without instruction. Some of the acoustic changes observed are similar to those observed in studies that ask healthy speakers to speak more clearly (Gordon-Salant, 1986; Picheny, Durlach, & Braida, 1985, 1986, 1989) or to emphasize or stress an utterance (Cooper, Eady, & Mueller, 1985). However, Letowski et al., (1993) reported that the spectra of Lombard speech contained more high-frequency (above 630 Hz) energy than normal or *raised* speech produced in quiet.

Therefore, Lombard speech is acoustically not identical to loud speech produced in a quiet environment.

Boone (personal correspondence, April 1999) reported "better" phonation in voice-disordered clients under speech-range masking conditions. Influenced by the Lombard effect, the clients demonstrated a voice with less perturbation, increased intensity, and more efficient aerodynamics (i.e., less air flow, greater subglottal pressure). Adams and Lang (1992) reported marked, immediate increase in voice intensity while under the influence of 90 dB SPL masking white noise in 10 patients with Parkinson's disease (one with YOPD). Five speakers increased vocal intensity by 6 or 7 dB, 3 speakers increased by 4 dB, and 2 speakers evidenced a 2-3 dB increase. Speech intelligibility increased in five patients, including the YOPD patient, in the masking noise condition. It is not clear whether these five patients were those who increased their vocal intensity by 6 or 7 dB. Adams and Lang noted that the failure to observe a consistent increase in all patients' intelligibility scores under masking noise influence was an unexpected finding. Their patients spanned a large age range (48 - 80 years) and disease/speech severity range (mild-severe). Therefore, such variable treatment results may be expected. In addition, Adams and Lang did not control for hearing loss, which may affect responsiveness to masking noise. They recommended that further study was needed using different measures of intelligibility, to determine if the Lombard effect could be exploited therapeutically to provide long-term benefits to PD patients who were experiencing low speech intensity.

Ho and colleagues (Ho, Bradshaw, Iansek, & Bradshaw, 1999) used masking procedures to investigate volume regulation in persons with PD. The participants included 12 elderly PD participants and 12 healthy age- and sex-matched controls. The participants were screened for hearing acuity, but only those requiring the use of hearing aids were excluded. With respect to volume regulation, participants with PD showed lower speech volumes than healthy controls for each of the reading and conversation conditions. Healthy controls increased volume incrementally as masking level increased, but no greater than 2 dB for the loudest masking condition (e.g., 30 dB HL). The persons with PD demonstrated an *abnormal pattern* of speech volume modulation as they failed to increase volume in response to masking noise. This study used low speech masking levels and failed to provide compelling evidence to support the hypothesis that individuals with PD do not demonstrate the Lombard effect similar to that observed in unimpaired individuals. The maximum increase of 2 dB in vocal intensity observed in the healthy controls is considerably less than the approximately 5 dB increase associated with the Lombard effect reported in other studies that presented the masking level at 90 dB SPL (Lane & Tranel, 1971; Letowski et al., 1993; Summers, Johnson, Pisoni, & Bernacki,1988). Finally, participants with mild and moderate hearing losses were included in the Ho, Bradshaw et al. (1999) study; therefore, hearing loss effects may have confounded this study.

In a subsequent experiment (Ho, Bradshaw et al., 1999) participants were given a “reverse Lombard” condition. Participants’ speech was fed back to them

via headphones at various levels of loudness. Healthy controls automatically decreased volume when presented with auditory feedback that amplified their speech, whereas the persons with PD showed minimal decreases in speech volume. These results provide further evidence to support the notion that self-monitoring of auditory feedback may be disturbed in persons with PD and supports the abnormal sensory gating hypothesis.

Observations regarding the improvement of speech with use of an intensity biofeedback device have been reported in two studies (Rubow & Swift, 1985; Zicker, Tompkins, Rubow, & Abbs, 1980). For each study a microcomputer-based wearable biofeedback device was designed to provide the person with PD with information about their vocal intensity level. Zicker et al. (1980) monitored vocal intensity in a female patient with PD during a reading task. The patient wore the biofeedback device with an alarm set at a higher threshold than the patient's habitually low loudness level. During the five minutes sampled, the alarm generated five tones and the patient raised her volume each time. However, she quickly dropped down to her normal soft voice after each tone generation. Rubow and Swift (1985) monitored vocal intensity using a similar device both in 18 clinical sessions of biofeedback and in 27 sessions of the patient receiving vocal intensity feedback outside the clinical setting. Acoustic and perceptual analyses were performed pre- and post-treatment (the patient was not wearing the device), and the results indicated improvement for 8 out of 12 perceptual dimensions for spontaneous speech and for 9 out of 12 dimensions for oral reading. The authors reported that the patient transferred the increased intensity to outside of the clinic

while receiving feedback from the device and reported improvement in quality of life. The results, however, were quite variable. They attributed the variable effects to the Parkinson L-dopa medication cycle. Importantly, the patient was of older-onset (i.e., age 67 years) with mild-moderate PD of 13 years duration. Nonetheless, these findings provide strong evidence that supports the use of external cueing to facilitate generalization outside the clinical setting.

Investigators have used voice amplification devices that increase the speech signal's intensity (Greene & Watson, 1968) or increase the signal's intensity and clarify disordered speech even in the presence of ambient noise (Cariski & Rosenbek, 1999). These studies were concerned with the effectiveness of amplification in improving speech intelligibility. In an anecdotal report, Greene and Watson summarized their experience with 20 patients with PD. They noted that a benefit was gained from speech amplification only for patients who demonstrated decreased vocal loudness without slurred articulation. No benefit was observed for those who evidenced both decreased vocal loudness and slurred articulation. Cariski and Rosenbek (1999) described changes in sentence intelligibility for two PD patients with severe functional speech impairment using a device known as the Speech Enhancer™ (SE). The SE is an assistive speech system device that was developed to amplify and clarify disordered speech even in the presence of ambient noise. Unlike simple amplification devices, the SE compares the incoming speech signal to a normalized model of speech and alters the signal in real-time to a best-fit approximation of the model (Speech Enhancer™, Electronic Speech Enhancement, Inc., St. Louis, MO). One of the

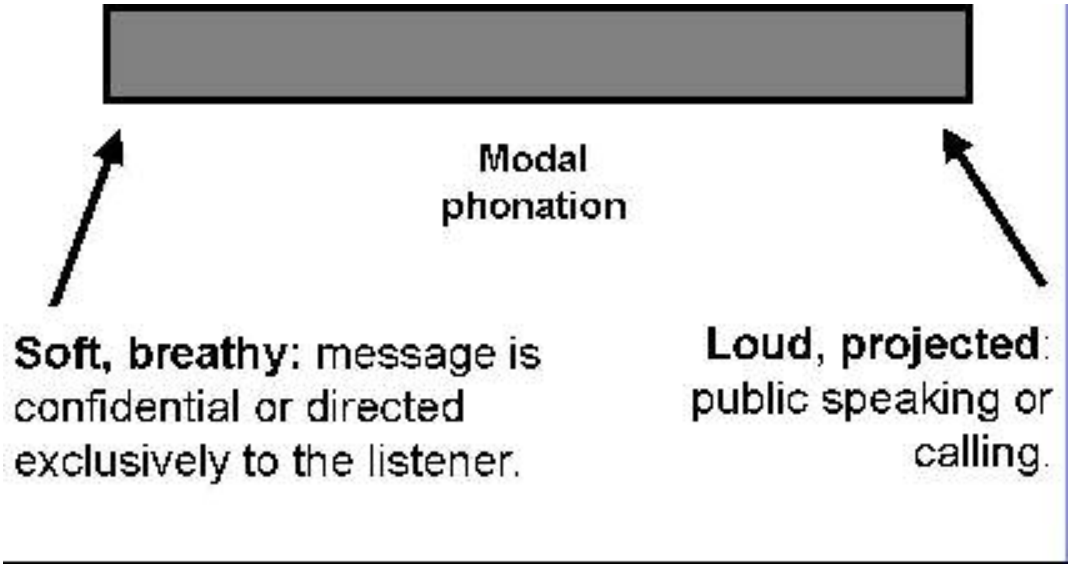
authors noted that subjectively the SE effectively improved sentence intelligibility in both patients. An interesting finding reported by the authors was that the greatest improvement in intelligibility (i.e., 67%) was observed for the condition that combined amplification with verbal cueing (i.e., “Get to a bigger size”). These results support the benefits of using SE in persons with severe phonatory and articulatory deficits as well as the effectiveness of verbal cueing.

In summary, clinicians and clinical researchers recognize that some persons with PD improve speech intelligibility using either behavioral speech changes or therapeutic devices that facilitate increased vocal effort. However, some patients fail to improve despite various treatments. Generalization of skills to other communication partners and environments may also be affected (Adams, 1997). The cause of therapy failure, poor generalization, and limited carry-over remains uncertain. Cognitive deficits may reduce the ability to monitor several sensorimotor parameters at once (Artieda et al., 1992; Brown & Marsden, 1991; Dagenais et al., 1999; Flowers, 1978; Forrest et al., 1989; Freeman, Cody, & Shady, 1993; Georgiou et al., 1993; McNamara et al., 1992; Pastor et al., 1992), or abnormal sensory gating may reduce the ability to monitor vocal effort (Fox & Ramig, 1997; Gandevia, 1982, 1992; Gandevia & Rothwell, 1987; Ho, Bradshaw et al., 1999; Ho, Iansek et al., 1999; McCloskey, 1981; Schneider, 1984; Schneider et al., 1986). Sensory and cognitive influences are implicated as is disease severity, and patient/family motivation (e.g., wants, needs, and values). Clinical failures, regarding generalization, as well as the need for more efficacious and cost-

effective treatments serve as an impetus to motivate continued efforts to search for additional treatment methods.

The literature suggests that additional studies are needed to develop therapeutic approaches that use biofeedback procedures, in particular with the use of assistive or prosthetic devices that target increased vocal effort. It can be hypothesized that PD speakers use a habitual laryngeal postural setting involving loose vocal fold adduction, resulting in an aspiration breathy voice. As illustrated in Figure 4, normal speaking contexts can range from a soft, aspirant or confidential voice to a loud projected voice (Colton & Casper, 1996). The middle point (modal phonation) represents the neutral setting where vocal efficiency and power are optimal.

Figure 4. Range of normal phonatory settings.



Under the influence of the Lombard effect, induced by a prosthetic device such as a speech masker, speakers increase their vocal effort. If a speaker's habitual setting is a full, clear voice, then use of a speech masker may result in a

pressed, harsh voice mode. However, if a speaker habitually uses an aspirant or breathy voice, then the influence of masking may result in better glottal closure resulting in a full, clear voice. Ramig has used a similar argument to explain the beneficial effects of LSVT™ (Ramig, Pawlas, & Countryman, 1995; Ramig et al., 1996).

Theoretical Implications: Direction for Current Research

There is documented evidence that vocal function is affected in the early stages of PD. However, nearly all research concerning the oromotor or laryngeal sequelae of PD has employed elderly persons or a patient group covering a wide age range. Clearly, age-related co-morbidities and the person's physical condition affect the results of studies that include elderly participants (Ramig et al., 1995). It would be valuable to study a YOPD group, unaffected by age-related changes, to obtain an uncontaminated view of parkinsonian vocal and speech deficits and to further elucidate the functions of the basal ganglia in voice production. The manner in which YOPD affects speech and voice has not been described; therefore, the purpose of this research was to delineate the multidimensional changes in voice characteristics of YOPD participants, who represent a form of PD uncontaminated by aging effects. This knowledge could facilitate the development of more effective voice interventions for this population.

In the preceding literature review, it was established that a common complaint of PD speakers and their family members is a soft voice. One purpose of the current study was to explore the hypothesis that the multidimensional changes of breathy voice quality and hypophonia reported in PD speakers suggest

a change in the speaker's voice mode or habitual *setting* of the laryngeal posture. There is compelling evidence suggesting that deficits in auditory monitoring or scaling of effort may disrupt the internal regulation of vocal loudness in persons with PD (Ho, Bradshaw et al., 1999; Ho, Iansek et al., 1999; Liotti et al., 1999; Liotti et al., 2000). That is, because of a deficit in internal speech volume regulation, people with PD perceive their normal conversational voice as loud. Furthermore, evidence suggests that persons with PD use greater respiratory drive and supraglottal construction to compensate for glottal insufficiency. This results in a greater effort than that used by healthy speakers (Baker et al., 1998). Therefore, persons with PD may adjust their habitual setting or mode to one similar to that used by healthy speakers when they are speaking in a quiet or confidential voice.

Recent treatments for PD-related voice disorders have focused on altering voice mode by increasing loudness or physical effort when talking (Ramig, 1995a, 1995b; Ramig, 1997, 1999; Ramig et al., 1994; Ramig et al., 1996; L. O. Ramig et al., 1995; Ramig & Dromey, 1996; Ramig & Verdolini, 1998). In LSVT™, patients are cued to "think loud." As reviewed earlier, this external cueing plays an important role in successful performance of tasks, such as speech volume regulation (Brown & Marsden, 1988; Fimm, Barti, Zimmerman, & Wallesch, 1994; Ho, Bradshaw et al., 1999; Nakatani & Dukes, 1977; Oliveira, Gurd, Nixon, Marshall, & Passingham, 1997). However, external cueing like "think loud" may impose an attentional problem and may explain why persons with PD who are treated with the LSVT™ have difficulty generalizing outside the clinical setting.

Importantly, speech masking serves as an external cueing technique for increased vocal effort in PD, and perhaps imposes less cognitive constraints.

The efficacy of using masking noise to increase vocal loudness is uncertain because prior studies have been confounded by effects of aging and hearing loss (Adams & Lang, 1992; Ho, Bradshaw et al., 1999). Therefore, studies have yet to determine the effectiveness of speech masking in persons with PD.

The current study investigated the effectiveness of voice mode manipulation on speaking intensity and phonatory offset-onset mechanisms reflected in spectrograph pattern codes and measures of intervocalic spectral energy change. Voice mode manipulations compared a speaker's habitual or conversational speech intensity and phonatory offset-onset control to that observed when (a) speaking in a *confidential mode,* whereby the speaker is using a breathy or aspiration voice, and (b) a *projected mode,* whereby the speaker is talking under the influence of speech masking noise designed to elicit a Lombard effect and increase speech intensity.

Research Objectives and Hypotheses

Objective #1: Identify self-perception of voice and speech symptoms and impact on QOL in YOPD

Objective 1 was to identify the voice and speech symptoms reported by individuals with YOPD and to determine if these individuals perceive these symptoms as having a negative impact on their quality of life (QOL). To this end, three questionnaires were administered as follows: (a) Visual Analogue Scale (VAS) (Ramig, Pawlas, & Countryman, 1995); (b) the Voice Handicap Index (VHI) (Jacobson et al., 1997); and (c) the 12-Item Short Form Health Survey (SF-12) (Ware, Kosinski, & Keller, 1995). For the VAS task, participants rated the perceived degree of occurrence of nine voice and speech symptoms commonly reported in persons with Parkinson's disease using a visual analogue scale (Ramig et al.). Two validated questionnaires concerning perception of general health (SF-12) and voice-related functioning (VHI) were given to speakers with YOPD and age-matched healthy controls (HC) to determine if there were perceived differences in health- and voiced-related QOL between the two groups (Ware et al., 1995; Jacobson et al., 1997).

Research Question #1(i)

Do YOPD speakers report a higher occurrence of speech and voice symptoms than HC speakers? It was hypothesized that YOPD speakers would report a significantly higher occurrence of voice and speech symptoms that HC speakers.

Research Question #1(ii)

Are there differences between YOPD and HC groups in perception of general health or emotional, functional, and physical restrictions placed on daily living that are specifically voice-related? Quality of life was assessed using six dependent measures derived from the SF-12 and VHI questionnaires. It was hypothesized that YOPD speakers would report perception of poorer general health and report greater psychosocial consequences that place functional, emotional, and physical restrictions on their daily living compared to HC speakers.

Objective #2: Identify speaker differences in clinical tasks of vocal function

Objective 2 was to identify aspects of vocal functioning that are impaired in YOPD speakers using sustained vowel phonation and two routine clinical tasks of laryngeal diadochokinesis (L-DDK). Eleven acoustic voice spectra measures (related to acoustic measures of frequency, intensity, and perceptual attributes of voice quality) were calculated from the sustained vowel phonation stimuli using the Multidimensional Voice Profile™ software package (Kay-Elemetrics, 1999b).

Acoustic measures of phonatory offset-onset control were obtained from two laryngeal diadochokinetic tasks, one involving a laryngealization phonatory offset-onset gesture (‘uh’) and the other involving an aspiration gesture (‘huh’). There were three dependent measures for each gesture (number of repetitions, percent of complete devoicing, and number of pauses).

Research Question #2(i)

In comparison with controls, do YOPD speakers demonstrate differences in acoustic voice spectra measures related to acoustic parameters of frequency, intensity, and perceptual attributes of voice quality? Voice spectra measures previously reported by Kent et al. (1999) as deviant in persons with Parkinson's disease were obtained from the 3-sec. vowel sustained phonation segment. The dependent measures included the following: (a) Measures of average fundamental frequency, variation in fundamental frequency [vF_o]), and perturbation measures of percent jitter and smoothed pitch perturbation quotient (SPPQ); (b) measures of vowel intensity (average peak vowel amplitude), peak amplitude variation (vAM), and perturbation measures of percent shimmer and smoothed amplitude perturbation quotient (SPPQ); (c) spectral tilt (soft phonation index); and (d) the number of unvoiced segments. The following were specific hypotheses with respect to group differences between YOPD and HC speakers:

a. YOPD speakers will demonstrate an increased average fundamental frequency, an increased variation in fundamental frequency, and an increased frequency perturbation.
b. YOPD speakers will demonstrate increased amplitude perturbation, more variability in amplitude and reduced vowel amplitude.
c. YOPD speakers will demonstrate a higher soft phonation index, reflecting a reduction of higher-frequency harmonic

energy (1600-4500 Hz) relative to the lower-frequency harmonic energy (70-1600 Hz) in voice spectra.

d. YOPD speakers will demonstrate a greater number of unvoiced segments.

Research Question #2(ii)

Unlike HC speakers, do YOPD speakers show deficits in phonatory offset-onset control involving the production of utterances that require mechanisms of rapid and repetitive laryngealization and aspiration? It was hypothesized that YOPD speakers would demonstrate impaired phonatory offset-onset control during the production of multiple syllable repetitions for utterances that required mechanisms of both laryngealization and aspiration. The following were specific hypotheses with respect to group differences between YOPD and HC speakers:

a. YOPD speakers will demonstrate fewer number of syllable repetitions per second.

b. YOPD speakers will demonstrate a reduced percentage of complete phonatory offset-onset gestures for both of the L-DDK tasks.

c. YOPD speakers will demonstrate an increased number of pauses during L-DDK syllable tasks.

Objective #3: Effect of speaker group, phonetic context and speaking mode on mechanisms of phonatory offset-onset control

Objective 3 was to determine: (a) speaker group differences on mechanisms of phonatory offset-onset control; (b) the effect of manipulating phonatory offset-onset gestures by using four phonetic

contexts; and (c) the effect of speech production mode, that was manipulated by instructing speakers to read a fairy tale story three times using three different speaking mode conditions. The fairy tale story included 24 fabricated sentences designed to elicit phonatory offset-onset gestures in four phonetic contexts. The fabricated sentence stimuli embedded in the fairy tale included four phonetic contexts (V_V, V_H, V_S, S_V), intending to elicit different phonatory offset-onset mechanisms using six vowels (/i e ae a o u/). The contexts were as follows.

1. The V_V context elicited a phonatory offset-onset gesture requiring a laryngealization gesture without oral constriction.
2. The V_H context elicited phonatory offset-onset associated with an aspiration gesture without oral constriction.
3. The V_S context was associated with an aspiration gesture produced with oral constriction
4. The S_V context elicited both phonatory offset- onset gestures of aspiration (for /s/) followed by laryngealization (prior to vowel onset to mark the word boundary). The symbol _ denotes word boundary.

Each speaker read the story aloud three times using manipulated speaking mode conditions. The three speaking modes were (a) *confidential voice* as elicited by external verbal cueing to speak in a breathy voice; (b) *projected voice* as elicited by external cueing with auditory speech masking along with verbal

cuing to speak “up and over the noise” to elicit changes in vocal effort associated with speaking in a loud*;* and (c) using a *habitual voice* elicited by asking speakers to “read the story using your usual conversational voice.”

The dependent measures of phonatory offset-onset control included nonparametric descriptive codes (degree of devoicing and spectrographic pattern codes) and parametric acoustic measures of average peak vowel amplitude (dB) and intervocalic spectral energy change (dB).

Research Question #3(i)

Do YOPD speakers use average peak vowel amplitude values that are similar to values observed in HC speakers when speaking in a habitual, confidential, and projected mode in three phonetic contexts? The following were specific hypotheses:

a. With respect to speaker group differences, it was hypothesized that YOPD speakers would demonstrate lower peak vowel amplitudes in comparison with HC speakers.
b. With respect to phonetic context effects in both groups, it was hypothesized that peak vowel amplitude values would not differ across the three phonetic contexts.
c. With respect to speaking mode effects in both groups, it was hypothesized that peak vowel amplitude values would be lower in the confidential mode condition with respect to values observed in the habitual mode, and they would be higher in the projected mode relative to the habitual mode.

Research Question #3(ii)

Do YOPD speakers produce changes in intervocalic spectral energy change comparable to that observed in HC speakers? Also, are there differences in intervocalic spectral energy change values associated with different phonetic contexts? Finally, are there differences in intervocalic spectral energy change values associated with manipulation of speaking mode? The following were specific hypotheses:

a. In comparison with HC speakers, YOPD speakers will demonstrate less intervocalic spectral energy change, consistent with impaired phonatory offset-onset control.
b. Intervocalic spectral energy change values will be greater for the laryngealization gesture (V_V context) in comparison with the values observed for the aspiration gesture (V_H, V_S).
c. In comparison with the habitual mode, intervocalic spectral energy change values will be greater in the projected mode and less in the confidential mode, suggesting that speaking in a projected mode improves phonatory offset-onset control.

Research Question #3(iii)

In comparison with HC speakers, do YOPD speakers maintain phonatory offset-onset control as reflected in codes for spectrographic patterns during the production of utterances that require mechanisms of laryngealization (V_V), aspiration without oral constriction (V_H), and aspiration with oral constriction

(V_S) while speaking in habitual, confidential, and projected speech mode conditions? The following were specific hypotheses:

a. Both YOPD and HC speakers will maintain phonatory offset-onset control, as reflected in codes for spectrographic patterns appropriate for aspiration in the phonetic context involving oral constriction (V_S).
b. Unlike HC speakers, YOPD speakers will not maintain phonatory offset-onset control, as reflected in codes for spectrographic patterns appropriate for laryngealization (V_V) and aspiration without oral constriction (V_H) when speaking in both a confidential and habitual speech mode.
c. When speaking in a projected mode, YOPD speakers will demonstrate improved phonatory offset-onset control, as reflected in codes for spectrographic patterns appropriate for laryngealization (V_V) and aspiration without oral constriction (V_H).

Research Question #3(iv)

In comparison with HC speakers, do YOPD speakers use multiple phonatory offset-onset mechanisms when speaking in habitual, confidential, and projected voice modes? The following were specific hypotheses:

a. Unlike HC speakers, YOPD speakers will not demonstrate multiple gestures, as reflected in codes for spectrographic patterns appropriate for both aspiration and laryngealization in S_V phonetic context in the confidential and habitual speech modes.

b. When speaking in a projected mode, YOPD speakers will demonstrate an improvement in production of multiple gestures, as reflected in codes for spectrographic patterns appropriate for both aspiration and laryngealization in S_V phonetic context.

Significance

Findings pertaining to these three research objectives provide the first comprehensive investigation of vocal dysfunction in YOPD. The first objective identifies the impact of vocal dysfunction on physical, functional, and emotional aspects of quality of life. A significant difference in YOPD participants reporting a greater negative impact would suggest a need for voice intervention. The second objective provides acoustic evidence of impaired vocal function in clinical tasks of sustained vowel phonation and laryngeal diadochokinesis. The finding of such impaired vocal function provides potential avenues to explore effective treatment techniques in the YOPD population. Finally, understanding the theoretical issue of whether or not impaired vocal function can be accounted for by a disruption in the habitual setting of laryngeal posture and impaired vocal self-effort provides a an additional step towards future development of a treatment protocol for dysphonic persons with YOPD. Furthermore, the finding of improved vocal function associated with speech masking would focus future efficacy researched aimed at providing effective and efficient treatment techniques for the young-onset population.

Methodology

The current investigation was a prospective study of vocal function in YOPD speakers. Three tasks were completed by gender and age matched groups of YOPD and HC participants as follows: (a) Completion of questionnaires; (b) completion of two clinical tasks of sustained vowel phonation and L-DDK; and (c) reading a fairy tale passage aloud under three manipulated speaking modes.

For the first task, speakers completed three questionnaires. The questionnaire tasks (a) identified perception of voice and speech symptoms, (b) assessed impression of overall physical and mental health status, and (c) determined the negative impact that voice and speech problems had on their daily lives.

To determine vocal deficits, participants completed two routine clinical tasks that assessed phonatory function. The first included the production of a sustained vowel using a normal, habitual voice. Vocalizations were analyzed to derive acoustic spectra measures that correlate perceptually to attributes of voice quality. The second task involved repetitions of the sounds “uh” and “huh.” This task, referred to as laryngeal diadochokinesis (L-DDK), assesses a speaker’s ability to rapidly offset-onset vocal fold vibration using an adductory movement (“uh”) and an abductory movement (“huh”).

To determine speaker group differences on mechanisms of phonatory offset-onset control and the effect of speech production mode, participants read a fairy tale passage aloud. The fairy tale included 24 embedded sentences designed

to elicit different phonatory offset-onset mechanisms in varied phonetic contexts Speaking mode was manipulated by instructing the participants to read the story under three different speaking conditions.

Speaker Recruitment and Selection Criteria

Two matched groups of 12 adults (n=24) between the ages of 30-57 years of age served as speakers. The groups included individuals who were either persons with young-onset PD (YOPD speakers) or healthy controls (HC speakers). Participant selection criteria for both speaker groups are summarized in Appendix A and included a negative history for: (a) smoking, (b) head and neck surgery, (c) respiratory or laryngeal disorders unrelated to YOPD, (d) speech or voice symptoms unrelated to YOPD, (e) language, reading, or learning problems, and (g) substance abuse. Participants were Caucasian, native speakers of standard American English, and they had completed at least a high school education. All participants scored (a) < 19 on the Hamilton Depression Rating Scale, indicating no evidence of a moderate or severe depression (Hamilton, 1960; and (b) > 27 on the Mini-Mental Status, indicating no moderate or severe dementia (Folstein, Folstein, & McHugh, 1975). All participants passed a hearing screening demonstrating hearing sensitivity of at least 25 dB for frequencies .5 kHz, 1 kHz, and 2 kHz and 30 dB for 4 kHz in at least one ear.

YOPD Speakers.

YOPD speakers were recruited from YOPD Support Group meetings in the Baltimore, Pennsylvania, Washington, D.C. and Northern Virginia metropolitan areas (Appendix B). As reflected in Appendix A, YOPD speakers exhibited a

negative history for neurological disease other than PD and a Dysarthria Rating Severity score falling between 8 (speech easily understood but consistent voice symptoms present, speech rhythm and articulation not significantly disturbed) and 9 (speech entirely adequate, minor voice disturbances present). Therefore, YOPD speakers were included for study if they demonstrated no dysarthria or a mild dysarthria as evidenced by voice but not significant articulatory and speech rate disturbances. Also all YOPD speakers were in either a mild or moderate level of disease progression (Stages I – III, Hoehn & Yahr, 1967). If being treated with L-dopa medication, they did not experience extreme on-off fluctuations. Therefore, none of the YOPD speakers evidenced dyskinesia during the time of experimental testing.

The demographics for the YOPD group are summarized in Table 1. YOPD speakers ranged in age from 35-57 years of age and included 7 males and 5 females. This distribution was consistent with the male:female ratio in the PD population, with the incidence being 1.2-1.5 times greater in males (Duvoisin & Sage, 1996; Koller, 1992; Lieberman & Williams, 1993; Wooten et al., 2004). The mean age of symptom onset was 41.6 years (SD = 6.8), and the age of disease onset ranged from 30 to 49 years of age. Mean duration of disease (as reflected by the age the first symptom was noticed by the YOPD person) was 6.5 years (SD = 4.1 years) and ranged from 1 to 16 years. All YOPD speakers were in either a mild or moderate level of disease progression (Stages I-III, Hoehn & Yahr, 1967). Four persons (33%) were not taking anti-parkinson medications. The remaining persons were taking anti-parkinson medications, and all but one of these participants were

taking a combination of two or three drugs that included: (a) dopaminergic agonists (42%), (b) dopaminergic replacement (33%), (c) MAO B-inhibitors (33%), and (d) COMT Inhibitors (17%). In addition, four speakers (33%) were using antidepressants or antianxiety medications.

Table 1. YOPD speaker demographics.

Participant	Sex	Age	Age of symptom onset	Years post-onset	Hoehn & Yahr Stage (1967)	Medications*Δ
01	M	50	49	1	I	none
02	F	55	43	12	II	a,c
03	M	54	49	4	I	Δ
04	M	50	47	2.5	I	none
05	F	50	45	5	II	a,c
06	F	40	30	10	II	c,d,e
07	M	47	41	6	III	d,e,Δ
08	M	53	49	4	I	a
09	F	35	32	3	I	none
10	M	50	41	9	III	a,e,Δ
11	F	57	41	16	III	e
12	M	38	33	5	II	a,c,Δ
Mean (SD)		48.3 (7.0)	41.6 (6.8)	6.5 (4.1)		

*Parkinson medications
a = dopaminergic agonists (parlodel, permax, mirapex); b = anticholinergics (artane, cogentin)
c = MAOB-inhibitors (eldepryl); d = COMT inhibitors (talcapone, entapine); e = dopaminergic replacement (sinemet, lodosyn)
Δ= antidepressants or antianxiety medications (paxil, zoloft, lexoprol)

HC Speakers.

Healthy control speakers were recruited from the investigator's peer acquaintances and met selection criteria stated in Appendix A. These speakers were matched to YOPD speakers for age, gender, and race. The HC speakers demonstrated normal speech and voice quality as judged clinically by the

investigator (Dysarthria Severity Rating score of 10) and were not considered to be *soft-spoken*. Table 2 depicts a summary of age and gender for the YOPD and HC matched speaker pairs. The HC speakers ranged from 30 to 56 years of age with a mean age of 47.92 years. To ensure group equivalency of the two speaker groups, the age scores were tested using an independent *t* test. The age differences between the YOPD and HC groups were not significant $t(22) = 0.11$, $p > .05$).

Table 2. Age and gender characteristics for the two speaker groups.

	YOPD Speakers		HC Speakers	
Speaker Pairs	Age	Gender	Age	Gender
1	50	M	53	M
2	55	F	54	F
3	54	M	56	M
4	50	M	52	M
5	50	F	48	F
6	40	F	40	F
7	47	M	49	M
8	53	M	51	M
9	35	F	30	F
10	50	M	48	M
11	57	F	54	F
12	38	M	40	M
Mean Age	48.3 years		47.9 years	
SD	(7.0)		(7.6)	

Speaker Tasks

The speaker tasks included (a) three questionnaires that assessed participants' perception of voice and speech characteristics, voice-related functioning, and general health; (b) two clinical tasks of sustained phonation and laryngeal diadochokinesis (L-DDK); and (c) a reading task that involved reading aloud 24 fabricated sentences (embedded in a fairy tale passage) that manipulated phonetic contexts with different phonatory offset-onset mechanisms. The fairy tale was read under three counterbalanced speaking mode conditions that varied the speaking mode.

Questionnaires.

Speakers completed three questionnaires. The Visual Analog Scale (VAS) (Ramig et al., 1995) assessed participants' perception of voice and speech characteristics. The Voice Handicap Index (VHI) (Jacobson et al., 1997) assessed voice-related functioning to determine if vocal deficits adversely impacted quality of life. The SF-12 Health Survey (Ware et al., 1995) assessed general physical and mental health.

Visual Analogue Scale (VAS).

The VAS form required participants to indicate the degree of deviancy (using a scale ranging from 0% to 100%) for nine voice and speech characteristics typically reported by persons with PD (Ramig et al, 1995), as listed in Table 3. Four of the characteristics involved deviant voice characteristics of loudness, tremor, hoarseness, and monotone, while three represented deviant speech attributes related to speaker intelligibility (slurs, mumbles, understandability).

Finally, two pragmatic characteristics assessed typical initiation and participation in conversation.

Table 3. Voice and speech characteristics rated by participants using the Visual Analog Scale.

Self-perception of Voice	
Visual Analog Scales	
Loudness	Slurring
Monotone	Mumbling
Hoarse Voice	Understandability
Shaky Voice	Participates in Conversation
	Starts Conversation

Voice Handicap Index (VHI).

The VHI, a standardized tool, assesses an individual's perception of how a voice disorder negatively impacts upon his or her quality of life (Jacobson et al., 1997). As illustrated in Table 4, the VHI consists of thirty items that assess three components: (a) Functional ("My voice makes it difficult for people to hear me"); (b) Physical ("I run out of air when I talk"); and (c) Emotional ("I'm tense when talking with others because of my voice.") components. After reading each item, the participant rated his or her perception as (a) "Never", (b) "Almost never", (c) "Sometimes", (d) "Almost always", or (e) "Always".

Table 4. Summary of quality of life tasks (VHI and SF-12).

Quality of Life (QOL) Tasks	
Voice Handicap Index	SF-12 Health Survey
Total Score	Physical Component
Physical Component	Mental Component
Functional Component	
Emotional Component	

SF-12 Health Survey (SF-12).

The SF-12, a standardized and copyrighted tool, provides a generic measure of health status (Ware et al., 1995). As illustrated in Table 4, it includes one or two items from each of the eight health concepts related to physical functioning (e.g. role limitations due to physical health problems, bodily pain, general health, vitality, and social functioning) and mental health (psychological distress, psychological well being, and role limitations due to emotional problems). Permission to use the SF-12 was granted from the Medical Outcomes Trust (License Agreement F1-100402-12025).

Clinical Tasks.

Participants completed two routine clinical tasks. The tasks were sustained vowel phonation and laryngeal diadochokinesis (L-DDK). These two clinical tasks and dependent measures are summarized in Appendix D.

Sustained Vowel Phonation.

Sustained vowel phonation has been routinely used for assessment of speakers who have dysarthria. As noted by Duffy (1995) "The simplest task for isolating the respiratory-phonatory system for speech is vowel prolongation." (p. 83). The vowel "ah" was used in the present study because of its frequent use in dysarthria assessment (Duffy, 1995). Clinicians typically use sustained vowel phonations to make auditory-perceptual judgments related to (a) pitch and pitch stability; (b) loudness and loudness stability; (c) parameters of voice quality such as harshness, hoarseness, roughness, and breathiness; (d) tremor; and (e) pitch breaks. Audio-recorded sustained vowel phonations can be analyzed acoustically to obtain voice spectra measures that correlate with auditory perceptual judgments of voice quality; and the validity and reliability of voice spectra measures have been established (Kent, Vorperian, & Duffy, 1999). Therefore, the sustained vowel phonations provided measures to address one aspect of vocal deficits in YOPD speakers.

L-DDK Tasks.

Diadochokinetic tasks have been reportedly used for determining the speed and regularity of reciprocal movements of the speech articulators (Duffy, 1995). The two L-DDK tasks included in the present study consisted of rapid syllable repetitions for 7 s of the syllables "uh" and "huh." Rapid repetitions of the syllable "uh" during a seven second interval has been referred to as laryngeal adductory diadochokinesis (Ptacek et al., 1966). This task assesses the speaker's ability to produce rapid, successive offset-onset of phonation using an adductory

laryngealization gesture that involves an active mechanism relying on the actions of intrinsic laryngeal muscles. Performance with respect to syllable repetition rate per second is compared using normative means and standard deviations reported by Ptacek, Sander, Maloney, and Jackson, 1966 (adult male mean = 5.1/sec, standard deviation = 1.0; female mean = 5.3/sec, standard deviation = 0.8). A *z* score > 1 is considered abnormal (Ptacek et al., 1966).

Rapid repetition of the syllable "huh" during a seven second interval was used to assess the speaker's ability to produce rapid offset-onset of phonation using an abductory aspiration gesture also involving an active mechanism relying on the actions of intrinsic laryngeal muscles. Normative data are not available to permit calculation of *z* scores for this task, but measures were used for between-group comparisons in this investigation. Therefore, performance on these two L-DDK tasks, involving the ability to rapidly and repeatedly produce phonatory offset-onset gestures involving vocal adductory movement ("uh") and abductory movement ("huh") provided an additional means of describing vocal dysfunction in YOPD speakers.

Fairy Tale Passage.

The third speaker task involved reading aloud fabricated sentences embedded in a fairy tale story. Four phonetic contexts were manipulated to elicit phonatory offset-onset mechanisms associated with voice stoppage. The contexts included (a) a laryngealization gesture without oral vocal tract constriction (vowel to vowel word boundary); (b) aspiration (frication) without vocal tract constriction (vowel to /h/ word boundary); (c) aspiration (frication) with oral vocal tract

constriction (vowel to /s/ word boundary; and (d) a multiple gesture combination of aspiration (frication) with oral vocal tract constriction followed by a laryngealization gesture without oral vocal tract constriction (/s/ to vowel word boundary) (as summarized in Appendix E and Table 5). The experimental task was completed under three-counterbalanced speech mode conditions (Appendix F). The three speaking modes were (a) *confidential voice* that involved external verbal cueing to speak in a breathy, aspirant voice; (b) *projected voice* that was elicited by auditory speech masking (Lombard effect) along with verbal cueing to speak "up and over the noise" to elicit changes in vocal effort associated with speaking in a full, loud voice; and (c) *habitual voice* elicited by asking speakers to "read the story using your usual conversational voice."

Phonetic Context.

The voicing control sentences were constructed to elicit phonatory offset-onset mechanisms. The 24 sentences, listed in Table 5, are arranged according to six vowels (rows) across four phonetic contexts (columns).

Table 5. Summary of voicing control sentences for four utterance types and 6 vowel types.

Vowel	(V_H)	(V_V)	(V_S)	(S_V)
/i/	Thee_heap will be _heeding thee _heat.	Thee_ east will be _eating thee_ eve.	Thee_seam will be _seeking thee_ scene.	Miss _Eve was eating this _eave.
/u/	Thee _hoop will be _hooting the_ hoot.	Thee _ooze will be _oozing thee_ oot.	Thee _suit will be _soothing thee_ soup.	Miss_ Oot was oozing this_oop.
/e/	Thee_ hake will be _hating thee _Hague.	Thee_ aide will be _aching thee_ape.	Thee_ safe will be_ saving the_sage.	Miss _Aide was aching this_ ape.
/o/	Thee_ host will be _hosing thee_ home.	Thee_ oath will be _owning thee_ oak.	Thee_ soap will be _sewing thee_ soak.	Miss_ Ode was owning this_ oak.
/ae/	Thee_ hat will be _hashing thee_ hat.	Thee_ act will be _adding thee _add.	Thee_ sax will be _ sacking thee_ sash.	Miss_ Att was acting this _ad.
/a/	Thee _hop will be _hopping thee _hot.	Thee_ ox will be _oxing thee odd.	Thee_ sod will be _sopping thee_sock.	Miss_ Odd was oxing this_ ox.

- (_) denotes word boundary for phonatory offset-onset mechanism
- The symbol V refers to vowel, while all other consonants refer to the target phonemes (e.g. /h/ and /s/)

Each of the six vowel types (e.g., /i/, /u/, /e/, /o/, /ae/, /a/) included four sentences, one for each phonetic context (e.g. V_H, V_V, V_S, S_V). The symbol V refers to vowel and the underline (_) denotes a word boundary for elicitation of target phonatory offset-onset mechanisms. Each phonetic context was intended to elicit different phonatory offset-onset mechanisms as follows:

V_V = Phonatory offset-onset with a laryngealization gesture (glottal stop or glottal fry) (thee east).

V_H = Phonatory offset-onset with an aspiration gesture (frication) without vocal tract constriction (thee heap).

V_S = Phonatory offset with an aspiration gesture (frication) with oral vocal tract constriction (thee seam).

S_V = Combination of both phonatory offset-onset mechanisms (aspiration with oral vocal tract constriction [frication] and laryngealization (Miss Eve).

The phonetic contexts were blocked according to the six vowel sounds to control for potential vowel effects on phonatory offset-onset control (Allen, 1970; Higgens, Netsell, & Schulte, 1998; and Umeda, 1978). Only tense vowels were used, selected to control for tongue height and front/back differences (Kent, 1997).

Each fabricated sentence had a consistent subject-verb-object clause construction and included three phonatory offset-onset tokens of the same phonetic context, with the exception of the S_V context that combined both devoicing gestures. In these sentences, the second token included a voiced /z/ due to grammatical constraints. Therefore, only the first and last phrases were appropriate for measurement in each of the six S_V sentences. The phoneme

occurring prior to the phonatory offset-onset token was held constant for each phonetic context and was the stressed phoneme /i/. The rationale for choosing this phoneme was that the second and third formants for /i/ are close together in comparison with the other point vowels. The high frequency formant energy assisted in identifying the point where phonatory offset-onset occurred, particularly for the fricatives. Real words were used; however, nonsense words were required in five instances to achieve phonetic balance.

The 24 sentences were embedded in a fairy tale reading passage (Appendix F). At six intervals there were four sentences occurring consecutively, one sentence for each phonetic context. Each of the six intervals represented a different vowel-type. There were three versions of the fairy tale story to permit randomization of vowel order across the three speaking context conditions. That is, the story remained constant, but each version provided a different sentence vowel order, as described in Appendix F.

Speaking Mode.

Speakers read the fairy tale to elicit phonatory offset-onset. The order of the three speaking modes was counterbalanced (see Appendix F for detailed information regarding counterbalancing). The three speaking modes were as follows:

(a) *confidential voice* that involved external verbal cueing to speak in a breathy, aspiration voice; (b) *projected voice* that was elicited by auditory speech masking (Lombard effect) along with verbal cueing to speak "up and over the noise" to elicit changes in vocal effort associated with speaking in a full, loud voice; and (c)

a *habitual voice* elicited by asking speakers to "read the story using your usual conversational voice."

Data Collection Procedures

Experimental testing was completed during a two-hour session conducted either at the University of Maryland Voice and Fluency Lab (College Park, MD) or the Towson University Speech and Voice Lab (Towson, MD). Documentation of informed consent was obtained from all participants prior to beginning data collection procedures (Appendix C).

Testing Environment.

The participant screening procedure was completed in a quiet environment with only the investigator present. The hearing screening, questionnaires, and audio-recorded tasks were conducted in an IAC Sound Booth with an ambient room noise level of 68 dB SPL in both lab settings at Towson University and University of Maryland College Park.

Audio-recording Equipment.

Recordings of three trials of prolongation of the vowel "ah," both L-DDK tasks, and the fabricated sentences in a fairy tale story were made using a digital audiotape (DAT) recorder (Tascom Model DA-PI) with a sampling rate of 48kHz and an AKG C420 (AKG Acoustics) head-mounted microphone (with frequency range 20-20kHz). A constant 4 cm speaker mouth-to-microphone distance was maintained for all trials to decrease room noise contamination. Signal level was adjusted using the VU meter of the DAT to prevent overload distortion (signal peak clipping).

Calibration Procedure for Intensity.

Speech intensity calibration procedures were necessary to permit both within-speaker and between-speaker comparison of speech intensity for the sustained vowel phonation and sentence stimuli tasks. Procedures reported by Sapienza and Dutka (1996) and modified by Bassich (2001) and Gartner-Schmidt (2003) were used to calculate absolute dB SPL levels. Such modification was needed to account for differences in the acoustic analyses software programs used by Sapienza and Dutka and in the present investigation. These procedures were as follows: (Gartner-Schmidt, pp. 52-53):

> A pure tone audiometer (Maico, Model MA 41) was used as a tone generator positioned 4 cm from a digital sound level meter (SLM) (Radio Shack, Model 33-2055) at a 45-degree angle of incidence (directly in line with the SLM microphone). A 1 kHz tone was generated and presented by a Sony Active speaker (SRS_88PC). As the tone was generated, it was recorded by a microphone into the digital audio recorder at a 4 cm distance to simulate the precise experimental set-up with the participants. As the tone was being recorded, the numerical value was verbally noted from the digital SLM, using C-weighting as the intensity level and recorded onto the DAT for later reference. The Computerized Speech Laboratory (CSL™ Model 4300) (Kay Elemetrics, 1999a) was used to determine relative (dB) measures of the calibration tone. During subsequent analysis, this value was factored into the following equation to determine absolute dB SPL of the speech sample.

$$\mathbf{y = (b + x) - a}$$

y = absolute dB SPL of vocal utterance
b = relative dB value of vocal utterance from CSL
x = absolute dB SPL of Cal tone from SLM
a = relative dB of Cal tone from CSL

Auditory Masking Noise.

The auditory masking noise used to elicit a Lombard effect while reading the fairy tale aloud for the projected mode consisted of a speech-weighted noise source generated by a Maico audiometer (Model MA 41) presented binaurally through insert headphones (ER-2, Etymotic Research). The spectrum of the speech noise emitted a frequency range from 250Hz to 8kHz and consisted of white noise that was filtered 12 dB per octave above 1000 Hz to simulate the long-term average spectrum of conversational speech. This filtering of the white noise provided relatively more energy in the low frequencies thus approximating the frequency-energy distribution of speech (Katz, 2002). The masking noise level was calibrated and checked at monthly intervals at the Towson University Speech, Language, and Audiology Clinic using a Bruel and Kjaer DB0138 2-cc adaptor and 4152 coupler, 1 inch condenser microphone (type 4144) and a 2209 sound level meter.

Auditory Masking Procedure.

Procedures reported by Ho et al. (1999) were used to determine the appropriate masking level used to elicit a Lombard effect. Prior to recording the projected mode condition, speakers were fitted with insert earphones, and the masking noise was presented binaurally at the lowest level possible for the audiometer. This level was below the auditory threshold level for all speakers. The noise level was increased in 2 dB increments until it was detected by the speaker. Noise was then decreased by 10 dB, and again increased in 2 dB increments to confirm the previously detected auditory threshold level. This

procedure was repeated a third time, and the modal auditory threshold value was noted.

After the threshold level was determined for the masker, the speaker began reading the fairy tale story while the investigator increased the noise level in five dB increments until the speaker was using an *optimal* projected voice mode. Optimal was operationally defined as > 5 dB increase in speech intensity (SPL) from the habitual speaking condition as measured by a sound level meter. Once the appropriate masking level was determined, the speaker was instructed to begin the story again, and the audio-recorder was turned on. Speakers were instructed to alert the investigator if the noise level became uncomfortable. The masking level did not exceed 90 dB for any of the speakers. None of the speakers reported that the masking level was uncomfortable. All masking levels were below the standards specified by Federal Regulations, which allow for exposure of 100 dB SPL for up to 30 minutes (Walsh-Healy Noise Standard, as reported by Durrant & Loverinic, 1994; Lipscomb, 1994). Group data for the noise threshold level (dB) and masking level (dB SPL) are presented in Appendix J. The mean noise threshold for YOPD speakers was 8.9 (SD = 7.6), and the mean for the HC speakers was 8.6 (SD = 5.0). This difference was not statistically significant. Also, the mean masking level for YOPD speakers was 71.4 (SD = 8.7), and the mean for the HC speakers was 70.6 (SD = 6.5); again this difference was not statistically significant

Testing Protocol.

Prior to participation in the study, all participants answered brief interview questions (Appendix A). During the interview, the investigator, a speech-language pathologist with 25 years experience with PD, used criteria from the Dysarthria Rating Scale to perceptually classify the presence and severity of voice and speech symptoms for each participant (Yorkston, Miller, & Strand 1995). Also, YOPD speakers were interviewed with respect to their disease progression and symptoms. The information was then used to determine the level of disease progression (Hoehn & Yahr, 1967).

After the initial interview, participants completed the Hamilton Depression Rating Scale (Hamilton, 1960) and were administered the Mini-Mental Exam (Folstein et al, 1975). Appendix H summarizes the YOPD and HC group mean and 95% confidence interval for the depression, dementia, and dysarthria severity rating scores, verifying that all participants met pre-determined selection criteria.

Following the completion of the depression and dementia tasks, all participants passed a pure tone hearing screening test at 25 dB HL for frequencies .5 kHz, 1 kHz, 2 kHz, and 30 dB HL @ 4kHz in at least one ear. The hearing screening was conducted in a sound attenuated booth using a Maico audiometer (Model MA 41) that was calibrated at monthly intervals.

Following completion of the hearing screening, data collection was initiated and included the order as follows:

1. L-DDK Task
2. Familiarization Task

3. Sustained Vowel Phonation Task
4. Fairy Tale Passage: Experimental Condition 1
5. Break (filling out questionnaires)
6. Fairy Tale Passage: Experimental Condition 2
7. Break (filling out questionnaires)
8. Fairy Tale Passage: Experimental Condition 3

The order of tasks was not randomized as the protocol included a hierarchy of speech motor tasks. The first two tasks, L-DDK and sustained phonation, are performance tasks that may be adversely affected by vocal fatigue, particularly in persons who are neurologically impaired. Therefore, data were collected in a timeframe that intended to avoid fatigue effects.

L-DDK Task.

Participants were instructed to repeat the syllable (either "uh" or "huh") as rapidly and distinctly as possible. The investigator modeled the task for a 7-sec interval using a syllable repetition rate of 5 per second, producing complete glottal stops between each syllable repetition for "uh," and a complete abductory (aspiration) phonatory offset-onset gesture between each syllable repetition for "huh." Participants practiced each L-DDK task and were provided with feedback to "go as fast as they can while keeping each syllable precise and distinct." Following practice, one trial of each syllable repetition string was audio-recorded. The order of syllables was randomized across speakers. The investigator instructed participants to "Start," and then "Stop" at the end of a 7-sec duration.

Familiarization Task.

Following the recording of the L-DDK tasks, speakers were given a list of the 24 fabricated sentences designed to elicit various phonatory offset-onset mechanisms. The investigator corrected any mispronunciations. Then the entire fairy tale story was read for practice and familiarization. The familiarization task was audio-recorded but not analyzed. This procedure provided a "warm-up" and controlled for practice effects. The familiarization task lasted approximately 10 minutes.

Sustained Vowel Phonation Task.

Participant instructions for the sustained phonation tasks were identical to those used by Kent et al. (1999) who reported reliability of these procedures in 33 individuals with dysarthria (10 participants had PD). Participants were instructed to maintain a steady phonation using their *best, clear voice*. The investigator demonstrated the task performance. Three consecutive sustained vowel productions were audio-recorded and used to derive acoustic measures of voice spectra that relate to perceptual parameters of pitch, loudness, and voice quality.

Fairy Tale Passage: Experimental Conditions.

Following audio recording of the sustained vowel phonation task, the speaker was given one of the three versions of the fairy tale and instructed to read the story aloud using one of the three speaking modes. The order of the three speaking modes was counterbalanced. See Appendix F for a description of the counterbalancing technique. In the habitual mode speakers were instructed as follows:

> I would like you to read this Fairy Tale aloud, using your normal conversational voice. Pretend that you are reading this tale to a small group children, so speak clearly and distinctly. The bolded sentences that you practiced earlier are tricky and some of them do not make sense. If you make a mistake or misread a word, please repeat the entire sentence.

For the confidential mode speakers were instructed as follows:

> I would like you to read this Fairy Tale aloud, using a very quiet and hushed voice, like this. (Investigator demonstrates breathy quality or a *confidential voice*). Pretend you are reading to the young children in a secretive or confidential manner. That is, use the voice you would use to tell a very important secret, being careful not to be overheard by anyone else. But, be careful not to whisper. The bolded sentences in the story that you practiced earlier are tricky and some of them do not make sense. If you make a mistake or misread a word, please repeat the entire sentence.

For the projected mode, speakers were instructed as follows:

> You are going to hear a rushing sound in the earphones. I want you to read the fairy tale aloud. It is important that you speak up and over the noise so that you may be heard. Pretend that you are reading and communicating to a group of people. Also the bolded sentences in the story that you practiced earlier are tricky and some of them do not make sense. If you make a mistake or misread a word, please repeat the entire sentence.

Break 1.

After completing the first experimental reading, participants were allowed a 10-minute voice break. Participants were encouraged to drink bottled water to maintain vocal fold hydration and to leave the sound treated room to walk around and stretch. Following the break, they returned to the sound treated room and completed one or two of the three questionnaires.

Completion of Questionnaires.

The VAS, VHI, and SF-12 questionnaires were each completed at the end of the 10-minute rest period. The VHI was completed during one break, and the VAS and SF-12 were completed during the other break. The order of the completion of questionnaires was randomized across the participants.

For the VAS task, participants were given a form that listed nine deviant voice and speech characteristics. For each characteristic, the attribute was described in layperson terminology. Below the attribute there was a horizontal line, 16.5 cm in length, with anchors provided at the left ("Always a shaky voice") and right ("Never a shaky voice"). Each participant was verbally instructed to place a mark on the line between the two anchors that best represented his or her typical speech. Participants were encouraged to ask questions if they were uncertain about the definition of a particular attribute. The instructor provided verbal description but did not model the attribute.

The directions for the VHI and SF-12 included written instructions in accordance with the standardized procedures reported for the normative studies (Jacobson et al., 1997; Ware et al, 1995). For the VHI, participants were asked to

read each of the 30 items, and indicate one of five choices that reflected their opinion ranging from “Never” to “Always.” The SF-12 included explicit written instructions and examples that guided the participants through the completion of the multiple-choice questions.

Following the break, the speaker was given another of the three versions of the fairy tale and instructed to read the story aloud using a different speaking mode from that used in Experimental Condition 1. Following the second experimental reading, the speaker had a 10-minute rest break similar to the first, and they completed the remaining questionnaire(s). Following the second break, the speaker was given the remaining version of the fairy tale and instructed to read the story aloud in the third speaking mode condition. Experimental testing was concluded at the end of this final reading.

Data Reduction Procedures-Dependent Measures

Prior to statistical analyses, the VAS, VHI, and SF-12 questionnaires were scored. Additionally, the audio-recorded clinical phonatory tasks and sentence stimuli were prepared for acoustical analyses.

Scoring of Questionnaires.

For the VAS data, measurement procedures used were identical to those reported by Ramig et al (1996). The distance between the beginning of a line (left) to the point where the mark was placed (in cm) was measured for each vocal attribute. The total length of the line was 16.5 cm. The measured distance was divided by the total length and multiplied by 100 to obtain a percentage of perceived deviance for each attribute. Higher values indicated greater impairment.

The VHI was scored by summing the number of total points. Additionally, a Physical Component, Emotional Component, and Functional Component score was obtained for each speaker. A response of "Always" was scored 4 points and "Never" was scored 0 points. The remaining responses were scored between 1 ("Almost never") and 3 points ("Almost always"). The component scores were also summed to determine a Total Voice Handicap Score. The component and total score values were compared to published norms obtained from 65 voice disordered patients seen in the Voice Clinic at Henry Ford Hospital (Jacobson et al, 1997).

The SF-12 was scored electronically as arranged through the License Agreement. A Physical and Mental Component score was calculated for each participant. The standardized (based on a sample of 2,329 persons) mean for each component is 50 and the standard deviation is 10. Therefore, the components scores were transformed to a normalized *z* score prior to statistical analyses.

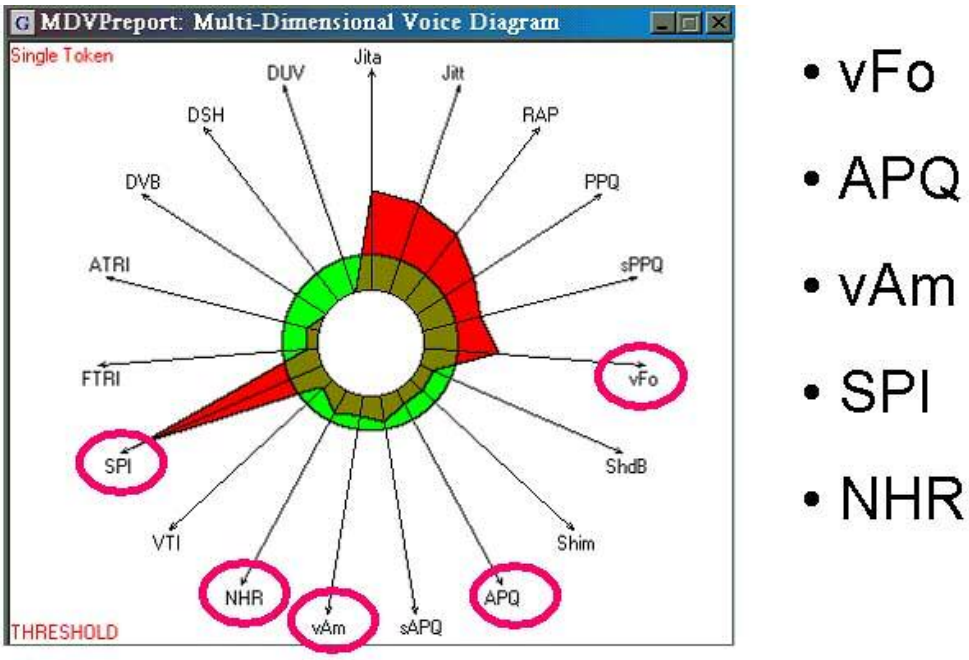
Preparation of Audio-Recorded Tasks for Acoustic Analyses.

The three sustained vowel phonations, the two L-DDK syllable repetitions, the 24 sentences embedded in each fairy tale speaking mode condition, and the calibration tone for each speaking mode were transferred to the Computerized Speech Laboratory (CSL™ Model 4300) (Kay-Elemetric, 1999a) by connecting the digital DAT audio-recorder output to CSL digital input during capturing. This ensured a sampling rate of 48kHz. The captured signal streams were stored for acoustic analyses.

Acoustic Analyses of Sustained Vowel Phonations.

The three sustained vowel phonations were analyzed using the Multidimensional Voice Profile™ software program (MDVP) (Kay-Elemetric, 1999b) and procedures reported by Kent et al. (1999). Figure 5 illustrates the MDVP generated Voice Spectral Radial Graph. The radial graph depicts the speaker's values for 19 extracted parameters of voice quality. The circles represent the normal threshold. Values that fall outside of the circles represent deviant performance (as represented by 2 SD from the norms reported by the manufacturer, Kay Elemetrics, 1998b). Reliability of MDVP generated analysis of Parkinson's speakers' voices has been established by Kent et al. (1999) for sustained phonations. The five parameters that are circled have been reported to be most deviant in PD speakers. As this study was the first to explore parameters in YOPD speakers, eight parameters were chosen to represent values of frequency variation (jitter) along with the intensity variation (shimmer) measures. Also, pilot analyses indicated that some YOPD speakers demonstrated increased values in the degree of unvoiced segments (DUV). In addition to these eight parameters, the average fundamental frequency and average speech intensity level was also included for measurement. In Figure 5, the five parameters included in the Kent et al. study are circled and those used also in the present study are noted by an asterisk.

Figure 5. Summary of MDVP voice spectra measures.



*VF_o = Variation in Fundamental Frequency
APQ = Amplitude Perturbation Quotient
*vAM = Variation in Amplitude
*SPI = Soft Phonation Index
NHR = Noise Harmonics Ratio

* indicates parameters used in the present study

The present investigation included the following parameters (as described in the MDVP Manual, pp. 15-19, Kay-Elemetrics, 1999b):

1. Fundamental Frequency (F_o) Measures
    a. Mean F_o (Hz) represents the averaged fundamental frequency for the three second analyzed voice sample.
    b. F_o Variation (%) (vfo) is the relative standard deviation of the period-to-period calculated fundamental frequency. It reflects the long-term variations of F_o and provides a measure of cycle-to-cycle phonatory stability.
    c. Frequency Perturbation Measures
        i. Jitter Percent (% Jitter) is the relative evaluation of the period-to-period (very short-term) variability of F_o within the 3-second analyzed voice sample, excluding voice breaks.
        ii. Smoothed Pitch Period Perturbation Quotient (%) (SPPQ) is the relative evaluation of the short or long-term variability of the cycle period length within the analyzed three second analyzed voice sample with a smoothing factor of 55 periods. Voice break areas are excluded.
2. Intensity Measures
    a. Average Intensity (dB) for the 3 sec analyzed voice sample. The relative dB value obtained from the MDVP program was converted to absolute dB using the calibration procedures previously described.
    b. Peak Amplitude Variation /%/ (vAM) is the relative standard deviation of the period-to-period calculated peak-to-peak amplitude. Like vF_o, it reflects the long-term amplitude variations of the voice sample.
    c. Amplitude Perturbation Measures
        i. Smoothed amplitude perturbation quotient (SAPQ) is the relative evaluation of the period-to-period variability of the peak-to-peak amplitude (e.g. shimmer) using a smoothing level of 11 periods.
        ii. Percent Shimmer is the relative evaluation of the period-to-period (very short-term) variability of the peak-to-peak amplitude within the 3 sec analyzed voice sample.
3. Soft Phonation Index (SPI) provides an estimate of spectral tilt, as it is the average ratio of the lower-frequency harmonic energy in the range 70-1600 Hz to the higher-frequency harmonic energy in the range 1600-4500 Hz.

4. Number of Unvoiced Segments detected within the 3 sec analyzed voice sample, including glottal fry.

Using the Kent et al. (1999) procedures, a 3-sec voice sample that avoided proximity to the initial and terminal portions of the vowel was marked on the digitized recordings to exclude variations occurring with phonation onset and offset. A beginning cursor was placed 500 ms after phonatory onset, and the end cursor was placed 3 sec from the beginning cursor. In conformance with procedures reported by Kent et al., three sustained vowel phonations were measured for each speaker. Therefore, values of the three tokens were averaged automatically for each parameter and the mean values for each speaker were used for purposes of statistical analyses.

Acoustic Analyses of L-DDK Tasks.

The 7-sec L-DDK segments for "uh" and "huh" were displayed using spectrograms with a frequency range of 0-7 kHz. using CSL™ Model 4300 (Kay-Elemetric, 1999a). The analysis size was set to 512 points and the default window weighting parameter was used (Blackman). Figure 6 illustrates the spectrogram for "uh" and Figure 7 illustrates the one for "huh" for a healthy control speaker. The number of complete syllable productions was counted.

Figure 6. Waveform (top) and spectrogram (bottom) for a 7-sec segment of "uh."

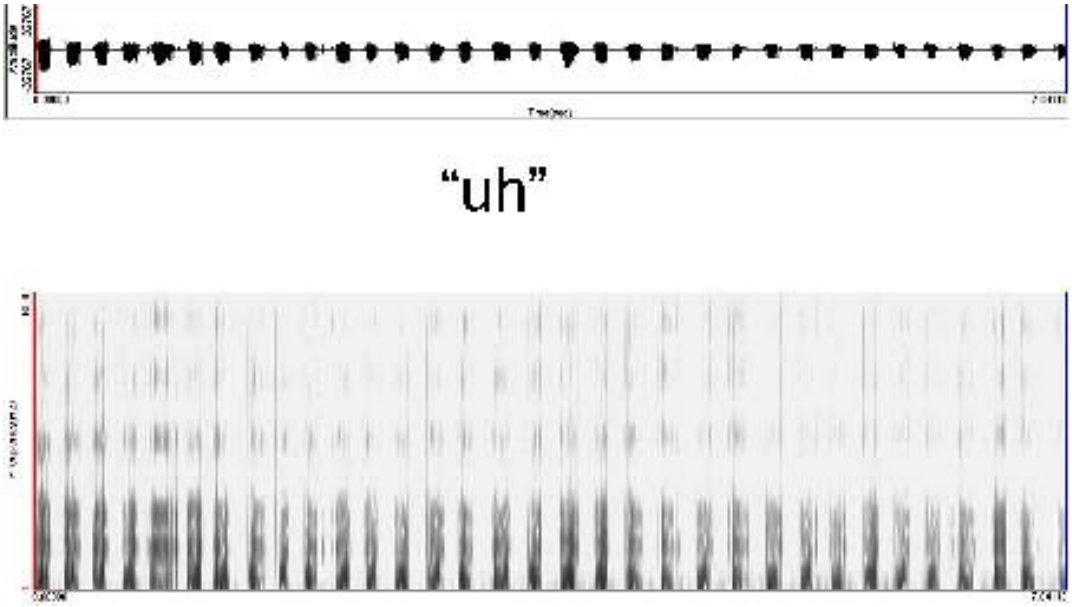


Figure 7. Spectrogram for a 7-sec segment of "huh."

Furthermore, smaller 1-sec segments, that included only four or five syllables, were subsequently displayed to permit a more detailed view of the intervocalic segments where phonatory offset-onset should occur, Figure 8 illustrates the narrowband spectrogram for “uh” and Figure 9 illustrates the spectrogram for “huh.”

Figure 8. Spectrogram for a 1-sec segment of "uh."

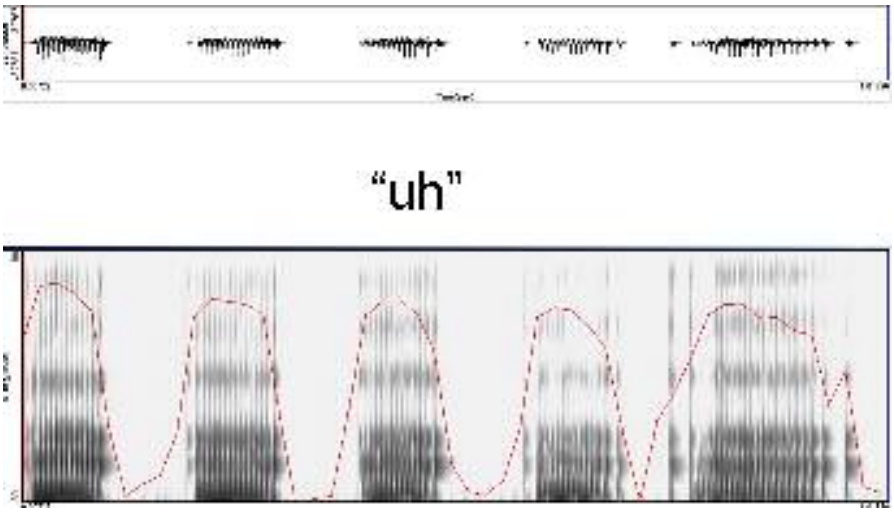
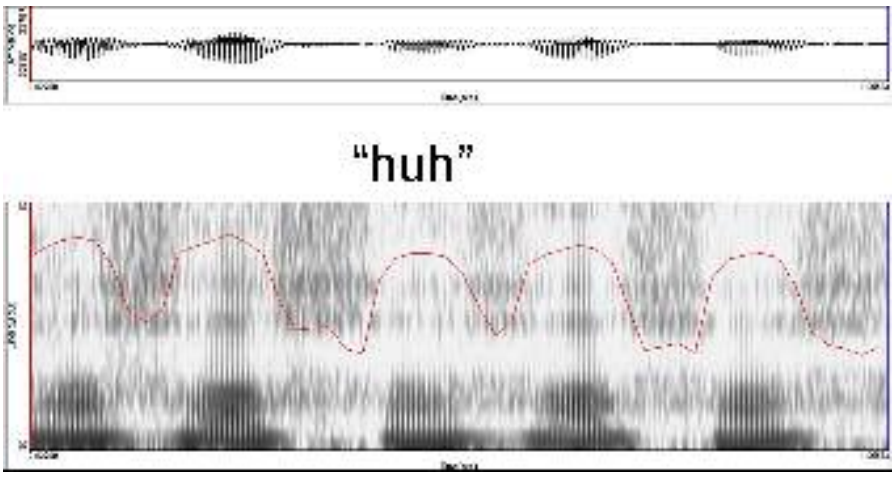


Figure 9. Spectrogram for a 1-sec segment of "huh."



Using this enhanced time resolution, all intervocalic segments were nominally coded as “complete devoicing”, “partial devoicing”, or “no devoicing.” The adductory “ah” intervocalic segment was coded as *complete* when a complete glottal stop was produced, defined as a 25 ms or greater gap. However, durations > 200 ms were operationally defined as pauses and not considered to be glottal stops. Pauses were edited out of the segment and compensatory adjustments in the 7-sec utterance segments were made. The number of complete glottal stops was divided by the total number of syllables produced in the 7-sec segment to determine the percent of complete adductory devoicing. Similarly, for the abductory “huh” syllable, the number of complete phonatory offsets was counted. Complete phonatory offset for this syllable was defined as complete cessation of vocal fold vibration and accompanied by frication. The number of complete aspiration phonatory offset-onsets was divided by the total number of syllables produced in the 7-sec segment to determine the percent of complete abductory devoicing.

Acoustic Analyses of Sentence Stimuli.

Spectrograms were produced for each sentence using CSL with a frequency range of 0-7 kHz, analysis size of 512 points, and the default Blackman window. When coding each phonatory offset-onset in an utterance, only the target phrase was displayed to permit a detailed view of the intervocalic segment where a word boundary occurred. An energy contour was superimposed on the spectrogram, with a 20 ms frame advance and 20 ms window display, as illustrated in Figures 10 – 18.

The dependent measures are summarized in Table 6 and Appendix E. Two dependent measures involved a descriptive categorical coding for (a) spectrographic patterns reflecting mechanisms of phonatory offset-onset; and (b) a numeric coding of the S_V phonetic context reflecting speaker production of multiple gestures. These coding schema were developed in a pilot study and are similar to the coding of HC and laryngectomy speakers by Gartner-Schmidt (2003). Two other dependent measures involved acoustic measures of average vowel intensity (dB) and intervocalic spectral energy change (dB).

Table 6. Summary of dependent measures for sentence stimuli.

Dependent Measures
Spectrographic pattern codes
Production of a single versus a double gesture
Mean peak vowel amplitude (Speech Intensity)
Intervocalic spectral energy change

Spectrographic Pattern Codes.

A coding system was developed to categorize spectrographic patterns reflecting mechanisms of phonatory offset-onset voicing control. The coding system was applied to the first and second phonatory offset-onset tokens for the V_V, V_H, and V_S contexts. The phonatory offset-onset token occurring at the end of the sentence was not coded to exclude utterance end effects (Fledge & Brown, 1982). The V_V, V_H, and V_S phonetic contexts each contained two targeted intervals for coding (18 sentences x 2 tokens per sentence = 36 tokens for each phonetic condition, for each speaker). The spectrographic patterns were coded with a value ranging from 0 to 6, as illustrated in Table 7.

Table 7. Summary of spectrographic pattern codes for phonatory offset-onset gestures.

Code	Type	Spectrographic Pattern
0	No phonatory offset-onset	None
1		Continuous phonation without aspiration or laryngealization but with change in high frequency spectral energy or energy level
2	Laryngealization	Glottal fry without spirantization
3		Glottal stop without spirantization
4		Glottal fry or stop with spirantization
5	Aspiration	Aspiration with continuous voice bar
6		Aspiration with no voicing

Codes of 0 and 1 were applied to both the laryngealization and aspiration gestures.

Codes of 2, 3, and 4 were specific only to the laryngealization gesture and codes of 5 and 6 pertained to the aspiration gesture.

Code 0: Aspiration or laryngealization gestures associated with no change in vowel spectra or energy to suggest phonatory offset-onset during the word boundary or intervocalic interval. As illustrated in Figure 10, the speech waveform (upper panel) is continuous. The superimposed energy contour in the lower panel indicates no decrease in acoustic energy associated with word boundary. The speaker in Figure 10 did not use a laryngealization gesture for mark the word boundary. Similarly, the speaker in Figure 11 did not use an aspiration gesture appropriate for a glottal fricative.

Figure 10. Speaker producing the phrase "thee east" without using a laryngealization gesture to mark the word boundary.

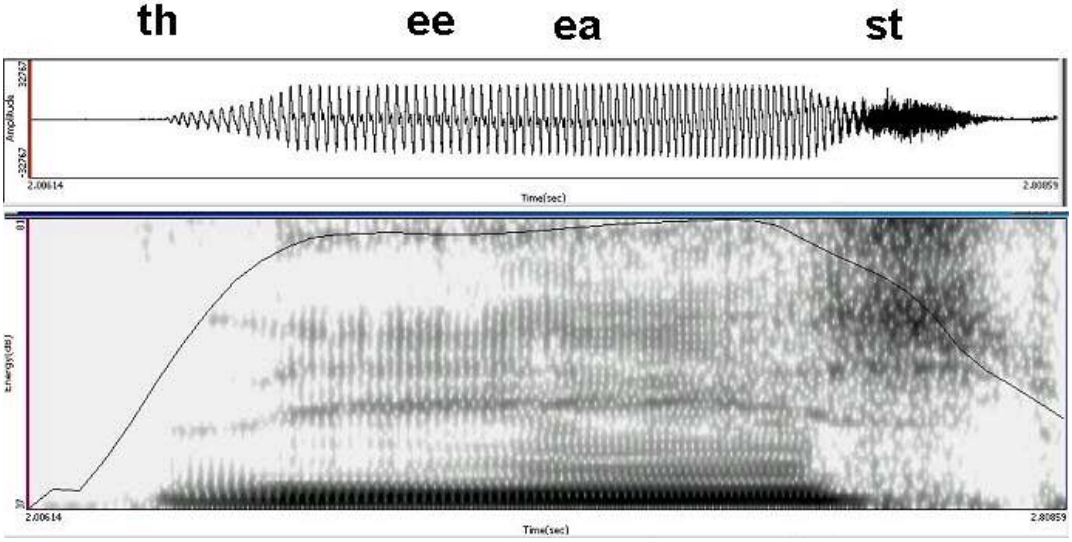
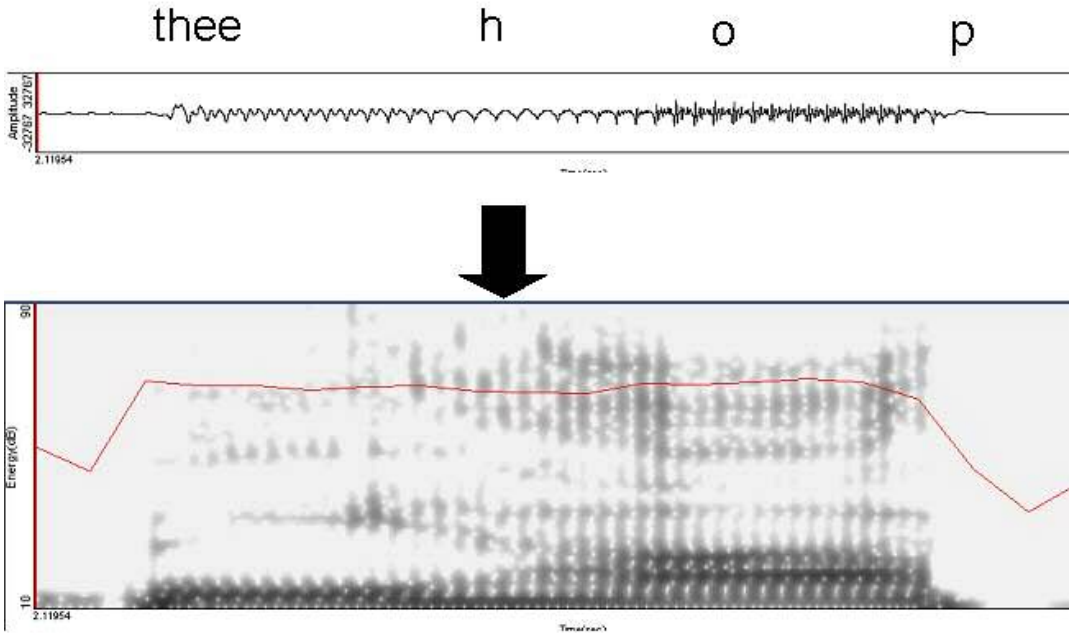
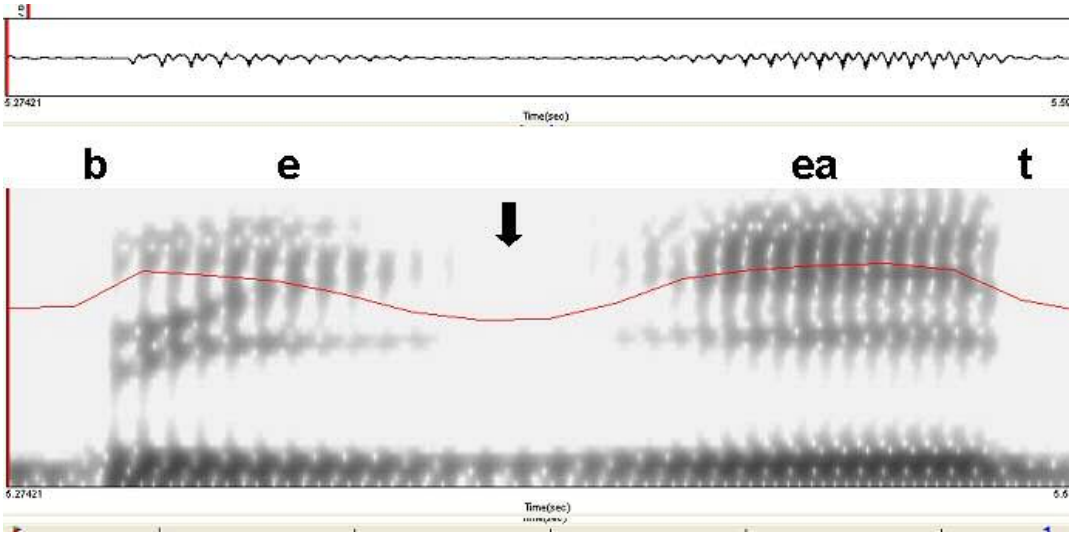


Figure 11. Speaker producing the phrase "thee hop" without using an aspiration gesture for a glottal fricative.



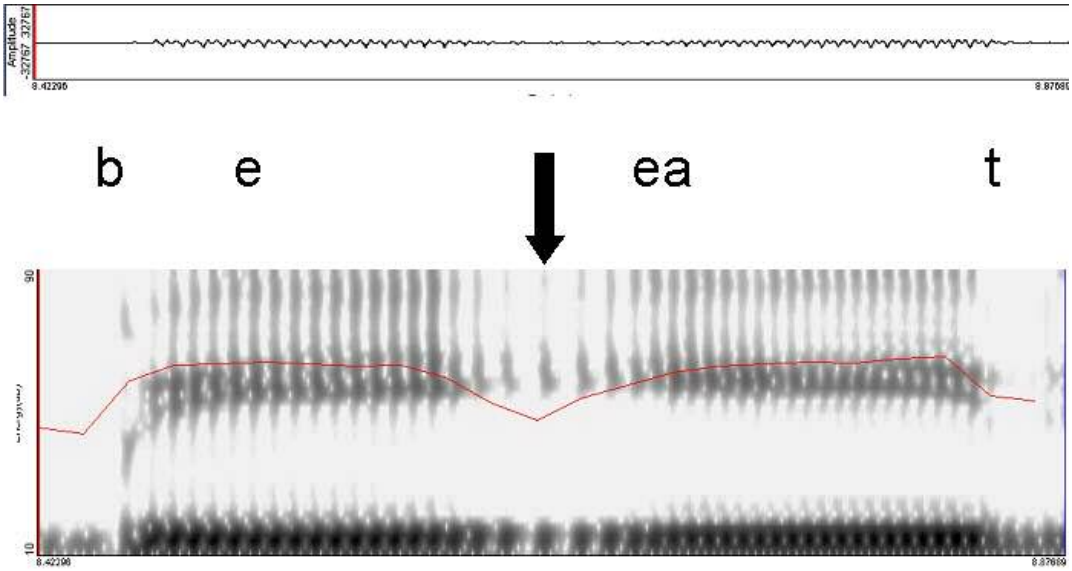
Code 1: Oral constriction gesture associated with continuous phonation without aspiration and with change in high frequency spectral energy and/or energy level. The voice bar is continuous across the word boundary. As illustrated in Figure 12 there is a slight amplitude drop in the superimposed energy contour. Oral constriction gestures may be used in lieu of both aspiration and laryngealization gestures.

Figure 12. Speaker using a oral constriction in lieu of a laryngealization gesture to mark the word boundary in the phrase "be eating" characterized by a continuous voice bar and a slight decrease in high frequency spectral energy.



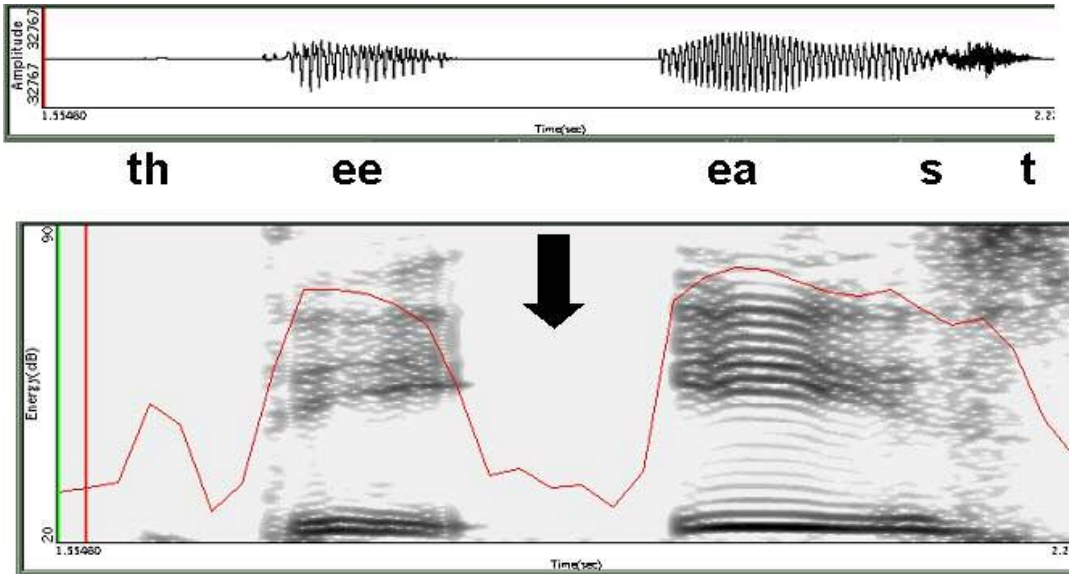
Code 2: Laryngealization gesture characterized by glottal fry or pulse mode without evidence of spectral noise, illustrated in Figure 13.

Figure 13. Speaker using a laryngealization gesture characterized by glottal fry without spirantization to mark word boundary in the phrase "be eating."



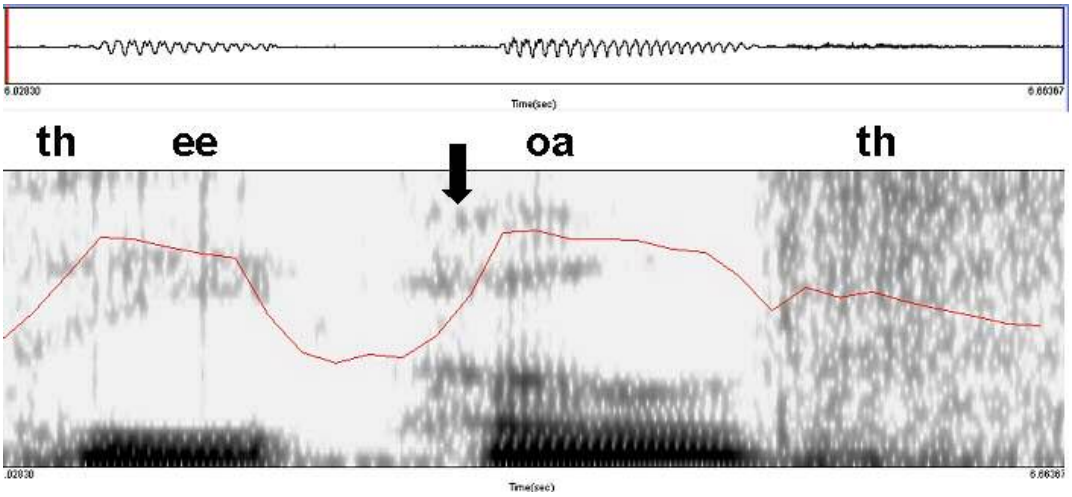
Code 3: Laryngealization gesture characterized by a complete glottal stop, defined as a clear gap without noise spectra that is between 25 to 200 ms. Gaps less than 25 ms were categorized as glottal fry (Hollien, 1974) and gaps greater than 200 ms were categorized as a pause and coded as missing data. Previously, Umeda (1989) defined glottal stops as no greater than 100 ms; there are no other published studies of glottal stop duration in American English. In the present investigation, gaps of up to 200 ms were clearly perceived as glottal stops (as opposed to pauses) by the author. This 200 ms criterion was also used by Gartner-Schmidt (2003). Figure 14 illustrates a speaker using a complete glottal stop to mark the word boundary in the phrase “thee east.” The spectral characteristics are suggestive of a laryngeal articulatory gesture involving tight closure, as indicated by the clear gap marked by the arrow.

Figure 14. Speaker using a complete glottal stop to mark the word boundary in the phrase "thee east."



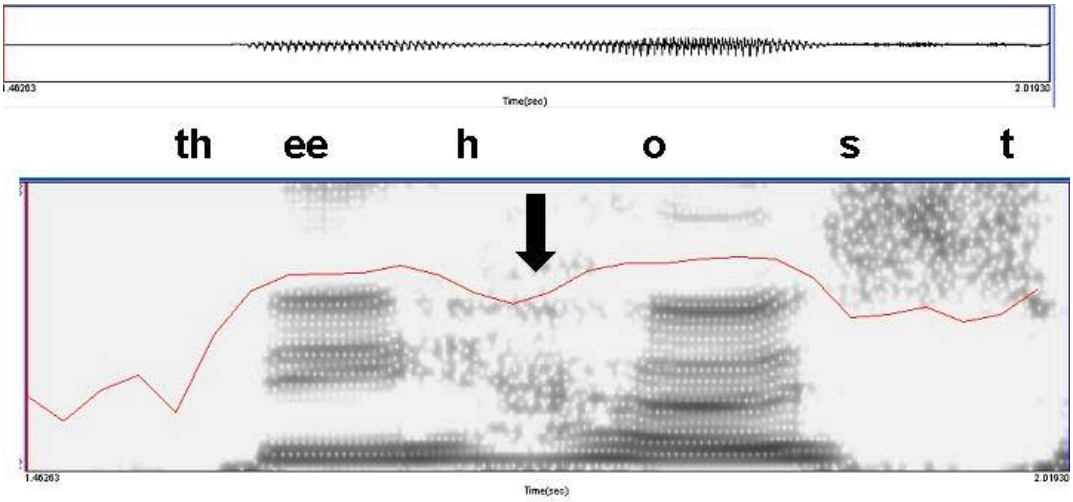
Code 4: Laryngealization gesture characterized by either a complete glottal stop or glottal fry associated with spirantization (Weismer, 1984b), characterized by evident noise spectra during the phonatory offset-onset gesture. As illustrated in Figure 15, spirantization is associated with incomplete glottal closure.

Figure 15. Speaker producing a glottal stop with spirantization to segment the word boundary in the phrase "thee oath."



Code 5. An aspiration gesture with frication associated with continuous voicing bar, indicating that the vocal folds are in vibration throughout the duration of the phonatory offset-onset gesture. However, there is clear evidence of noise spectra and loss of high frequency energy, suggesting glottal opening throughout the gesture, as illustrated in Figure 16.

Figure 16. Speaker using an aspiration gesture with continuous vocal fold vibration appropriate for a voiced glottal fricative to in the phrase "thee host."



Code 6. Aspiration gesture associated with complete cessation of the voice bar and complete loss of vowel spectral energy, replaced by noise appropriate for a voiceless glottal fricative (as illustrated in Figure 17) and a voiceless alveolar fricative (as illustrated in Figure 18).

Figure 17. Speaker producing a complete aspiration gesture for /h/ in the phrase "thee heap."

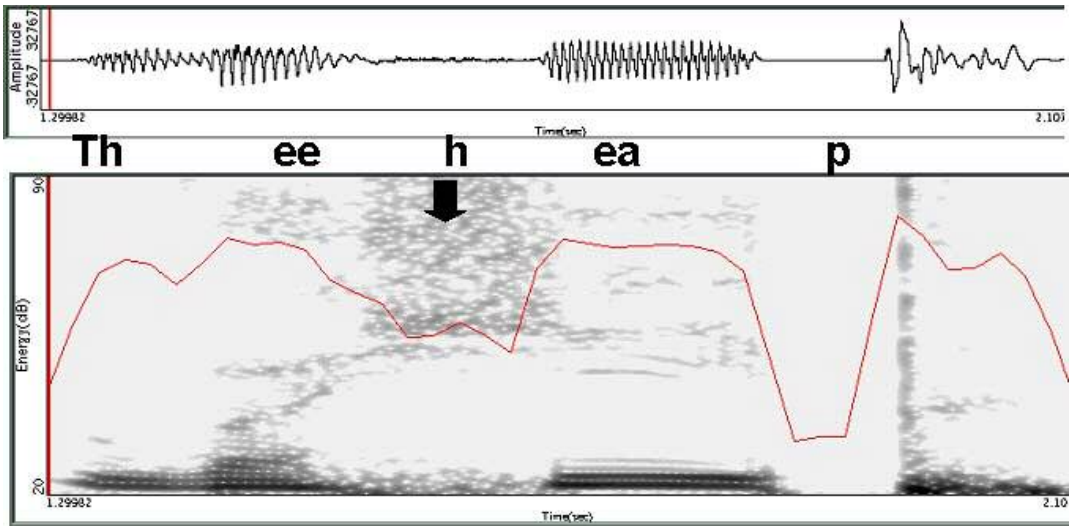
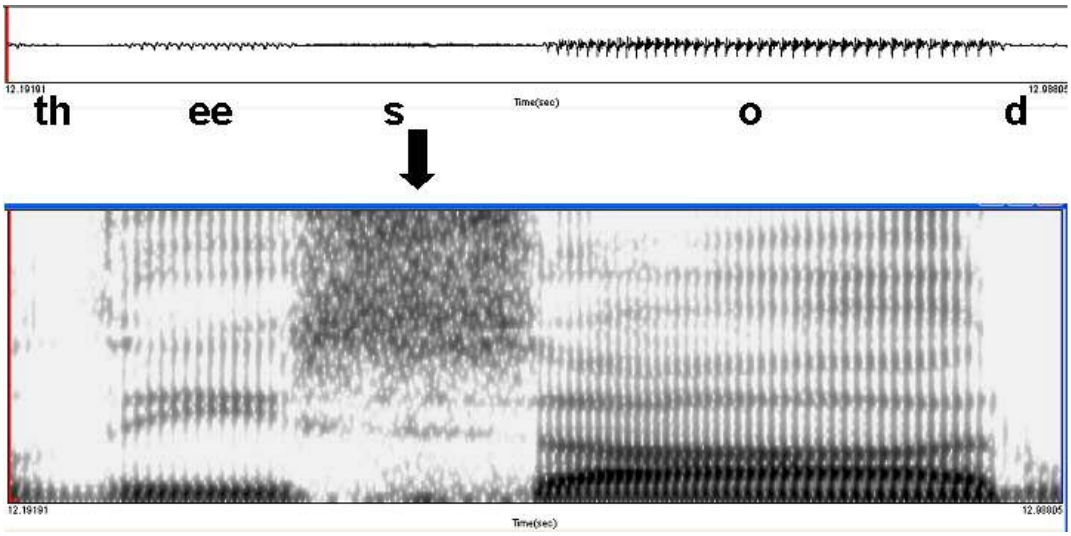


Figure 18. Speaker producing a complete aspiration gesture for /s/ in the phrase "thee sod."



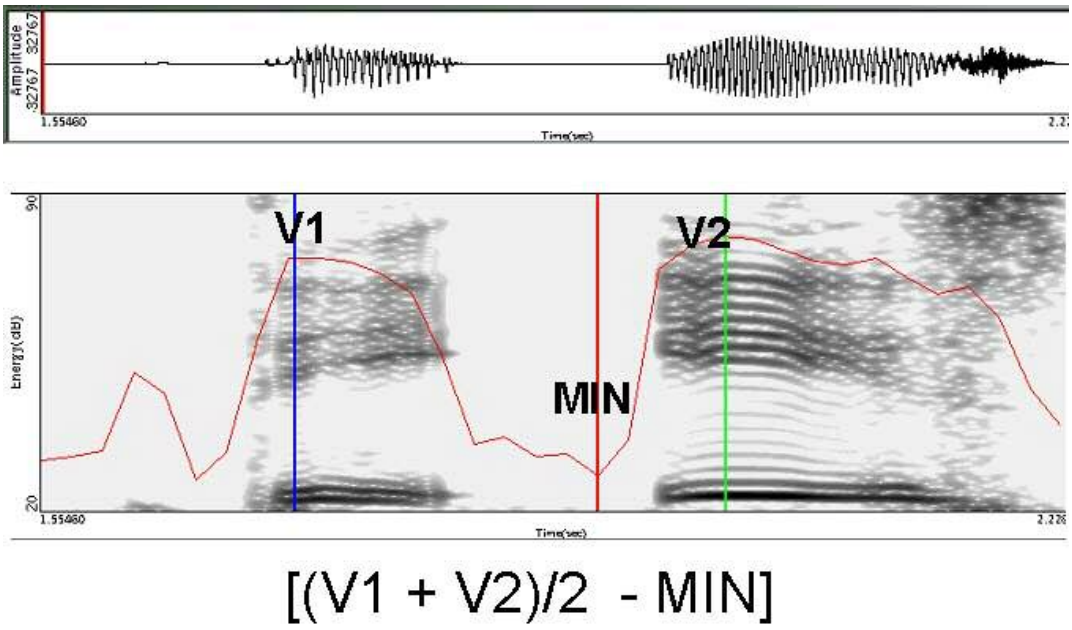
Descriptive Codes for Multiple Gestures.

A second nominal coding system was developed for the S_V context, which was intended to elicit multiple gestures of phonatory offset-onset (e.g. both the aspiration and laryngealization gesture). Spectrograms were displayed (using the parameters identical to the previous spectrographic coding system) and judgments of spectrographic pattern codes were made. Each token was coded a "1" to indicate that only the aspiration gesture was produced or "2" to indicate that both an aspiration and a laryngealization gesture were produced. Only spectrographic pattern codes of 2, 3, or 4 were considered to be a laryngealization gesture (as illustrated in Figures 13, 14, 15). Only the first utterance phrase was coded for multiple gestures to avoid utterance end effects (Fledge & Brown, 1982). Therefore, the S_V context had only 18 tokens (6 vowels x 3 speaking mode conditions) coded per speaker.

Measure of Vowel Intensity for Sentences.

A spectrogram, similar to that used for spectrographic pattern coding, was displayed. With the intensity contour (in dB) displayed, the peak amplitude for the first vowel (V1) and second vowel (V2) were measured (see Figure 19). These measures were completed for the first and second phonatory offset-onset phrase in each sentence. The four peak vowel amplitudes were then averaged to represent peak vowel amplitude (in dB) for each sentence. This measure of average peak vowel amplitude was determined by replication of six vowels x three phonetic contexts x 3 speaking modes x two speaker groups for a total of 5, 184 tokens.

Figure 19. Procedure used to measure peak vowel amplitudes (dB) and the minimal intervocalic segment (dB) to derive acoustic measures of vowel intensity and intervocalic spectral energy change.



Measure of Intervocalic Spectral Energy Change.

As illustrated in Figure 19, complete phonatory offset-onset associated with a glottal stop laryngealization gesture results in a significant change in energy level (i.e., MIN) in comparison with the peak vowel dB levels (V1 and V2). Thus a measure of intervocalic spectral energy change was developed to reflect a phonatory offset-onset mechanism that was quantified as an intervocalic spectral energy change. The change was calculated as:

½ (Peak V1 + V2) – minimum intervocalic value

where V1 represented the preceding vowel segment peak dB amplitude level and V2 represented the following vowel segment peak dB amplitude level. Higher intervocalic spectral energy change values indicated a greater energy change associated with phonatory offset-onset. Two intervocalic spectral energy change values were measured for the each of the V_V, V_H, and V_S phonetic contexts and averaged for each sentence and for each of the six vowels. Therefore, each speaking mode was associated with an averaged intervocalic spectral energy change of 12 values (2 per sentence x 6 vowel types) for each of the three phonetic contexts for each speaker.

Reliability.

Percentage of agreement was use to determine the reliability of coding measures for the (a) VAS task, (b) the sustained vowel (MDVP measures), (c) L-DDK tasks, (d) the phonatory offset-onset spectrographic patterns, and (e) the intervocalic spectral energy change measures (see Table 8). For each measure, the

unit-by-unit agreement ratio was applied to calculate agreement expressed in percentages:

$$\text{Unit-by-unit agreement index} = \frac{A}{A + D} \times 100$$

where A = number of agree tokens, D = number of disagree tokens (Young, 1975).

The Unit-by Unit Agreement Ratios (Young, 1975) for selected dependent measures are summarized in Table 8. Reliability for the VAS task was determined by randomly selecting and re-measuring 30% of the VAS forms. The percentage of agreement was 100%.

Table 8. Summary of Unit-by-Unit Agreement Ratios for VAS, MDVP, L-DDK, spectrographic pattern codes, and intervocalic spectral energy change measures.

Dependent Measure	Unit-by Unit Agreement Ratio
Visual Analogue Scale	100%
L-DDK Adductory Gesture	100%
L-DDK Abductory Gesture	100%
MDVP Measures	94%
Spectrographic Pattern Codes: • V_V Phonetic Context • V_H Phonetic Context • V_S Phonetic Context	 97% 99% 100%
*Peak Vowel Amplitude	98%
*Minimum Value of Intervocalic Segment	90%

* 2 dB agreement between the 2 measurements

For the L-DDK tasks all speakers were measured independently by two raters, resulting in 100% agreement for both the adductory and abductory tasks. With respect to the sustained vowel task, Kent et al. (1999, p. 129) have reported "very good" reliability for MDVP analyses between two captures of the same

vowel phonation in speakers with dysarthria. The present investigator recaptured one phonation for each participant and compared the radial graph between the first and second capture. Agreement was defined as a value falling within ±1 SD of the normative value for each measure as reported by the manufacturer (Kay-Elemetrics, 1999b). The unit-by unit agreement ratio (Young, 1975) was 94%.

Good inter-judge reliability for the spectrographic pattern codes reflecting mechanisms of phonatory offset-onset was reported in pilot analyses (Bassich, 2001). Percent of exact blind inter-judge agreement among 3 judges ranged from 92% for GNM and 97% for CBZ and JGS. Intra-judge reliability was assessed for the present investigation by randomly selecting 10% of the phonatory offset-onset tokens for recoding. The percentage of agreement (Young, 1975) ranged from 97% for the V_V phonetic context to 100% for the V_S phonetic context.

Finally, 15% of the data was randomly selected for a repeated measure of peak vowel amplitude and the minimum value of the intervocalic spectral energy. For these measures, a reasonable margin of error was determined and values that fell within the margin of error were scored as *agree.* For example, a pilot investigation for the present investigation (Bassich, 2001) showed that there was a 2 dB variation between two repeated measurements secondary to rounding errors and minor deviations in the software program associated with different window displays. Therefore, values within 2 dB were considered acceptable intra-judge agreement for all amplitude-dependent measures. Mean percentage of agreement for peak vowel amplitude was 98% and was 90% for spectral energy change. In

summary, Unit-by Unit Agreement Ratios for the dependent measures that required investigator measurement were 90% or better.

Statistical Analyses

All statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS V.11). Statistical analyses for research questions #1(i) and #1(ii), which addressed speaker group differences with respect to reported voice and speech symptoms (VAS) and the impact of vocal dysfunction on quality of life (VHI and SF-12), included independent sample *t* tests for nine VAS, three VHI, and two SF-12 dependent measures. Also, independent sample *t* tests were used to determine speaker group differences with respect to acoustic measures of phonatory offset-onset control for both the adductory and abductory L-DDK tasks (research question #2[i]) and the acoustic voice spectra measures related to frequency, intensity, and voice spectra (research question #2[ii]). Following the results of a Levene's test for equality of variances, either pooled variances or separate variance *t* tests were used, depending on whether or not the assumption of homogeneous variance was met. In instances where this assumption for parametric tests was not met, a *t* test using separate variance estimates (as reflected by an adjusted degrees of freedom) was used (Kirk, 1968).

For research questions #3(i) and #3(ii), mixed three-way ANOVA designs were used to determine between group speaker effect and within group effects of phonetic context (V_V, V_H, V_S) and speaking mode manipulation (habitual, confidential, and project) on acoustic measures of average peak vowel amplitude and intervocalic spectral energy change (Kirk, 1968). The follow-up exploration of

main effects involved a factor that was a repeated measure (three levels). The Fisher's Least Significant Difference (LSD) procedure was used (Fisher, 1966), which is appropriate provided there are no more than three levels in the repeated measures. Scheffe or Newman-Keuls tests were not appropriate for these data because the assumption of sphericity was not met for all contrasts (Andrews, 1974). A Bonnferoni correction of $\alpha = 0.166$ would also be appropriate, but would have increased the probability of a Type II error because of the decreased power. In using the Fisher's LSD procedure the appropriate critical value for statistical significance was set at $\alpha = 0.05$. If the outcome of the Mauchly's test of sphericity was significant, indicating that the parametric assumption of sphericity was not met, the degrees of freedom associated with each F-test was adjusted using the Huynh-Feldt correction (Kirk, 1968).

For research question # 3(iii), bar graphs of descriptive codes for spectrographic patterns were used to describe mechanisms of phonatory offset-onset control in YOPD and HC speakers for each of the three phonetic contexts across three speaking mode conditions. Bar graphs of descriptive codes were used to determine the occurrences of phonatory offset-onset mechanisms associated with (a) laryngealization gestures, (b) aspiration gestures produced without vocal tract constriction, and (c) aspiration gestures produced with oral constriction. The bar graphs were designed to compare descriptive codes between speaker groups (YOPD and HC Speakers) within each phonetic context, and separate figures were designed for each of the three speaking modes. Statistical analyses were not appropriate, as the data consisted of nominal values.

For research question #3 (iv) bar graphs were used to display the percentage of single and double gestures produced for the S_V context comparing each speaker group for each speaking mode condition. Again statistical analyses were not appropriate, as the data consisted of nominal values.

Results

The results are organized into three sections. The first section summarizes the findings for Objective 1. Speaker group differences were identified with respect to the speech and voice symptoms reported by YOPD and HC participants. Also, group differences were investigated with respect to participants' perception of the negative impact of these symptoms on their quality of life. Results of speaker group comparisons are described for the: (a) Visual Analogue Scale (VAS) (Ramig, Pawlas, & Countryman, 1995); (b) the Voice Handicap Index (VHI) (Jacobson et al., 1997); and (c) the 12-Item Short Form Health Survey (SF-12) (Ware, Kosinski, & Keller, 1995).

The second section summarizes the findings for Objective 2. Speaker group differences were examined for the acoustic measures of vocal function on clinical tasks (e.g., L-DDK and sustained vowel phonation). Results of speaker group comparisons are described for the dependent measures of the two laryngeal diadochokinesis tasks and the eleven acoustic voice spectra measures (related to frequency, intensity, and voice spectra) that were calculated from the sustained vowel phonation stimuli using the Multidimensional Voice Profile™ software package (Kay-Elemetrics, 1999b).

The third section summarizes the effect of speaker group, phonetic context, and speaking mode on acoustic measures of mechanisms of phonatory offset-onset control. First, the results of a mixed 2 x (3) x (3) three-way ANOVA used to test the statistical significance of differences among the measure of peak vowel amplitude (dB) are summarized. Then, the results of a mixed 2 x (3) x (3) three-

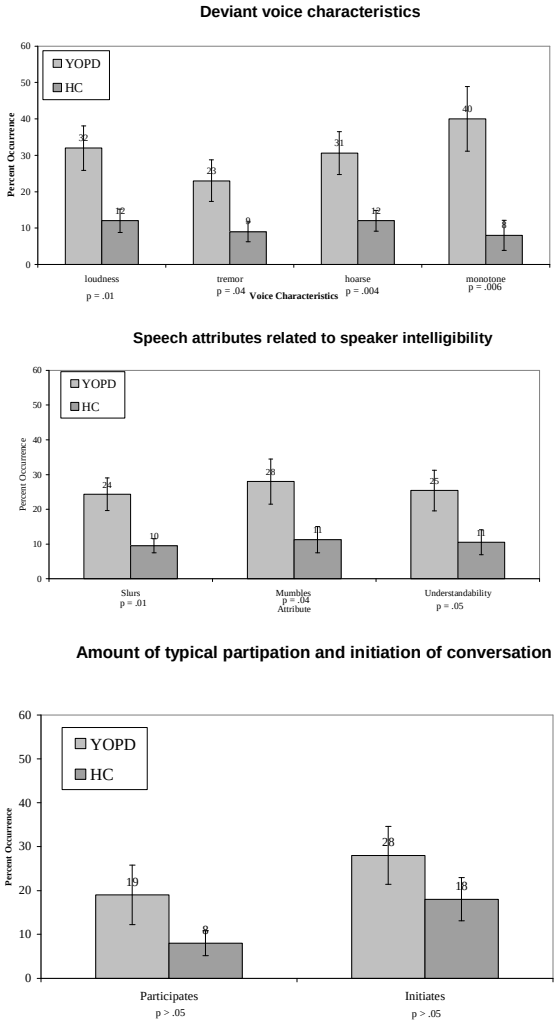
way ANOVA used to test the statistical significance of differences among the measures of phonatory offset-onset control as reflected in the intervocalic spectral energy change (dB) are summarized. Also, intra-group speaker differences in phonatory offset-onset control in varying phonetic contexts and speaking modes are examined. Finally, two descriptive analyses are presented that summarize the nominal coding systems of spectrographic patterns reflecting phonatory offset-onset. These analyses include (a) spectrographic patterns reflecting phonatory offset-onset mechanisms associated with laryngealization and aspiration gestures, and (b) a coding of the speaker production of single versus double gestures for the S_V context.

Perceived voice and speech symptoms and impact on QOL

Group data for the Visual Analog Scale (VAS) (Ramig et al., 1995) are presented in Appendix I [Table I(1)] and are illustrated in Figure 20. To evaluate group differences, independent *t* tests were run for the nine vocal attributes. As expected, significant group differences were found for the four voice characteristics of (a) loudness, mean difference (MD) = 20%, $t(16.6) = 2.88$, $p = .01$; (b) tremor, MD = 14% (shaky voice) $t(15.7) = 2.21$, $p = .04$; (c) hoarseness (scratchy voice), MD = 19%, $t(21) = 3.25$, $p = .004$; and (d) monotone, MD = 31%, $t(15.6) = 3.19$, $p = .006$. The YOPD speakers reported a significantly higher percent of occurrence for all four voice characteristics. Also, significant group differences were detected for the three speech attributes related to speaker intelligibility, with a mean group difference of (a) 14% for the attribute of slurs $t(15.1) = 2.92$, $p = .01$; (b) 17% for mumbles $t(17.6) = 2.23$, $p = .04$; and (c) 15%

for understandability $t(17.9) = 2.14$, $p = .05$. As predicted, the YOPD speakers reported a significantly higher percent of occurrence of these attributes in comparison with the HC speakers. However, there were no significant group differences for the pragmatic attributes of the percent occurrence of participating and initiating conversation. Therefore, YOPD persons perceived a greater degree of voice symptoms and reduced speech intelligibility, but these symptoms did not appear to impact functionally with respect to initiating or engaging in social conversations.

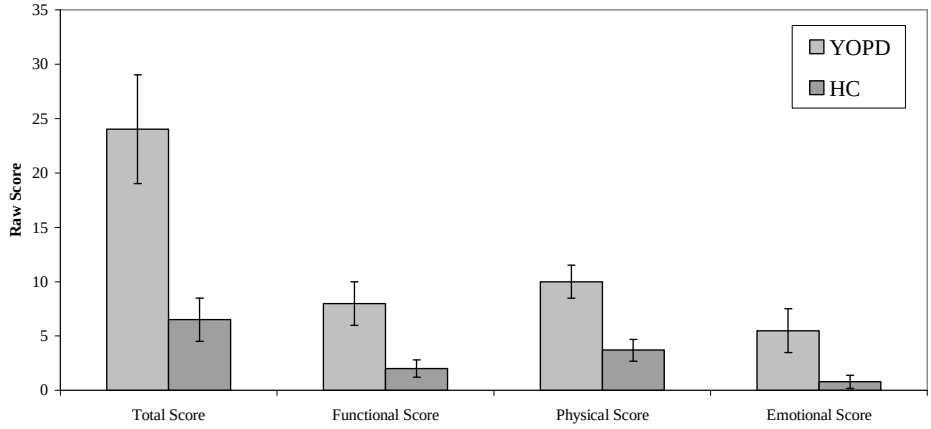
Figure 20. Mean and 95% confidence intervals for the reported deviant voice symptoms, speech intelligibility, and typical participation and initiation of conversation reported by YOPD and HC Speakers using a visual analog scale.



Group data for the Voice Handicap Index (VHI) (Jacobson et al., 1997) are presented in Appendix I [Table I (2)] and illustrated in Figure 21. Independent two-tailed *t* tests were run to test for group differences. The mean Total VHI Score for the YOPD speakers was 23.9 (SD = 17.4), and the mean for the HC speakers was 6.6 (SD = 7.2). This difference was statistically significant

(t [14.7] = 3.19, p = .006). Furthermore, the mean group differences were significant for the three components of (a) functional score, MD = 6.2, t(14.2) = 2.91, p = .011; (b) physical score, MD = 6.5, t(22) = 3.54, p = .002; and (c) emotional score, MD = 4.7, t(13.7) = 2.34, p = .04. In comparison with the HC speakers, YOPD speakers reported a significantly higher score for each component, indicating their voice symptoms were negatively impacting on their lives. Using the normative data provided by Jacobson et al. (1997), YOPD speakers reported a wide range in total VHI score as raw scores varied from 0 to 54. According to Jacobson et al. (1997) persons with (a) a mild voice disorder report a mean score of 33.69 (SD = 5.60); (b) persons with a moderate voice disorder report a mean score of 44.37 (SD = 3.88); and (c) persons with a severe voice disorder report a mean score of 61.39 (SD = 4.21).

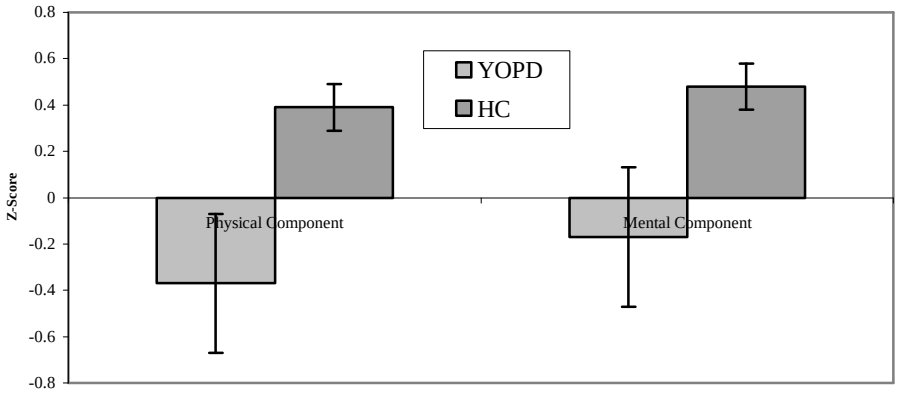
Figure 21. Mean and 95% confidence intervals for the Voice Handicap Index Scores for YOPD and HC Speakers.



Group data for the SF-12 (Ware et al.) are presented in Appendix I [Table I (3)] and illustrated in Figure 22. The SF-12 Physical and Mental Component raw

scores were transformed to a normative *z* score, on which negative scores reflected values lower than the national average. Independent *t* tests were run comparing the YOPD and HC groups. As expected, YOPD speakers reported a lower mean Physical Component *z* score of -0.37 (SD = 1.0) in comparison to the HC speakers (M = 0.39; SD = 0.48). The group difference was significant ($t(15.7) = -2.39$, $p = .03$). Although the YOPD mean score was also lower for the Mental Component *z* score, group differences were not statistically significant ($p > .05$). Therefore, the YOPD speakers reported a significantly lower overall physical health score, but the mental health score did not differ statistically from that of the HC speakers.

Figure 22. Mean and 95% confidence intervals for the Physical and Mental Health *z* scores on the SF-12 Health Survey for YOPD and HC speaker groups.



Speaker group differences for acoustic measures of vocal function on clinical tasks

Two routine clinical tasks (sustained phonation and L-DDK) were used to determine speaker group differences indicative of vocal dysfunction in YOPD.

Independent *t* tests were used to compare group differences for ten MDVP voice spectra measures of sustained phonation and six acoustic measures of L-DDK.

Voice Spectra Measures

Voice spectra measures obtained from a 3-sec segment of sustained vowel phonation for two speaker groups included ten MDVP measures as follows: (a) two fundamental frequency measures (mean F_o,[Hz} & F_o variability[vF_o] [Hz]); (b) two intensity measures (mean intensity [dB] & intensity variability [Hz]); (c) two frequency perturbation measures (smoothed pitch perturbation quotient [%SPPQ] & jitter [%]); (d) two intensity perturbation measures (smoothed amplitude perturbation quotient [%] and shimmer [%]); (e) soft phonation index [SPI]; and (f) number of unvoiced segments. The measures are summarized in Figures 23, 24, and 25 and Appendix I [Table I (4)] for the two speaker groups. None of the ten group comparisons were significant, indicating that the performance on sustained vowels by YOPD speakers appeared to be acoustically similar to HC speakers.

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Figure 23. Mean and 95% confidence intervals for the fundamental frequency Multidimensional Voice Profile (MDVP) measures for a 3-sec segment of sustained vowel phonation produced using a habitual voice mode by YOPD and HC Speaker Groups.

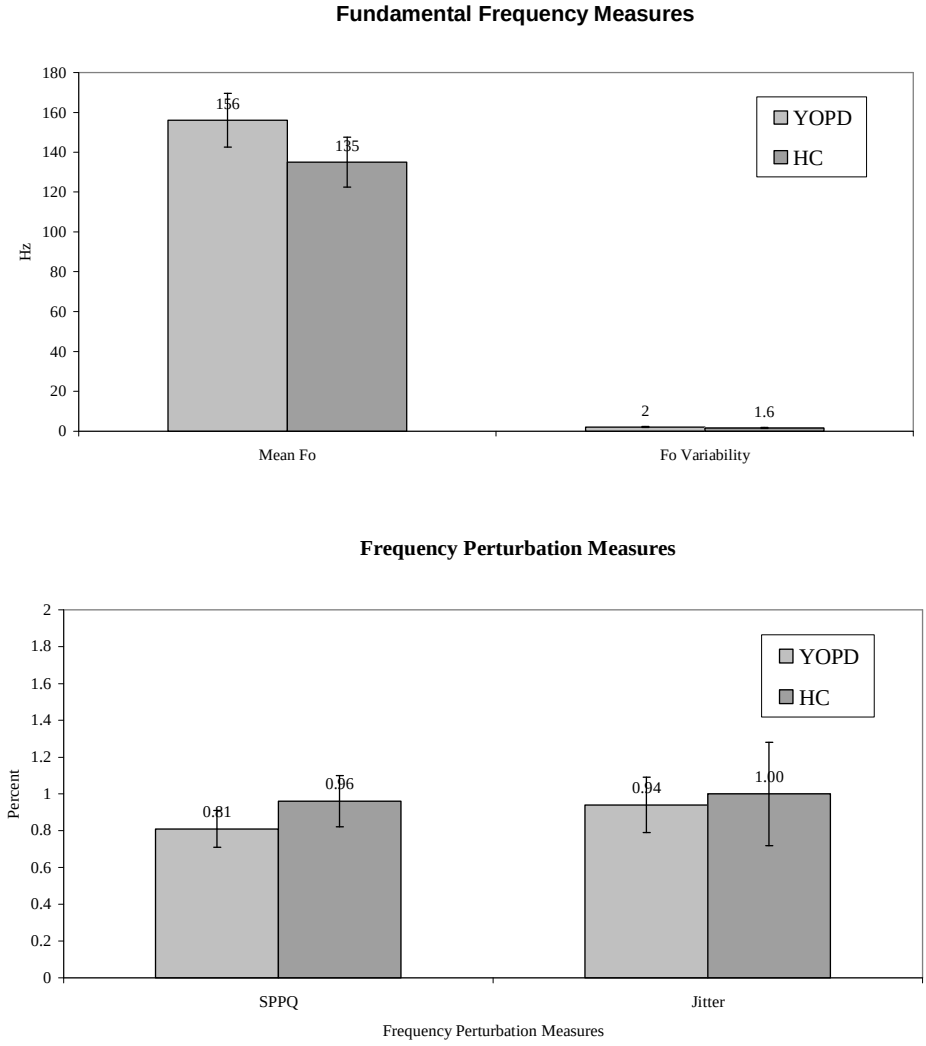

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Figure 24. Mean and 95% confidence intervals for the intensity Multidimensional Voice Profile Measures (MDVP) during a 3-sec segment of sustained vowel phonation produced using a habitual voice mode by YOPD and HC Speakers.

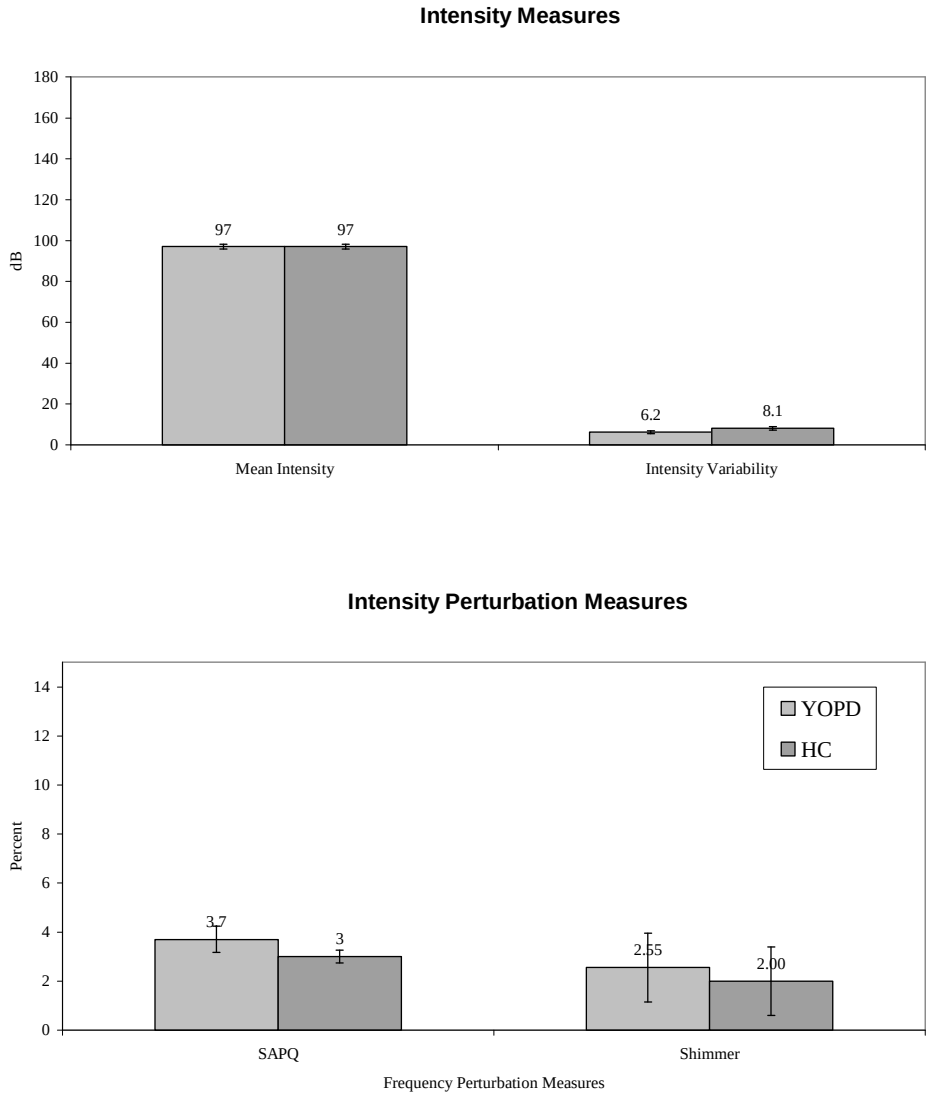
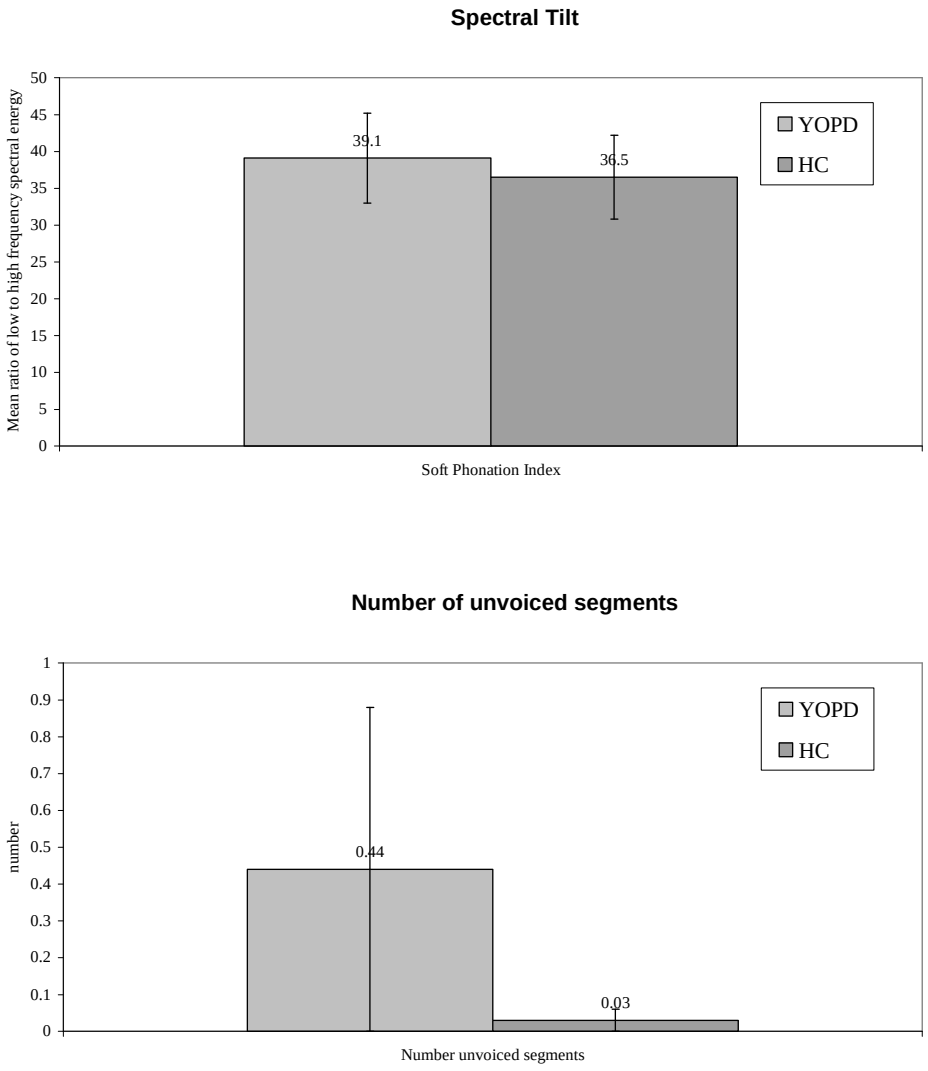


Figure 25. Mean and 95% confidence intervals for soft phonation index (spectral tilt) and number of unvoiced segments occurring during a 3-sec segment of sustained vowel phonation produced using a habitual voice mode for YOPD and HC speakers.

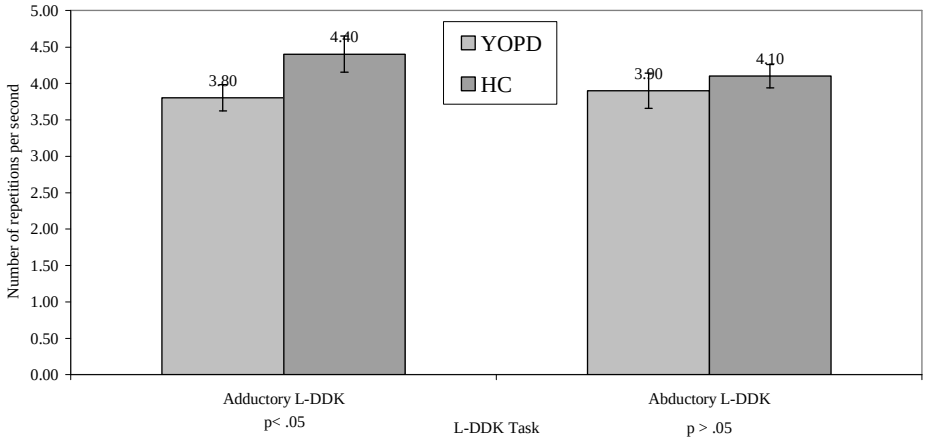


L-DDK Measures

Independent *t* tests were run to compare speaker group differences with respect to (a) the number of syllable repetitions per second, (b) the percentage of complete phonatory offset-onset (e.g., glottal stops), and (c) the number of pauses for the adductory "uh" and the abductory "huh" tasks, as illustrated in Figures 26,

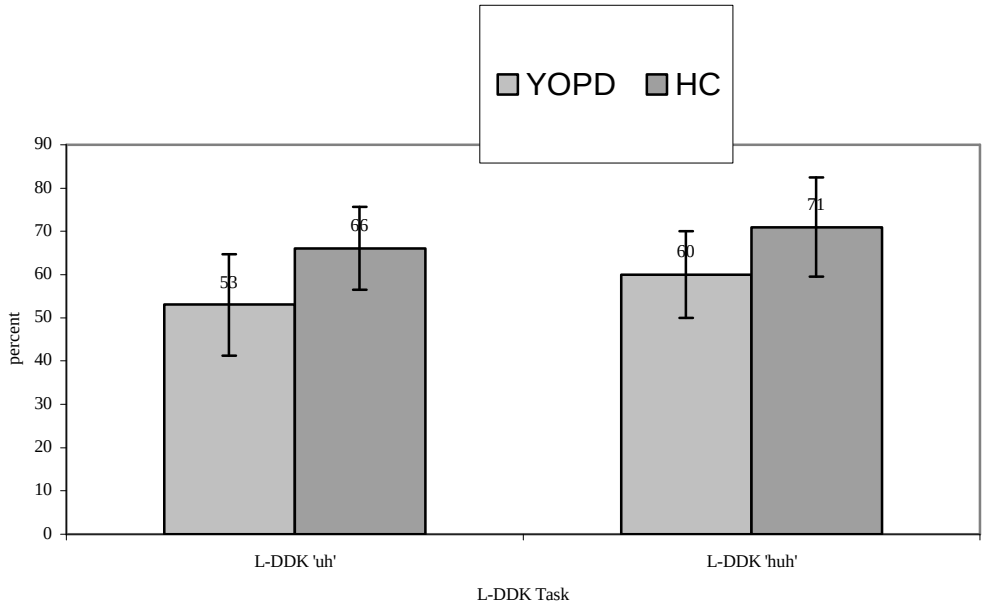
27, and 28, respectively. Means and standard deviations are summarized in the Appendix I [Table I (5)]. As illustrated in Figure 26, the group mean difference of 0.6 for the number of syllables per second for the adductory L-DDK gesture was significant ($t(22) = -2.0$, $p < .05$). However the mean group difference of 0.2 for the abductory L-DDK gesture was not significant ($p > .05$). Therefore, in comparison with HC speakers, the YOPD speakers had a significantly slower repetition rate for the adductory but not for the abductory L-DDK task.

Figure 26. Mean and 95% confidence intervals for the number of syllable repetitions per second produced by YOPD and HC speakers for the adductory and abductory L-DDK tasks.



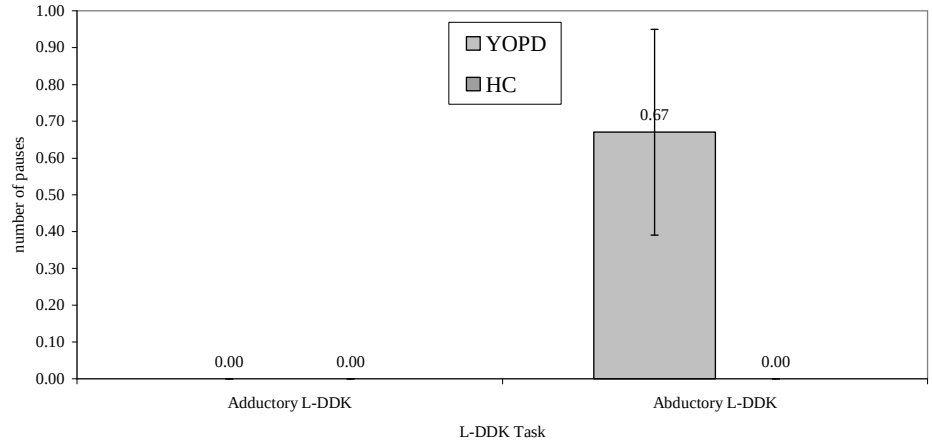
Group means and the 95% confidence interval for the percent of complete phonatory offset-onsets for the adductory and abductory L-DDK tasks are illustrated in Figure 27 and Appendix I [Table I (5)]. There were no significant group differences ($p > .05$) with respect to the percentage of complete phonatory offset-onsets for either the adductory or abductory L-DDK gestures.

Figure 27. Mean and 95% confidence intervals for the number of complete phonatory offset-onsets produced by YOPD and HC speakers for the adductory and abductory L-DDK tasks.



With respect to the number of pauses during the adductory and abductory L-DDK tasks, neither speaker group produced pauses during the adductory L-DDK task. For the abductory task, only the YOPD speaker group produced breath pauses, and the mean difference of 0.67 was significant ($t[11] = 2.35$, $p = .04$). (See Figure 28 and Appendix I Table I (5).

Figure 28. Mean and 95% confidence intervals for the number of pauses in a 7-sec segment produced by YOPD and HC Speakers for the adductory and abductory L-DDK tasks.



In summary, performance on two measures of adductory and abductory L-DDK was significantly impaired in the YOPD group in comparison with the HC group. For the adductory L-DDK measures, there was a significantly decreased rate of syllable repetition. For the abductory L-DDK measure, the YOPD speakers used a greater number of pauses to maintain syllable repetitions for a 7-sec segment. However, YOPD speakers did not demonstrate a fewer number of complete phonatory offset-onsets as compared to the HC speakers for both L-DDK tasks, nor did they demonstrate a slower syllable repetition rate for the abductory L-DDK task. Finally, there were no significant speaker group differences with respect to the number of pauses produced during the adductory L-DDK task.

Speaker group differences in phonatory offset-onset control in varying phonetic contexts and speaking modes

Mixed 2 x (3) x (3) three-way analyses of variance were used to test the statistical significance of differences for the measure of speech intensity (peak vowel amplitude [dB]) and intervocalic spectral energy change across speaker groups (YOPD and HC speakers), phonetic contexts (V_V, V_H, and V_S) and speaking modes (habitual, confidential, and projected).

Intensity Measure

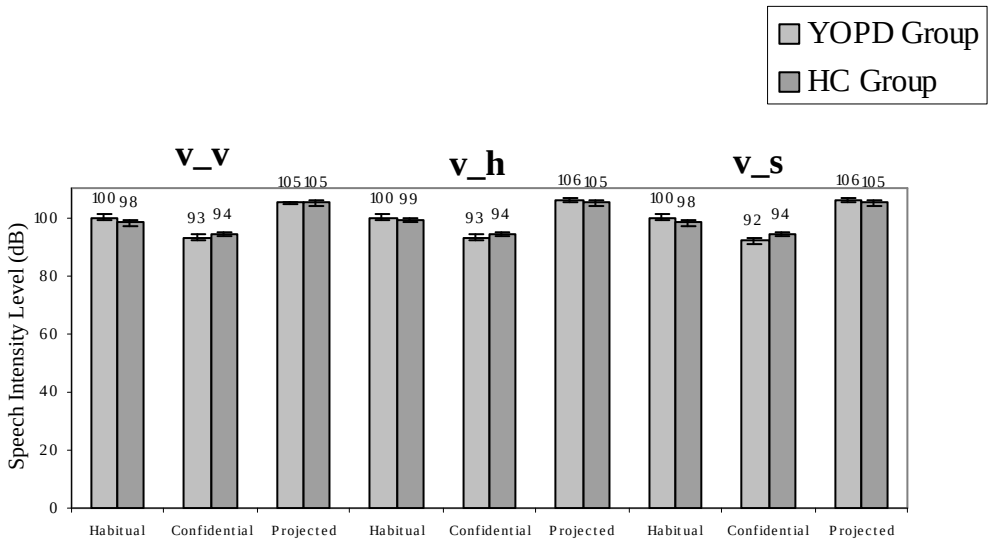
Results of a three-way analyses of variance for peak vowel amplitude are summarized in Table 9, while the mean and 95% confidence interval for the average intensity level for vocalic segments for three phonetic contexts across three speaking modes for YOPD and HC speaker groups are presented in Figure 29. Group means and SD are summarized in Appendix I Table I (7).

Table 9. Mixed 2 x (3) x (3) three-way ANOVA for peak vowel amplitude for speaker groups at three different phonetic contexts and three speaking mode conditions.

Source	*SS*	*df*	*MS*	*F*	*p*
Group	8.56	1	8.56	0.16	> .05
Phonetic Context	12.7	2	6.3	15.1	< .001
Speaking Mode	5139.5	2	2569.7	121.3	< .001
Group x Context	.9	2	.45	.4	> .05
Group x Mode	61.4	2	30.7	1.5	> .05
Context x Mode	.24	4	.06	.2	> .05
Group x Context x Mode	2.5	4	.6	1.8	> .05

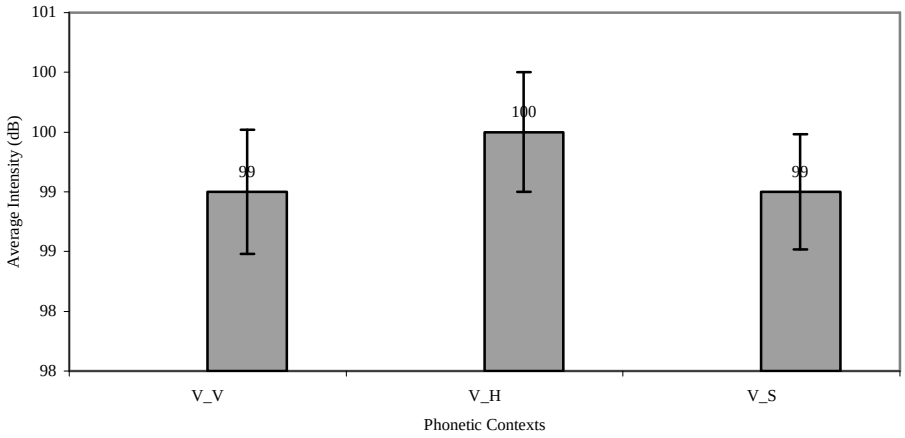
As shown in Table 9, significant phonetic context and speaking mode effects on peak vowel amplitude were observed. No significant group differences were found, and there were no significant interaction effects.

Figure 29. Mean and 95% confidence intervals for the average intensity level for vocalic segments for three phonetic contexts across three speaking modes for YOPD and HC Speaker Groups.



The significant phonetic context effect was probed by computing the average speaking mode score for each speaker within each phonetic context (see Figure 30). Speaker groups were combined because there was no significant between group difference in speech intensity. Paired *t* tests were then run to compare differences between the three phonetic contexts. The speakers' speech intensity level was significantly increased (by 1 db) in the V_H context in comparison with the V_V context $t(23) = 5.3$, $p < .001$ and V_S context$t$ (23) = 4.6, $p < .001$. However, there was no significant difference ($p > .05$) between speakers' speech intensity levels in the V_V and V_S phonetic contexts.

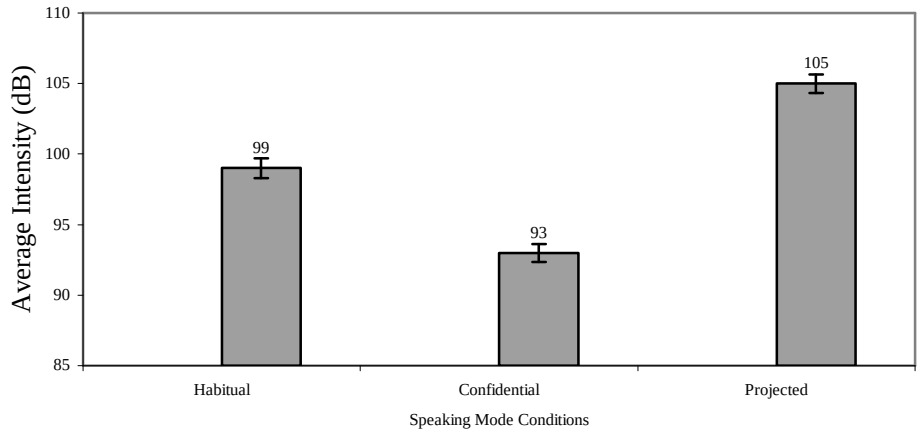
Figure 30. Mean intensity and 95% confidence intervals for both speaker groups averaged for three speaker modes within each phonetic context (Phonetic Context Effects).



The significant speaking mode effect was probed by computing the average phonetic context score for each speaker within each speaking mode (see Figure 31). Again, speaker groups were combined because there were no significant between group differences in speech intensity level. Paired *t* tests were run to compare differences between the three speaking modes. As predicted, the speakers' speech intensity level was significantly reduced in the confidential speaking mode in comparison with both the habitual mode $t(23) = 6.98$, $p < .001$ and the projected mode $t(23) = -14.6$, $p < .001$. Also, speakers' speech intensity level significantly increased in the projected mode as compared with the habitual mode $t(23) = -9.17$, $p < .001$. These findings provided experimental verification of the speaking mode tasks in that speakers demonstrated a 6 dB decrease in speech

intensity from habitual to confidential modes and a 6 dB increase in speech intensity from the habitual to projected modes.

Figure 31. Mean and 95% confidence intervals for the average vowel intensity level for both speaker groups combined and averaged for three phonetic contexts within each speaker mode (speaking mode effect).

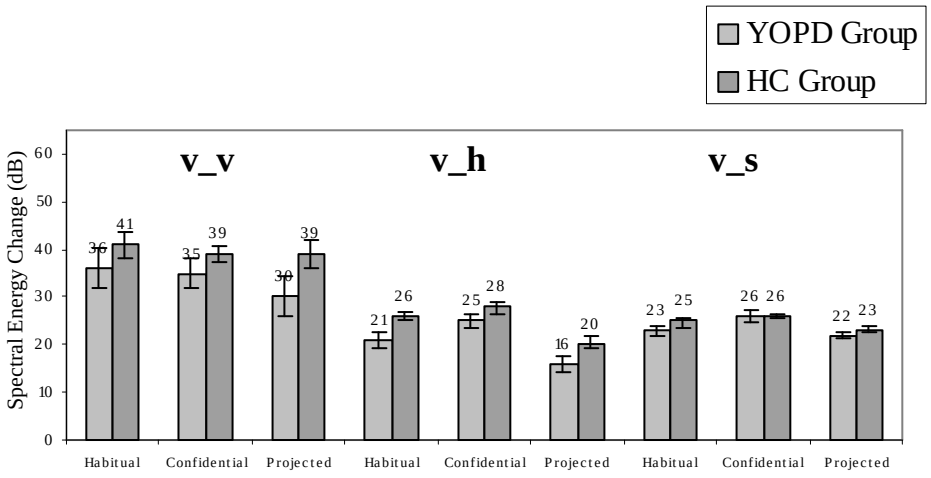


In summary, the research hypothesis that YOPD speakers would demonstrate decreased speech intensity levels in a habitual speaking mode, in comparison with the HC speakers, was not supported in this investigation, given that there was no significant between-group effects nor were there significant interaction effects. However, there was a significant main effect for phonetic context, reflecting a 1 dB greater intensity level observed for the V_H context in comparison with the other phonetic contexts. Finally, experimental manipulation was validated as both speaker groups combined demonstrated a 6 dB increase in speech intensity level from the habitual speaking mode to the projected mode. Speakers also demonstrated a 6 dB decrease in speech intensity level from the habitual speaking mode to the confidential mode.

Intervocalic Spectral Energy Change Measure

Figure 32 illustrates the mean and 95% confidence interval for the spectral energy change measure for three phonetic contexts across three speaking modes for YOPD and HC speaker groups. Means and standard deviations are also presented in Appendix I Table I (7).

Figure 32. Mean and 95% confidence intervals for the intervocalic spectral energy change measure for three phonetic contexts across three speaking modes for YOPD and HC Speaker Groups.



The results of a three-way analyses of variance are summarized in Table 10. Inspection of Table 10 reveals that there were no significant between-speaker group differences ($p > .05$). However, there were significant phonetic context and speaking mode effects on measures of intervocalic spectral energy change, although a significant phonetic context x speaking mode interaction effect was found as well.

Table 10. Mixed 2 x (3) x (3) three-way ANOVA for intervocalic spectral energy change for speaker groups at three speaking mode conditions and three phonetic contexts.

Source	*SS*	*df*	*MS*	*F*	*p*
Group	772.6	1	772.6	3.85	.06
Phonetic Context	8599.6	1.2	6.3	6894	< .001
Speaking Mode	957.2	2	478.6	32.2	< .001
Group x Context	222	2	111	1.0	> .05
Group x Mode	30.5	2	15.2	1.0	> .05
Context x Mode	267	3.12	85.6	6.7	<.001
Group x Context x Mode	63	4	15.8	1.6	> .05

The significant phonetic context x speaking mode interaction was probed by computing the average phonetic context for V_V, V_H, and V_S for each speaker for each speaking mode (Figure 33). Also, the average speaking mode score was computed for each speaker for each of the three phonetic contexts (Figure 34). Speaker groups were combined because there were no significant between group differences in intervocalic spectral energy change. The results of the 18 dependent *t* tests are summarized in Tables 11 and 12. Three paired *t* tests were computed to compare differences across three phonetic contexts for each speaking mode, yielding a total of nine post-hoc comparisons for the phonetic context effect. Also, three paired *t* tests (one for each speaking mode) were run to compare differences across three speaking modes for each phonetic context, yielding a total of nine post-hoc paired comparisons for the mode effect.

As illustrated in Table 11, a consistent phonetic context effect for the projected mode was found. However, phonetic context effects for the confidential and habitual modes were limited to the V_V/V_H and V_V/V_S comparisons; no phonetic context effect was observed for the V_H/V_S comparisons in the confidential and habitual modes.

Table 11. Summary of paired t-tests for phonetic context effect.

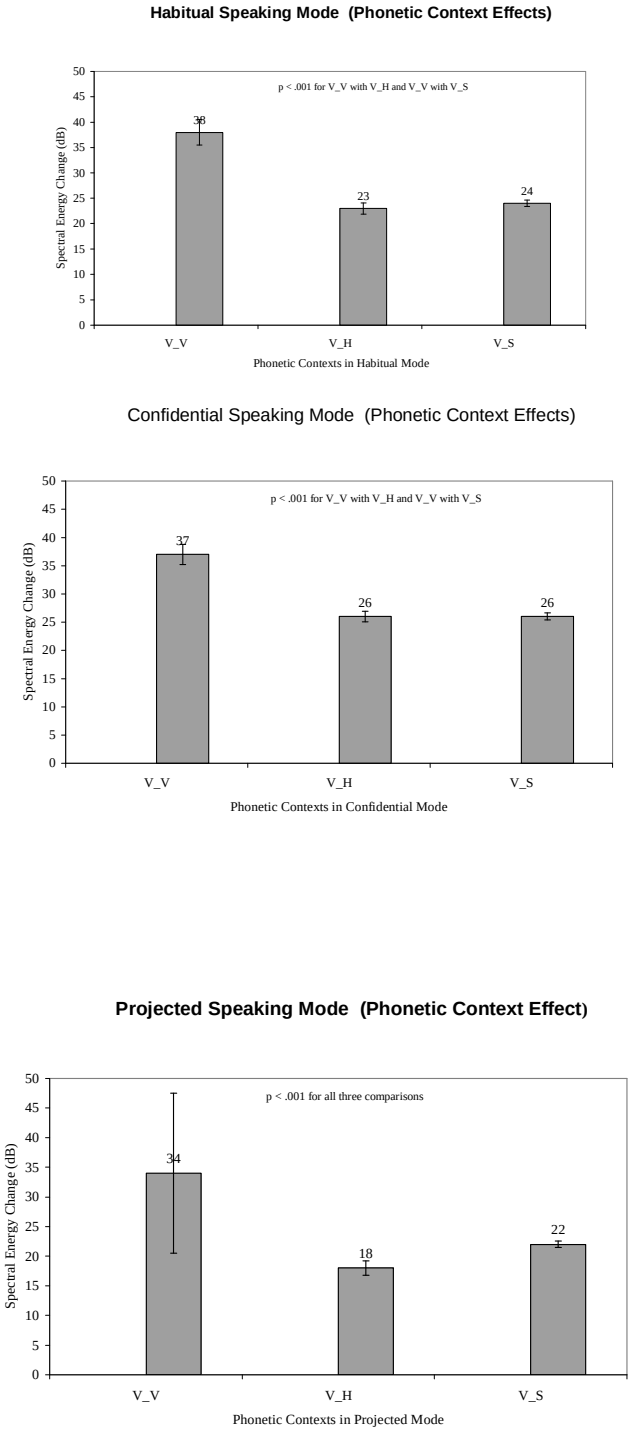
	Paired Comparisons		
Speech Mode	V_V/V_H	V_V/V_S	V_H/V_S
Habitual	$t(23) = 7.3$*	$t(23) = 5.5$*	$t(23) = -0.5$
	$p < .0001$	$p < .0001$	$p > .05$
Confidential	$t(23) = 7.9$*	$t(23) = 5.8$*	$t(23) = 0.3$
	$p < .0001$)	$p < .0001$)	$p > .05$
Projected	$t(23) = 7.3$*	$t(23) = 4.3$*	$t(23) = -3.9$*
	$p < .0001$	$p < .0001$	$p < .001$

*p < .05

As seen in Figure 33, for the projected mode there was a significantly greater change in the intervocalic spectral energy change observed in the V_V phonetic context in comparison with the V_H (t[23] = 7.3, $p < .0001$). Also, for this mode, there was a significantly greater intervocalic spectral energy change in the V_V phonetic context in comparison with the V_S context (t[23] = 4.3, $p < .0001$). Finally, there was a significantly greater change in intervocalic spectral energy for the V_S context in comparison with the V_H context (t[23] = -3.9, $p < .001$). Similarly, for the confidential speaking mode, there was a significantly greater change in the intervocalic spectral energy change observed in the V_V phonetic context in comparison with both the V_H (t[23] = 7.9, $p < .0001$) and

V_S (t[23] = 5.8, $p < .0001$) contexts. However, there was no significant difference in spectral energy change associated with the V_H versus V_S contexts in the confidential mode ($p > 05$). The same trend was observed for the habitual speaking mode. While there was a significantly greater intervocalic spectral energy change observed in the V_V in comparison with the V_H (t[23] = 7.3, $p < .0001$) and the V_S (t[23] = 5.5, $p < .0001$) phonetic contexts, there was no significant difference in spectral energy change associated with the V_H versus V_S contexts in the habitual mode ($p > .05$).

Figure 33. Mean and 95% confidence interval for the intervocalic spectral energy change for both speaker groups combined for each phonetic context at three speaking modes (phonetic context effect).



Inspection of Table 12 reveals a consistent effect across speaking mode for the V_H and V_S phonetic contexts. However the speaking mode effect for the V_V phonetic context was limited to the habitual/projected mode comparison. No speaking mode effect was observed for the habitual/confidential and the confidential/projected mode comparisons in the V_V phonetic context.

Table 12. Summary of paired t-tests for speaking mode effect.

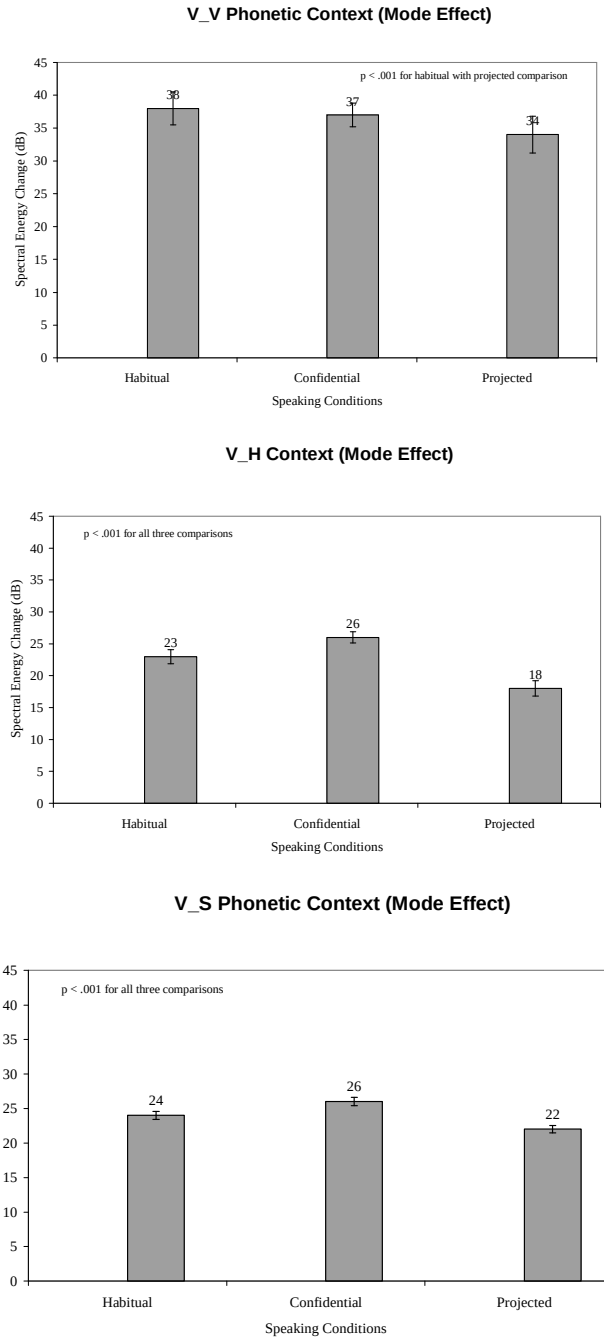
	Paired Comparisons		
Phonetic Context	Habitual/Confidential	Habitual/Projected	Confidential/Projected
V_V	$t(23) = 1.01$ $p > .05$	$t(23) = 2.8$* $p < .01$*	$t(23) = 1.7$ $p > .05$
V_H	$t(23) = -4.3$* $p < .0001$	$t(23) = 6.0$* $p < .0001$	$t(23) = 9.2$* $p < .0001$
V_S	$t(23) = -3.9$* $p < .001$	$t(23) = 3.8$* $p < .001$	$t(23) = 6.1$* $p < .0001$

*p < .05

As illustrated in Figure 34, for the V_H phonetic context, involving an aspiration gesture with an open vocal tract, the intervocalic spectral energy change was significantly greater for the confidential mode as compared to the habitual ($t[23] = -4.3$, $p < .0001$), while there was a significantly lower spectral energy change for the projected ($t[23] = 6.0$, $p < .0001$) mode in comparison with the habitual mode. Also, the intervocalic spectral energy change was significantly less for the projected speaking mode in comparison to the confidential mode ($t[23] = 9.2$, $p < .0001$. Similar results were noted for the V_S context, involving an aspiration gesture with oral constriction. Intervocalic spectral energy change was significantly greater for the confidential mode as compared to the habitual ($t[23] = -3.9$, $p < .001$) and was significantly less for the projected ($t[23] = 3.8$, $p < .001$)

as compared with the habitual mode. Also, the intervocalic spectral energy change was significantly less for the projected speaking mode in comparison with the confidential mode ($t[23] = 6.1$, $p < .0001$). For the V_V phonetic context, involving a laryngealization gesture, the intervocalic spectral energy change was significantly greater for the habitual speaking mode than the projected mode ($t[23] = 2.8$, $p < .01$). None of the other mode comparisons were significant ($p > .05$).

Figure 34. Mean and 95% confidence intervals for the intervocalic spectral energy change for both speaker groups combined for each speaking mode at three phonetic contexts (speaking mode effect).



Intra-group speaker differences.

Given the finding that speaker group differences with respect to intervocalic spectral energy change did not achieve but approached statistical significance (p = .06), intra-group speaker differences were examined. Figures were plotted whereby each of the 12 YOPD speakers' mean intervocalic spectral energy change values were compared against the mean and 95% confidence interval for the HC speaker group. These descriptive analyses provided information with respect to the intra-group speaker variability for the YOPD speakers in relation to the HC speakers. Data illustrated in Figures 35 to 37 reveal obvious speaker differences in the YOPD group that are most evident in the phonetic contexts involving an open vocal tract (V_V, V_H) in general and the laryngealization gesture (V_V) in particular.

Inspection of Figure 35 reveals that spectral energy change measures for five YOPD speakers were sufficiently below normal to be suggestive of impaired performance. In contrast, spectral energy change measures for six speakers in Figure 36 were near normal or indicative of mild impairment. Curiously, spectral energy change measures for one YOPD speaker in the V_V context were clearly above normal, as illustrated in Figure 37. It is interesting to note that this one speaker was one of two professional voice users included in the study. Although this speaker's control of phonatory offset-onset is clearly unimpaired, his Voice Handicap Score (see Figure 38) was indication of a moderate voice handicap. His voice characteristics and speaker intelligibility scores from the Visual Analog

Scales (see Figure 38) indicated a higher occurrence of hoarseness, monotone, slurring, mumbling, and being misunderstood.

Figure 35. Mean and 95% confidence intervals for the spectral energy change associated with the devoicing gesture for three phonetic contexts for five YOPD speakers who demonstrated impaired phonatory offset-on control for both the adductory and abductory gesture.

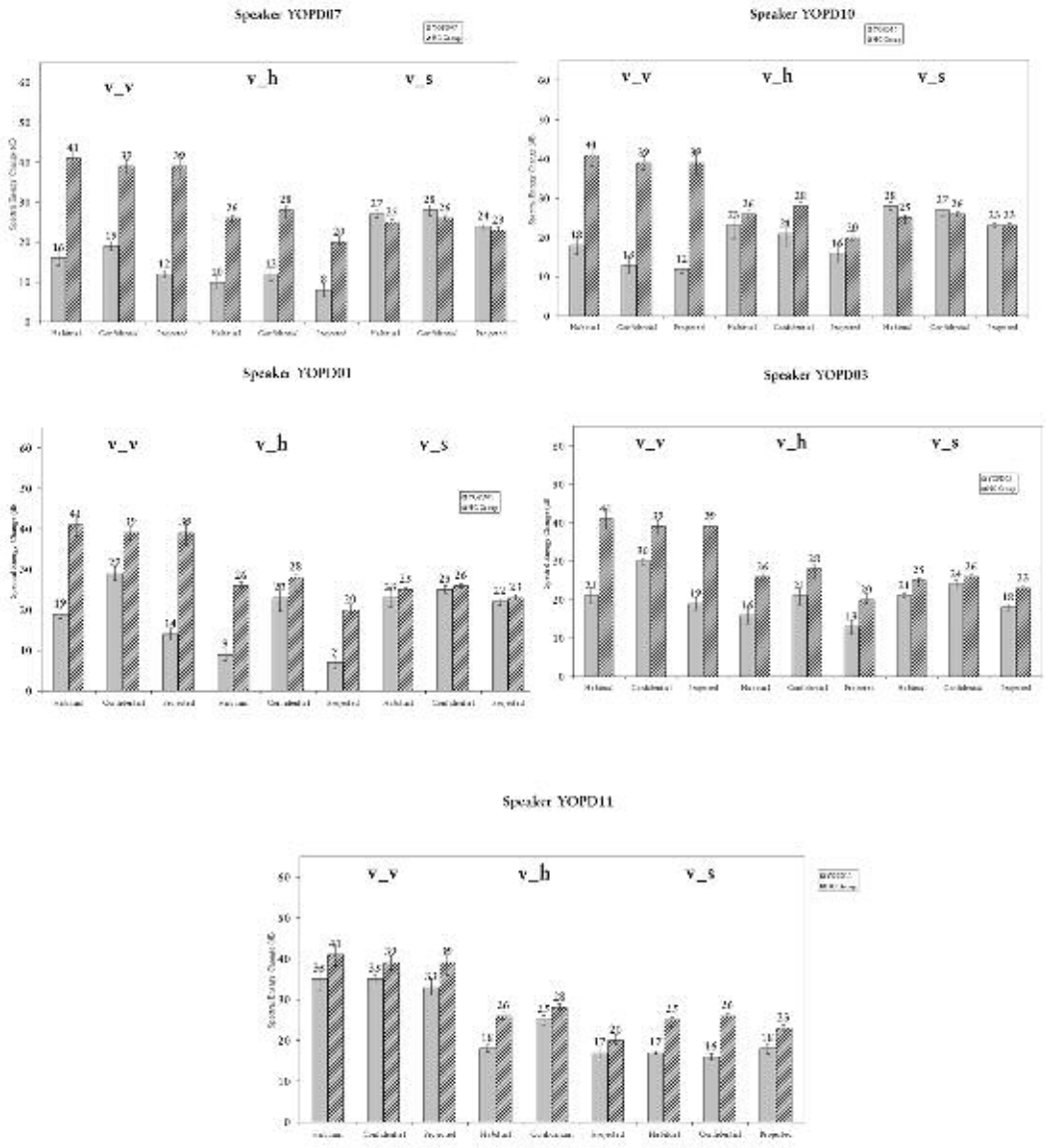


Figure 36. Mean and 95% confidence intervals for the spectral energy change associated with the devoicing gesture for three phonetic contexts for six YOPD speakers who demonstrated normal performance or mild impairment in phonatory offset-on control for a given phonetic context or speaking mode compared to the HC Speaker Group mean and 95% confidence interval.

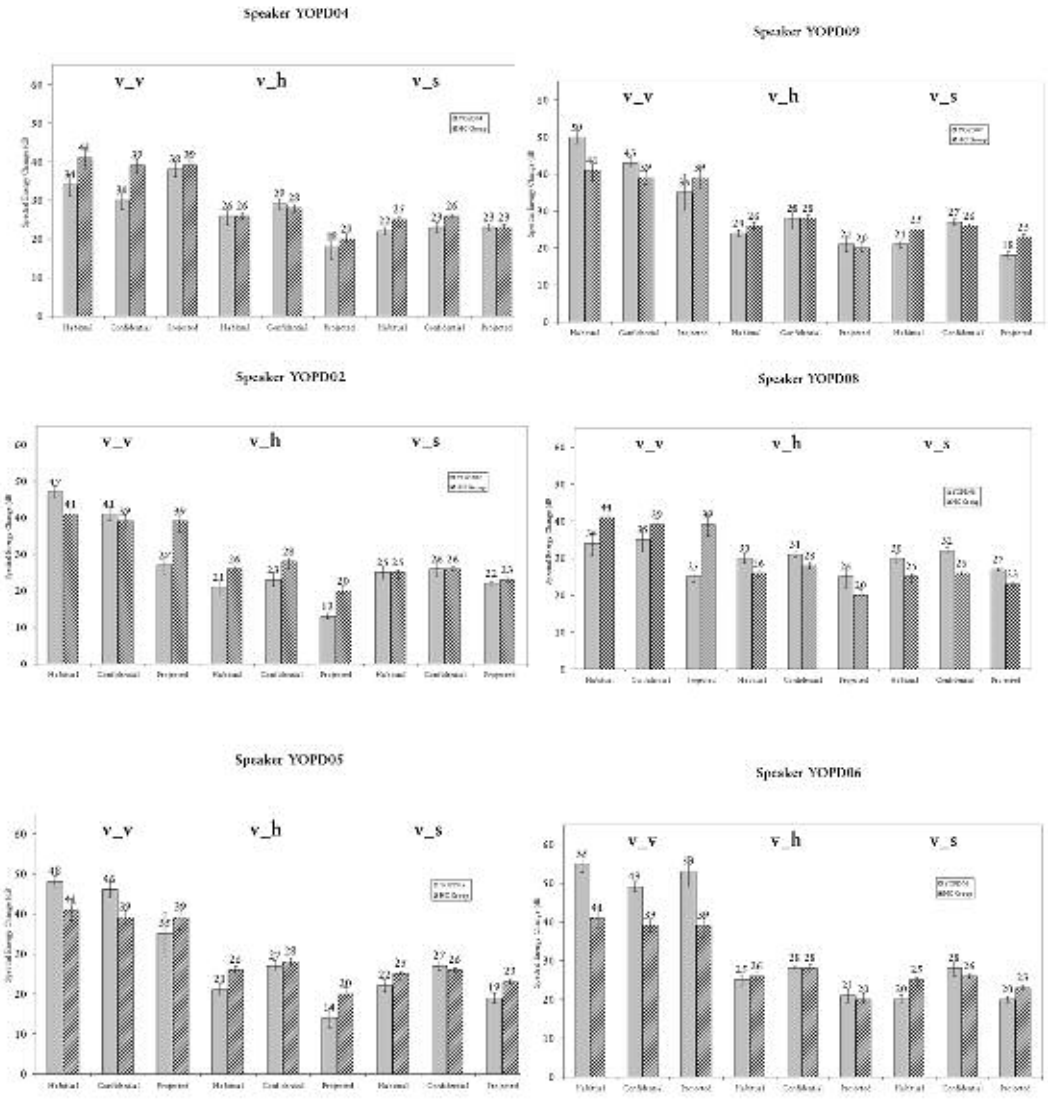


Figure 37. Mean and 95% confidence intervals for the spectral energy change associated with the devoicing gesture for three phonetic contexts for one YOPD speaker who demonstrated normal or superior phonatory offset-on control three phonetic contexts and three speaking modes compared to the HC Speaker Group mean 95% confidence interval.

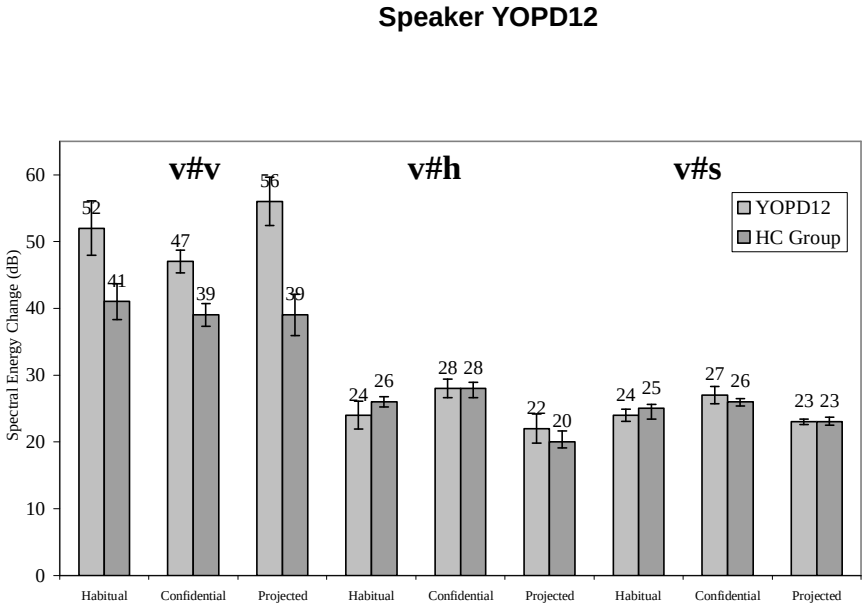


Figure 38. VHI scores for one YOPD speaker who demonstrated normal phonatory offset-onset control as compared to the mean and 95% confidence intervals for HC Speakers.

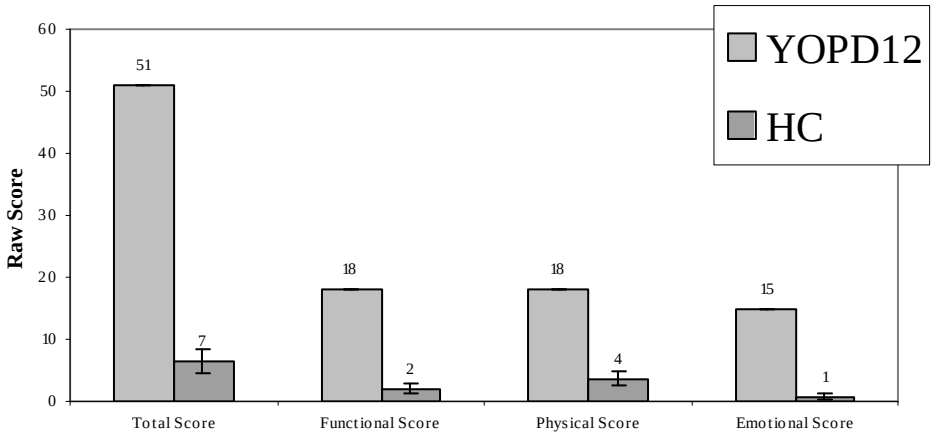
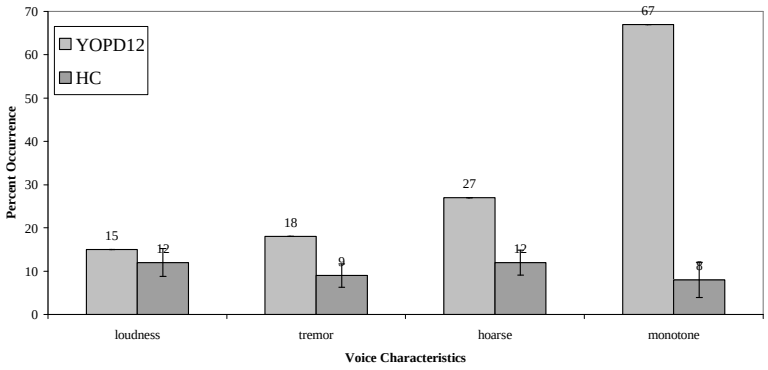
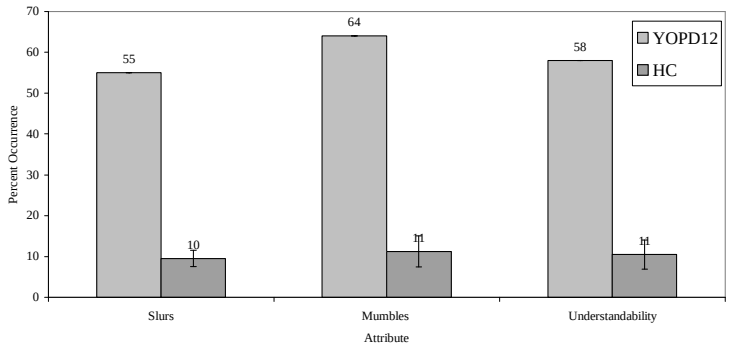


Figure 39. Voice and speaker intelligibility characteristics reported by one YOPD speaker who did not demonstrate impaired phonatory offset-onset control as compared to mean and 95% confidence interval reported by HC Speakers.





Descriptive analyses of spectrographic patterns

Descriptive analyses of spectrographic patterns coded for phonatory offset-onset mechanisms are presented in the following sections. These data are organized according to (a) phonatory offset-onset mechanisms produced without oral constriction (V_V and V_H phonetic contexts); (b) phonatory offset-onset mechanisms involving an oral constriction (V_S); and (c) phonatory offset-onset mechanisms involving multiple gestures (S_V).

Mechanisms involving an open vocal tract.

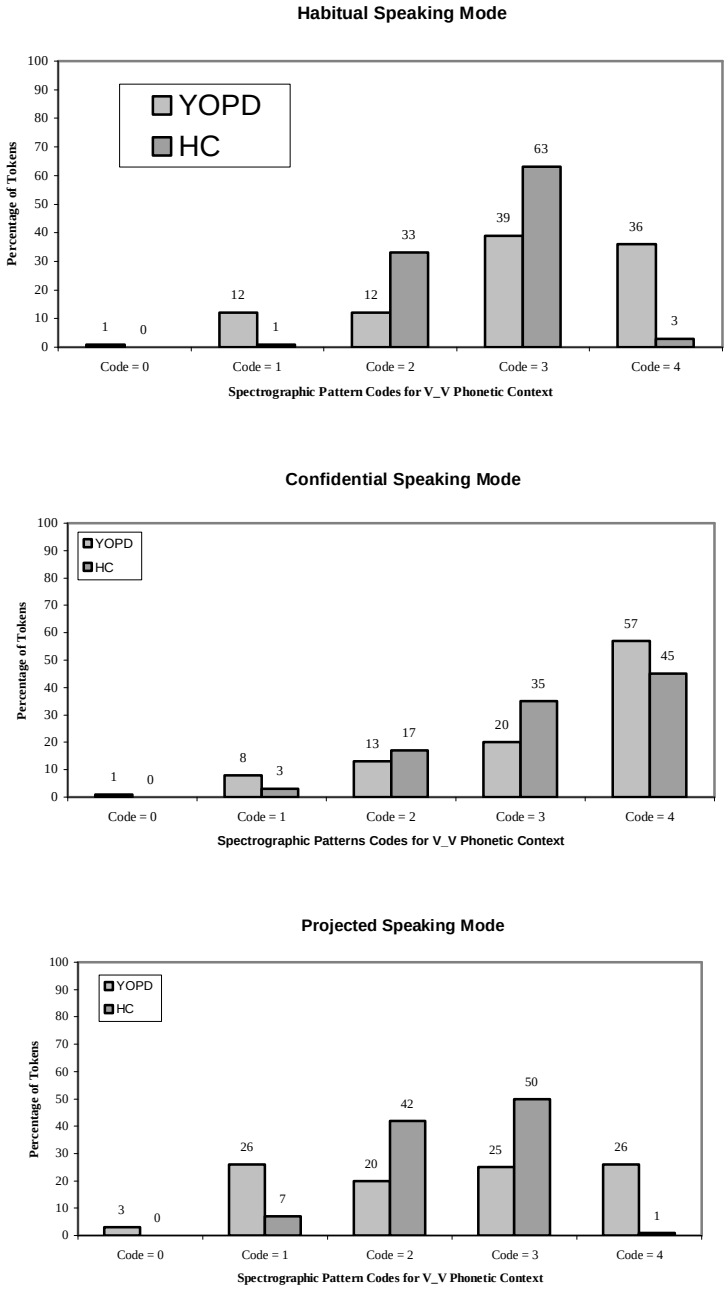
Examination of bar graphs for spectrographic patterns reflecting phonatory offset-onset mechanisms in an open vocal tract (e.g. active mechanism involving the use of intrinsic laryngeal musculature) demonstrated marked differences in spectrographic patterns in YOPD speakers compared to HC speakers in the habitual speaking mode. As illustrated in Figure 40, the hypothesis that YOPD speakers would not demonstrate appropriate phonatory offset-onset in the V_V context, which requires a laryngealization gesture, was generally supported. In the habitual speaking mode, 96% of the tokens produced by the HC Speakers were either a glottal fry (Code 2) or a glottal stop (Code 3), whereas only 1% of the tokens involved minimal devoicing (Code 1), and 3% were associated with

spirantization (Code 4), indicative of incomplete glottal closure during phonatory offset-onset. In contrast, only 51% of the tokens produced by the YOPD speakers were either a glottal fry (Code 2) or glottal stop (Code 3), whereas 13% of the tokens involved either no gesture (Code 0) or a minimal gesture (Code 1). Also, 36% of the tokens were produced without complete glottal closure (Code 4), as demonstrated by spirantization. In the confidential speaking mode, the YOPD speaker group behaved more similarly to the HC group. In comparison with the performance noted in the habitual mode, both speaker groups demonstrated an increase in spirantization, as would be expected when speaking in an aspirant mode. However, YOPD speakers continued to demonstrate a greater percentage of spirantization (57%) than the HC speakers (45%). In addition, a greater percentage of minimal phonatory offset-onset continued to be observed for the YOPD speakers (8%) in comparison with the HC speakers (3%).

Similar changes were also observed for both speaker groups in the projected mode, as compared to the habitual mode. Both groups decreased the percentage of complete glottal stops (code 3) and spirantizations (Code 4) and increased the percentage of glottal fry (Code 2) and minimal devoicing (Code 1). However, the YOPD speakers had a smaller percentage of complete glottal stops (25%) in comparison to that observed in the HC speaker group (50%). Similarly, the percentage of spirantizations was higher for the YOPD speaker group (26%) as compared with the HC group (1%). While the percentage of glottal fry tokens remained greater for the HC speaker group (42% versus 20%), the occurrence of minimal or no devoicing was higher for the YOPD speakers (29% versus 7%). In

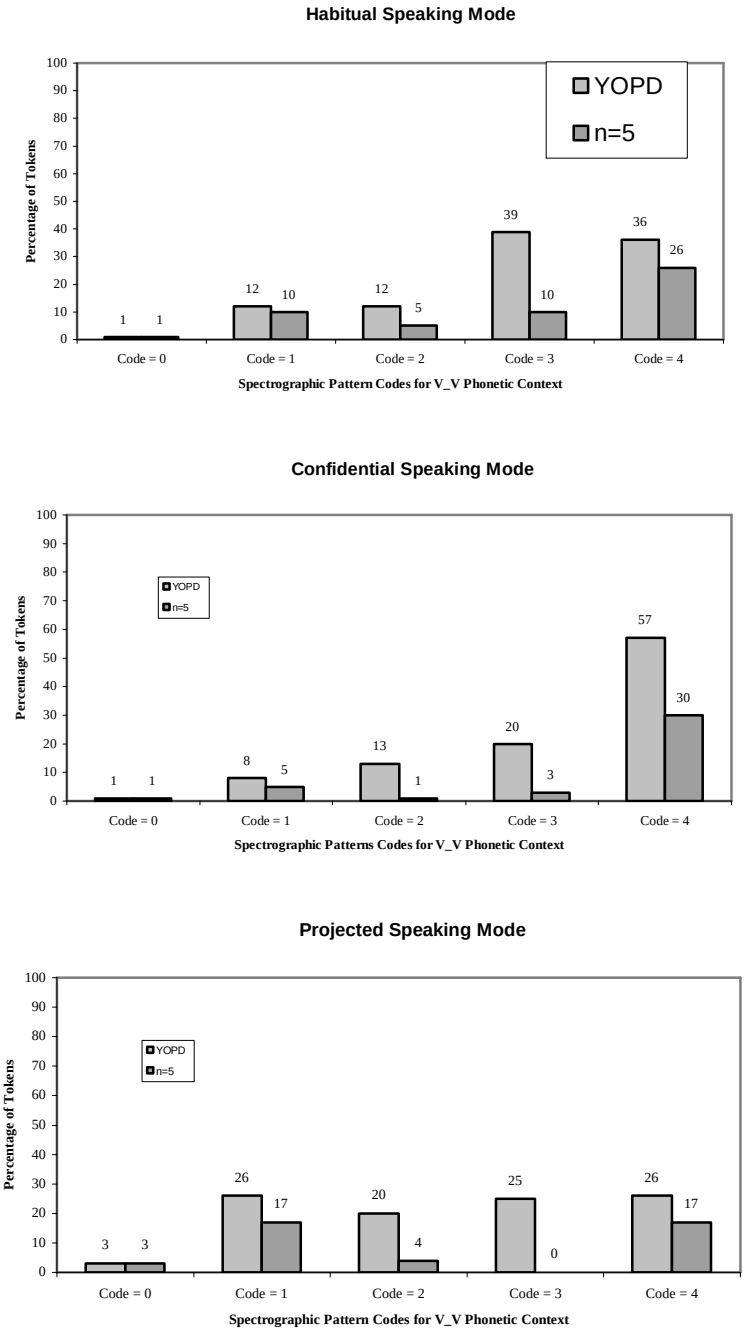
summary, apparent speaker group differences were more evident for the habitual and projected modes, whereas the two speaker groups behaved more similarly in the confidential mode.

Figure 40. Percentage of tokens coded for spectrographic patterns appropriate for laryngealization phonatory offset-onset in the phonetic context V_V across three speaking mode conditions for YOPD and HC Speaker Groups.



Data for the five YOPD speakers, identified previously as clearly impaired in their measures of spectral energy change during phonatory offset onset (see Figure 35), provided verification of apparent group differences and trends described previously. Figure 41 illustrates the spectrographic pattern codes for the laryngealization gesture. These speakers accounted for 70% of tokens produced by YOPD speakers with spirantization (Code 4) in the habitual mode while demonstrating a low percent of glottal fry (Code 2) and glottal stops (Code 3) without spirantization. Furthermore, these speakers accounted for all tokens produced by the YOPD speaker group in the habitual mode with no phonatory offset (Code 0) as well as 83% of the tokens with minimal phonatory offset-onset (Code 1). In the confidential mode, like the YOPD speakers as a group, these five speakers decreased their percent of tokens with spirantization (Code 4). In the projected mode, these five speakers accounted for 69% of tokens produced by the YOPD speaker group with no or minimal phonatory offset-onset (Codes 0 and 1) and 65% of the YOPD speaker tokens produced with spirantization (Code 4). In summary, these five YOPD speakers were major contributors to the speaker group differences described previously for the laryngealization gesture, and they corroborated the characteristics or group trends that appeared to differentiate the YOPD and HC speaker groups.

Figure 41. Percentage of spectrographic pattern codes for the V_V context accounted for by the five impaired YOPD speakers as compared to the total percentage of codes observed in the YOPD speaker group for three speaking modes.



The percentage of tokens coded for spectrographic patterns appropriate for phonatory offset-onset with aspiration in the phonetic context V_H across three speaking mode conditions for YOPD and HC speakers is presented in Figure 42. The greatest group difference with regards to complete phonatory offset-onset (Code 6) was observed for the habitual speaking mode in that 92% of the tokens produced by the HC Speakers were complete while only 68% of the tokens produced by the YOPD speakers were complete. In contrast, YOPD speakers produced a greater percent of tokens coded as partial (16% versus 6% for Code 5), minimal (13% versus 3% for Code 1) or no phonatory offset-onset (3% versus 0% for Code 0) than HC speakers. Again, YOPD speakers became more similar to HC speakers in the confidential mode. While HC speakers generally maintained the same high level of complete phonatory offset-onsets, for the V_H phonetic context, YOPD speakers increased their percent of complete phonatory offset-onset tokens from 68% to 81%, thus becoming more similar to HC speakers. However, the YOPD speaker group did maintain a higher percent (10% versus 2%) of tokens with minimum devoicing (Code 1). Similar adjustments in spectrographic patterns of phonatory offset-onset from the habitual to the projected mode were observed in both speaker groups. Both speaker groups decreased their percent of tokens with complete phonatory offset-onset and increased their percent of tokens with both partial (Code 5) and no or minimal (Codes 1 or 2) phonatory offset-onset. Again, the YOPD and HC speakers groups became quite similar in the projected mode. In summary, speaker group differences were observed for the V_H phonetic context in the habitual mode. However both speaker groups

behaved more similarly in the confidential and projected modes. Speaker group differences were less evident in the confidential mode and more evident in the projected mode.

Again, data for the five YOPD speakers considered to be clearly impaired in their measures of spectral energy change during phonatory offset-onset, especially in phonetic contexts involving an open vocal tract (See Figure 35) provided validation of the apparent group differences suggested above. As illustrated in Figure 43, like the YOPD speakers as a group, these five speakers produced a low percentage of tokens with complete phonatory offset-onset (Code 6) in the habitual mode; furthermore, they accounted for 100% of tokens produced by the YOPD group with no or minimal phonatory offset-onset (Codes 1 or 2). While moderately increasing the percent of their tokens with complete devoicing in the confidential mode (Code 6), these five speakers accounted for the majority of YOPD tokens with minimal (Code 1) and partial (Code 5) phonatory offset-onset. Similarly, these five speakers accounted for 100% of YOPD group tokens with no or minimal phonatory offset-onset (Codes 1 or 2) and were major contributors to the partial offset-onset (Code 5) tokens. In the projected mode, these speakers were minor contributors to the complete phonatory offset-onset (Code 6) tokens demonstrated by the YOPD group and accounted for 100% of the YOPD group tokens with no (Code 0) or minimal (Code 1) phonatory offset-onset and 35% of the partial phonatory offset-onset (Code 5). In summary, group differences summarized previously were clearly manifest in five YOPD speakers considered to be impaired.

Figure 42. Percentage of tokens coded for spectrographic patterns appropriate for aspiration phonatory offset-onset in the phonetic context V_H across three speaking mode conditions for YOPD and HC Speaker Groups.

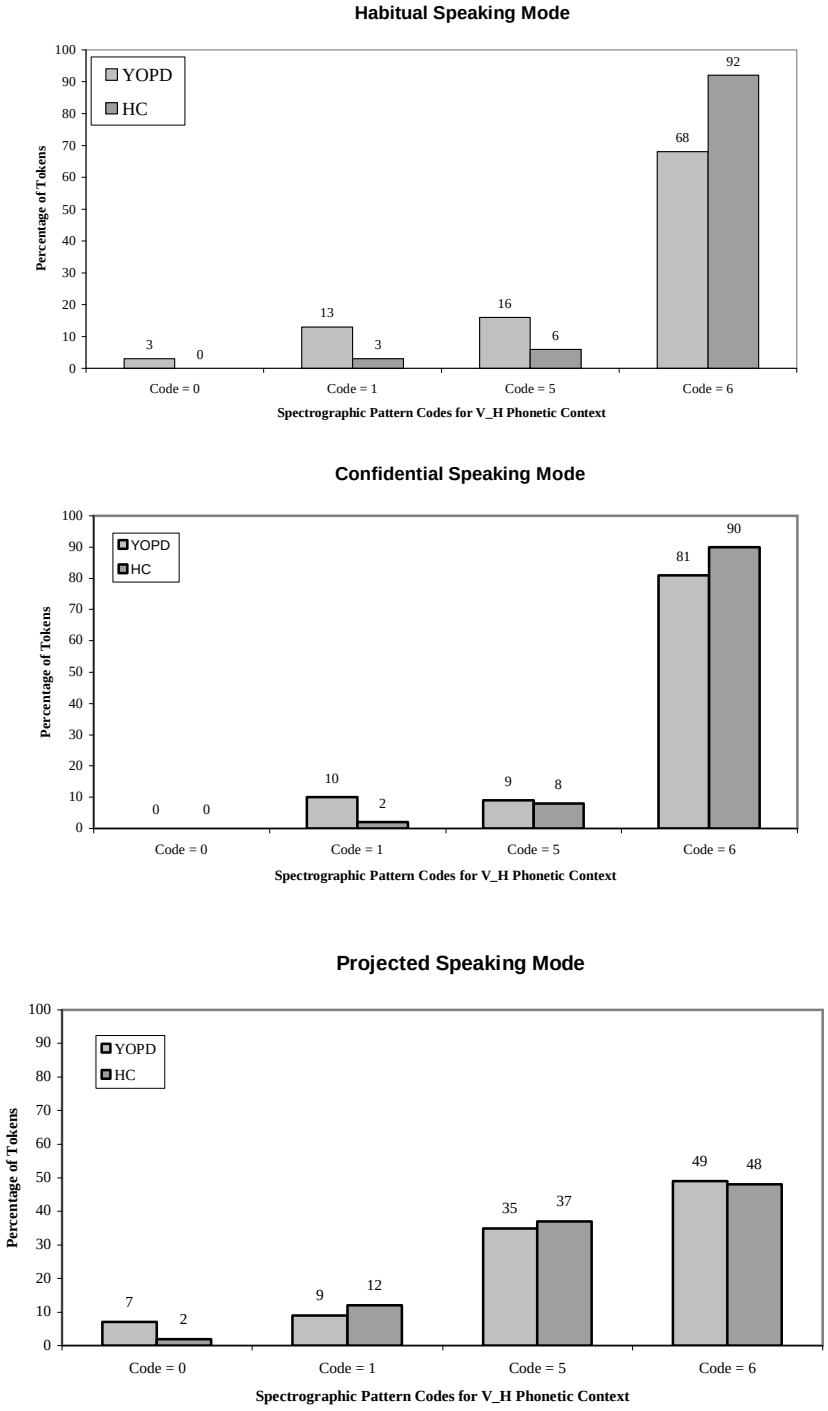
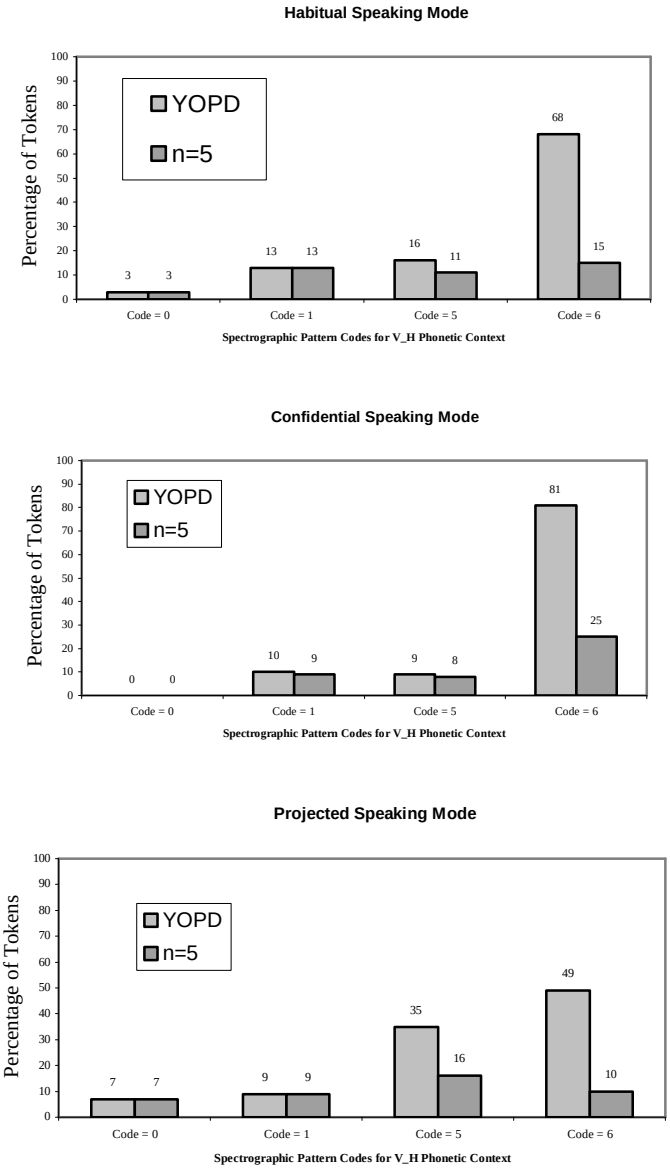


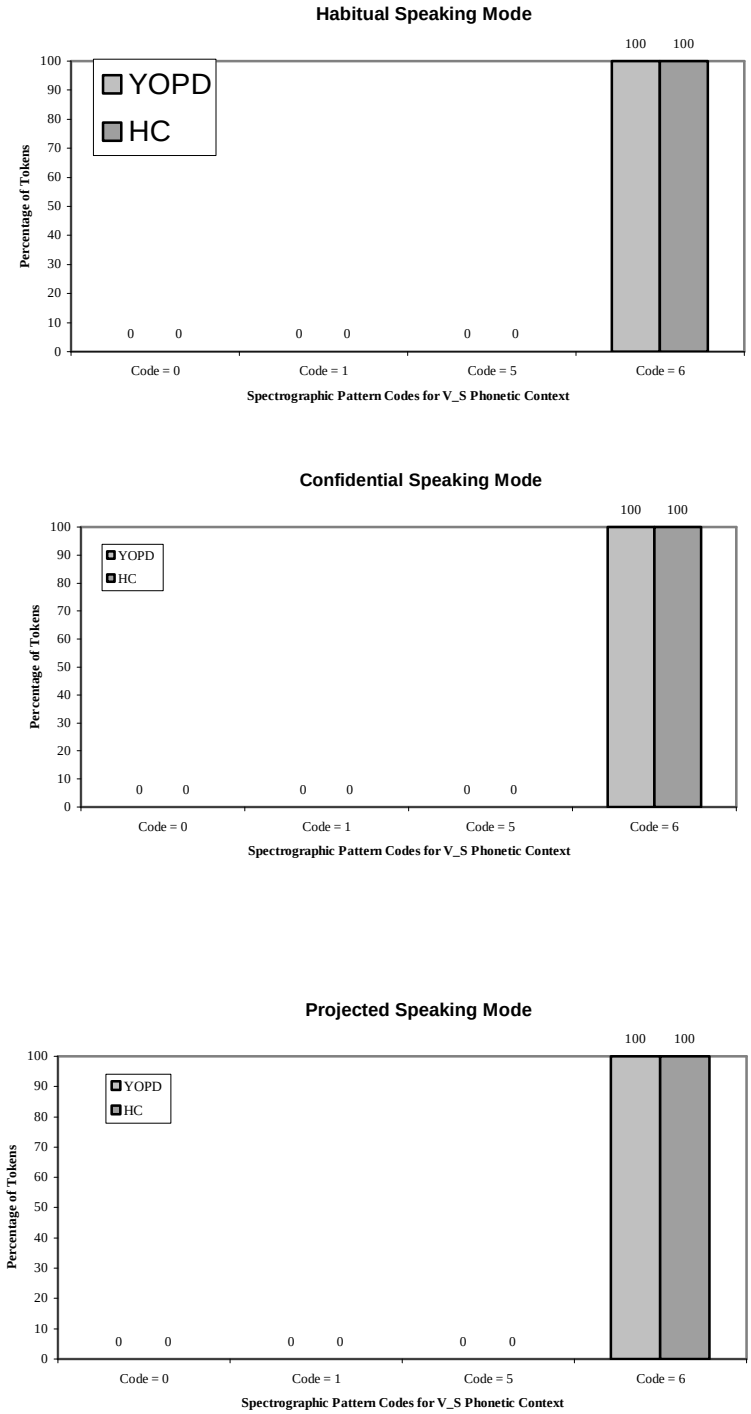
Figure 43. Percentage of spectrographic pattern codes for the V_H context accounted for by the five impaired YOPD speakers as compared to the total percentage of codes observed for the YOPD speaker group for three speaking modes.



Mechanisms involving in an orally constricted vocal tract.

As illustrated in Figure 44, neither of the speaker groups demonstrated impaired phonatory offset-onset control for the aspiration gesture assisted by oral constriction (e.g., V_S). Both speaker groups demonstrated 100% complete devoicing for each of the speaking modes. This finding supported the research hypothesis that YOPD speakers would demonstrate good phonatory offset-onset control when assisted by an oral constriction.

Figure 44. Percentage of tokens coded for spectrographic patterns appropriate for aspiration phonatory offset-onset in the phonetic context V_S across three speaking mode conditions for YOPD and HC Speaker Groups.



Multiple phonatory offset-onset mechanisms.

Unlike HC speakers, YOPD speakers did not demonstrate consistent multiple mechanisms of phonatory offset-onset control associated with production of both an aspiration and laryngealization gesture in the phonetic context S_V. As shown in Figure 45, the hypothesis that YOPD speakers would inconsistently exhibit multiple phonatory offset-onset control (i.e., double gestures) associated with use of an aspiration gesture followed by a laryngealization gesture in the S_V context was supported, particularly in the habitual and projected speaking modes. YOPD speakers used single gestures (e.g., only an aspiration gesture) in 21% of the tokens in the habitual mode and 26% of the tokens in the projected mode as compared to the HC speakers, who used single gestures in only 3% of the habitual tokens and 10% of the projected tokens. Speaker group differences were less apparent in the confidential mode, in that both speaker groups produced a high percentage of double gestures (93% for YOPD speakers, 99% for HC speakers).

The percentage of single gestures for the S_V phonetic context accounted for by the five impaired YOPD speakers is illustrated for three speaking modes in Figure 46. For the habitual mode, 92% of the single gestures observed in the YOPD speaker group can be attributed to these five impaired speakers, whereas 80% of the single gestures were accounted for by these speakers in the confidential and 63% in the projected speaking modes. Therefore, the majority of the production of single gestures was observed in these impaired speakers, which corroborated the group trends suggested above.

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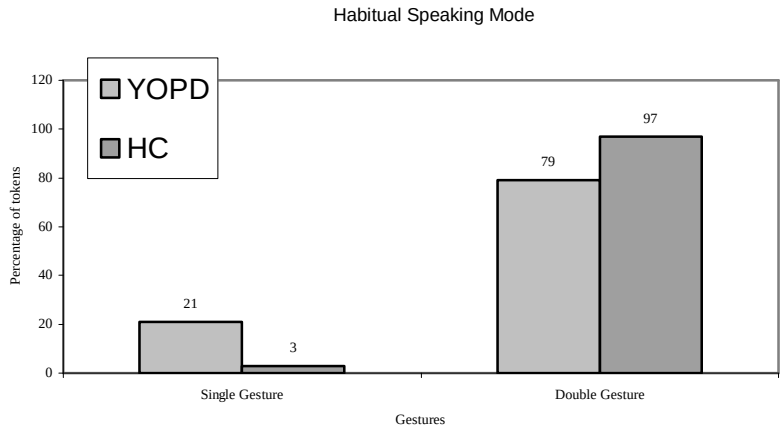
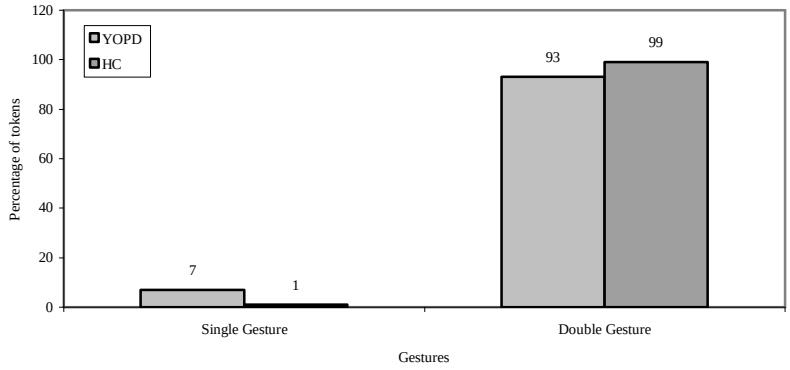
Figure 45. Percentage of tokens coded for spectrographic patterns reflecting single and double gestures in S_V phonetic context across three speaking mode conditions for YOPD and HC speaker groups.





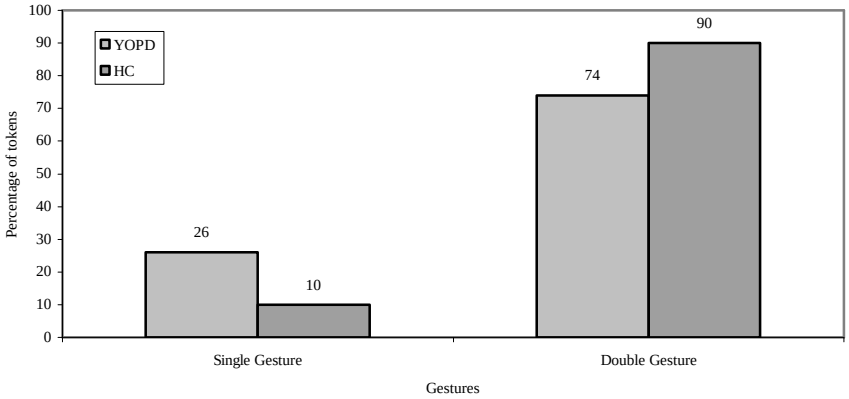
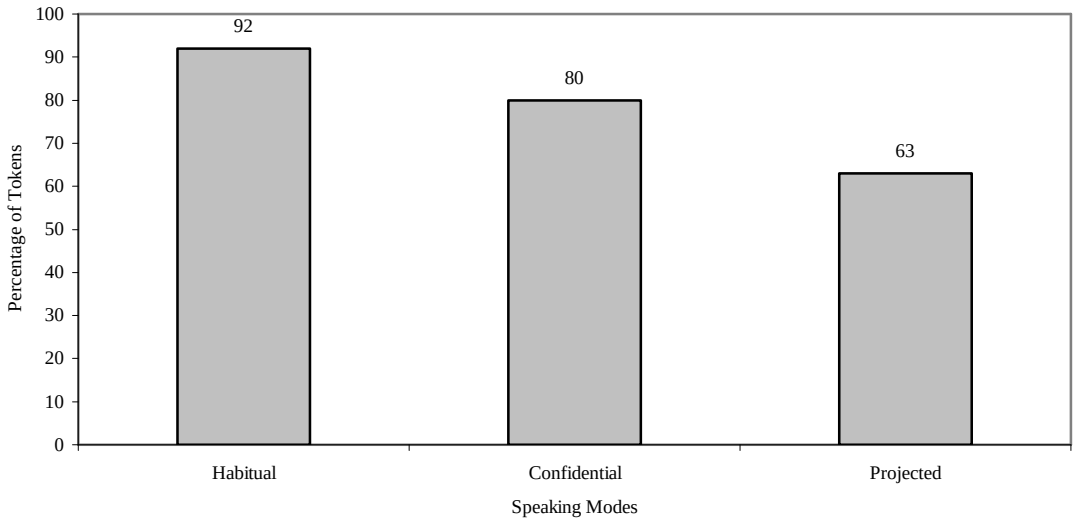


Figure 46. Percentage of single gestures for the S_V phonetic context accounted for by the five impaired YOPD speakers for three speaking modes.



Discussion

This study serves as a preliminary investigation of vocal dysfunction in persons with YOPD. Group comparisons across five vocal parameters provide a basis for future treatment of vocal dysfunction for this population. The parameters include (a) the voice and speech symptoms reported by YOPD speakers, (b) the negative impact of these symptoms on QOL, (c) performance on clinical tasks of sustained vowel phonation and L-DDK, (d) speech intensity, mechanisms of phonatory offset-onset control reflected in a measure of intervocalic offset-onset spectral energy change, and spectrographic pattern codes; and (e) the effect of phonetic context and voice mode manipulation on speaking intensity, intervocalic offset-onset spectral energy change, and measures of spectrographic pattern codes.

Voice and speech symptoms and the negative impact on QOL

As expected, YOPD speakers reported a significantly greater percentage of occurrence of deviant voice characteristics related to loudness, tremor, hoarseness, and monotone in comparison to those reported by HC speakers. Furthermore, YOPD speakers reported a significantly greater percentage of occurrence of attributes related to impaired speaker intelligibility (slurs, mumbles, and understandability). The novel findings of this study provide the first empirical data to demonstrate the occurrence of self-perceived deviant voice characteristics in YOPD speakers that are similar to those reported in the literature by persons with older-onset PD (Sapir et al., 2001). It is clear that, regardless of aging effects, PD is associated with vocal dysfunction.

This study corroborates the findings of Hanson et al. (1984), Logemann et al., (1978) and Tetrud (1991) that voice symptoms can appear early in the disease or precede the development of limb symptoms. In the present study, 4 participants (33%) stated that they noticed symptoms of hypophonia, hoarseness, or restricted pitch range prior to the onset of limb symptoms of rigidity or tremor. Thus, the association of vocal dysfunction with the early stages of PD was corroborated in this study. Recently, Del Tredici, Rub, De Vos, Bohl, and Braak (2002) have suggested that the substantia nigra may not be the induction site in the brain for PD. Instead, these researchers suggested that PD might start in non-catecholaminergic neurons of the dorsal glossopharyngeus-vagus complex, among other places. The dorsal glossopharyngeus-vagus complex drives the motor and sensory neurons that control the laryngeal system.

The findings from this study are the first to document that vocal dysfunction in YOPD has significant psychosocial consequences for these individuals that place emotional, functional and physical restrictions on their daily living. These findings are not consistent with those reported for persons with older-onset PD (Yorkston & Beukelman, 1984). While older-onset persons with PD complained of communication deficits, they indicated that these deficits did not interfere with their social lifestyles. However, differences in psychosocial consequences of PD in young-onset and older-onset groups have been reported previously (Shrag et al., 1998). In comparison to persons with older-onset PD, Shrag and colleagues reported that persons with YOPD reported lower marital satisfaction, poor stigma, greater depression, a higher occurrence of low

employment, and an increased disruption of family life. As suggested by Brod et al. (1998), persons with YOPD reported impaired communication skills as a major factor of disability, often leading to changes in career goals and early retirement. In summary, findings of the current study provide evidence for the need to assess and treat vocal dysfunction in the YOPD population. Mild, subtle voice symptoms have a significant negative impact in the lives of persons with YOPD, and there is a critical need for early intervention and prevention of the handicapping effects of vocal dysfunction in activities of daily living.

As expected, the YOPD speaker group reported a significantly lower Physical Component Score on the SF-12 Health Survey than the HC speaker group. Although the YOPD speakers' Mental Component Score was lower in comparison with the HC speaker group, the difference was not statistically significant. This latter observation was consistent with previous findings reported by Sapir et al. (2001). In their study, no significant difference in depression was noted between the "younger group" of participants (ages 32 to 56 years) and the "older group" (ages 70 to 83 years). However the findings of the present study and those of Sapir et al. are not consistent with two investigations that have reported a greater incidence of depression in young-onset in comparison with older-onset persons with PD (Starkstein et al., 1989; Stern et al., 1994). It is important to note that four participants (33%) in the present study were taking medications for depression. It is possible that their pharmacological treatment served to mask true group differences in depression.

Performance on clinical tasks

The findings from this study indicated no significant speaker group differences with respect to production of sustained vowel phonation. First, there was no significant difference observed for mean F_o between the two speaker groups. This finding is consistent with the literature in that researchers have not reported differences in mean F_o in persons with older-onset PD as compared to healthy control speakers (Darley et al., 1969; Ludlow & Bassich, 1983).

Comparison of speech intensity for sustained phonation in both speaker groups also did not reveal significant differences. This finding is inconsistent with documentation that reduced loudness (hypophonia) is generally recognized as one of the predominant voice symptoms in older-onset PD (Canter, 1963; Darley et al., 1969a; Ramig et al., 2002) and occurs in the early stages of the disease as well (Stewart et al., 1995; Tetrud, 1991). While perceptual studies of conversational speech have typically found parkinsonian speech to be reduced in volume (Ackermann & Ziegler, 1991; Darley, Aronson, & Brown, 1969b), researchers who have studied speech intensity levels or loudness levels for isolated tasks of sustained phonation have not reported reductions in vocal loudness or intensity (SPL) (Ramig et al., 2002). Therefore, the findings in the present study corroborate previous studies in that hypophonia in PD is not typically revealed in a sustained vowel phonation task.

Finally, no significant group differences were found for the voice spectra perturbation measures (jitter and shimmer), reflecting acoustical attributes related to voice quality parameters such as breathiness, hoarseness, roughness or

harshness. This suggests that changes in voice quality were not evident in the sustained phonation task. Also, there was no speaker group difference observed for the measure of soft phonation index (SPI). This measure is an indicator of how completely or tightly the vocal folds adduct during phonation. Increased values suggest incomplete vocal fold closure associated with sustained vowel phonation. The normative mean provided by the software manufacturer (Kay Elemetrics, 1999b) is 14.12. In the present study, both speaker groups demonstrated considerably higher values than this norm (M = 39.1 for YOPD and M = 36.5 for HC speaker groups). This suggests that both groups either sustained vowel phonation using a soft glottal attack, or they sustained phonation using a loosely adducted phonatory posture. Thus, current findings did provide indirect evidence of a breathy voice quality expected to occur in YOPD speakers, but not expected in HC speakers.

Current findings for the sustained vowel task are generally inconsistent with previous research reported by Kent et al. (1999) for predominantly older-onset PD speakers. These researchers reported abnormal parameters for five of the measures used in the present study. Their participants included nine persons with older-onset PD and only one person with young-onset PD. Also, it is important to note that the aim of the Kent et al. study was to determine the reliability of the voice spectra measures. The PD participants were not compared with a healthy control group. Rather, they were compared to the normative data provided by Kay Elemetrics (1999b) that is based on a small cohort. The validity of these norms has been questioned (Radionoff, 1996; Samlin, 2003, personal correspondence), and

the manufacture has indicated that the normative thresholds are based on a small sample of 20 persons (Kay Elemetrics, 1999b). Thus, the findings of Kent et al. may be attributable in part to inadequate norms. Alternatively, one could posit that capturing voice spectra measures in an initial 3-sec segment of sustained vowel phonation is not a sensitive tool for detecting vocal dysfunction. The laryngeal dynamics involved in sustained vowel phonation are markedly different from those dynamics involved during conversational speaking or reading, where phonatory offset-onset occurs. It is noteworthy that the investigator in the present study was aware of deviant voice quality symptoms in all the PD speakers based on perceptual judgment of their conversational speech, but not the HC speakers.

As expected, speaker group differences were found for both L-DDK tasks. For the adductory task, the YOPD speakers demonstrated a significantly slower rate of syllable repetition than HC speakers, although their proportion of production of abductory syllable sequences was comparable to the performance of the HC speakers. It is important to note that speakers in the present study were instructed to keep the syllables distinct, which encouraged production of glottal stops. The slower rate of repetition in YOPD speakers represents impairment in their ability to rapidly produce glottal stops. This finding is inconsistent with the previous research reported by Ludlow and Bassich (1983) and Kent et al. (1994) who investigated the adductory L-DDK gesture in a group of older-onset PD speakers. When not specifically instructed to keep the syllables distinct, older-onset speakers demonstrated a tendency towards continuous voicing and used a

rapid syllable repetition rate. Thus, this inconsistency in findings might be attributable to procedural differences.

Speaker group differences were found for the production of the abductory L-DDK, in that only the YOPD speakers evidenced pauses during the 7-sec interval. This finding is consistent with previous studies that reported older-onset PD speakers use more pauses and fewer syllables per breath group (Hammen & Yorkston, 1996; Hammen, Yorkston, & Beukelman, 1989; Metter & Hanson, 1986; Pitcairn et al., 1990; Solomon & Hixon, 1993; Tjaden, 2000; Volkmann, Hefter, Lange, & Freund, 1992). Impaired function for the abductory L-DDK task in PD speakers may suggest an inefficient valving of the pulmonary airflow through the glottis that results in increased pauses secondary to a rapid depletion of the exhalatory breathstream.

Speaker group differences in speech intensity and phonatory offset-onset control of laryngeal and aspiration gestures in various speaking modes

In the following sections, relevant findings are discussed with respect to speaker group differences observed for the sentence stimuli, the comparison of phonatory offset-onset gestures (phonetic contexts) and the effects of speaking mode manipulation. The dependent measures included acoustic measures of speech intensity, intervocalic spectral energy change, and spectrographic pattern codes.

Speaker group differences

Statistically significant speaker group differences were not observed for the acoustic measures of speech intensity and intervocalic spectral energy change; however, descriptive analyses of spectrographic pattern codes tentatively suggest speaker group differences, particularly for the habitual and projected speaking modes, for both the laryngealization and aspiration gestures produced without oral constriction. Furthermore, intra-speaker group variability was observed for the YOPD speakers. The highlights are reviewed in the following three sections for each dependent measure (speech intensity, intervocalic spectral energy change, and spectrographic pattern codes.

Speech Intensity for Reading.

As seen with sustained vowel phonation, the hypothesis that YOPD speakers would demonstrate lower values of speech intensity during reading was not supported in the present study. Again this finding is inconsistent with the occurrence of reduced loudness or hypophonia that is generally recognized as one of the predominant voice symptoms in older-onset PD (Ackermann & Ziegler, 1991; Canter, 1963; Darley, Aronson & Brown, 1968b; Ramig et al., 2002; Stewart et al., 1995; Tetrud, 1991). However, some investigators have not reported reductions in vocal loudness or intensity (SPL) during reading (Boshes, 1966; Ludlow & Bassich, 1984; Metter & Hanson, 1986). More recently, Ramig et al. (2001) reported task related differences in mean SPL for 12 persons with PD two years post LSVT™. The tasks included sustained vowel phonation, reading, and monologue. Reported values for mean speech intensity (dB SPL) for the three

tasks were as follows: (a) sustained vowel phonation, M = 76.5, SD = 4.1; (b) reading, M = 69.78, SD = 3.19; and (c) monologue, M = 67.02, SD = 1.87. Hypophonia was more evident for the monologue task, which is more similar to conversational speech as compared to sustained phonation or reading. Ho, Bradshaw et al. (1999) also commented that persons with PD increase vocal loudness when they are more attentive to their speech execution. It is possible that the PD participants in the present study were more attentive to their speech execution during the experimental reading tasks. Furthermore, the majority of the YOPD speakers in the present study indicated hypophonia as a predominant feature in their self-reporting of voice symptoms. Also, this investigator noted incongruence in the perceptual impression of loudness between conversational speech during the initial participant interview and the loudness level used by speakers during the experimental reading and sustained phonation tasks. Reduced loudness was evident for conversational speech, but not apparent during the experimental reading and sustained phonation tasks.

Intervocalic Spectral Energy Change.

Speaker differences did not achieve statistical significance (p = .06) for the phonatory offset-onset of intervocalic spectral energy change. The finding of no significant speaker group difference in this acoustic measure may be attributed to intra-group variability within the YOPD speaker group. Five YOPD speakers were clearly impaired, and they accounted for a large proportion of the spectrographic pattern codes of partial or no phonatory offset-onset, while six YOPD speakers demonstrated near normal or mild impairment. One YOPD speaker, who was one

of two professional voice users in this investigation, demonstrated intervocalic spectral energy change values that were greater than the average values observed in the HC speaker group for the adductory devoicing gesture. Furthermore, this speaker's measures for the abductory devoicing gesture were similar to values obtained for the HC speaker group. Such findings suggest this speaker demonstrated a more consistent and greater degree of complete phonatory offset-onset, especially for the laryngealization gesture. Although this speaker did not evidence voicing control deficits, he reported a moderate voice handicap related to voice symptoms of hoarseness, monotone, and decreased speech intelligibility. This speaker did not report hypophonia as a predominant symptom.

Spectrographic Pattern Codes.

The group trends in spectrographic pattern codes of phonatory offset-onset provided documentation of impaired voicing control in a habitual speaking mode of persons with YOPD. In comparison with HC speakers, the spectrographic pattern codes of YOPD speakers demonstrated a higher percentage of partial or no phonatory offset-onset for both laryngealization and aspiration gestures produced without oral constriction. Additionally, YOPD speakers produced fewer tokens with multiple phonatory offset-onset gestures. Interestingly, spectrographic pattern codes for phonatory offset-onset aspiration gestures associated with oral constriction (V_S) were similar to those produced by HC speakers. The observation of no apparent group differences for this phonetic context involving oral constriction was consistent across speaking modes. Clearly, YOPD speakers were able to achieve phonatory offset-onset when assisted by a passive mechanism

associated with oral constriction. Stevens (1991, 1999) has pointed out that an oral constriction and the resultant increase in intraoral pressure can create a force on the surface of the vocal folds that dampens glottal vibrations.

Evidence of individual variability was again observed in the YOPD participants, consistent with the hypothesis of heterogeneity of variance. Patterns observed in five clearly impaired speakers demonstrated the group trends of the YOPD speaker group that were most evident in phonetic contexts involving an open vocal tract (V_V, V_H) in general and the V_V context in particular. These five speakers' performance confirmed that speaker group differences noted in spectrographic patterns do in fact represent impaired function.

The signs of vocal dysfunction reflected in spectrographic pattern codes corroborated the findings from the intervocalic spectral energy change measures. Group trends were reflected in the data of five speakers with clear impairment of mechanisms of phonatory offset-onset control. In fact these impaired speakers accounted for a large proportion of the apparent group differences, thus confirming a lack of homogeneity with respect to vocal dysfunction in YOPD speakers. The observed heterogeneity in the YOPD speaker group may be accounted for by (a) gender differences or (b) may represent the occurrence of subtypes of PD. With respect to gender differences, Kent et al. (1994) reported that males demonstrated a greater phonatory offset-onset deficit in producing "at" versus "hat" in comparison to females, who did not demonstrate a higher error rate for this contrast. In the present study, four of the five impaired speakers were males. The male:female ratio in this study was 7:5. Therefore, the males accounted for a

proportionately greater percentage of the observed impairment in phonatory offset-onset control

A more plausible explanation for the observed heterogeneity in YOPD speakers may rest on anatomical- and functionally-based hypotheses that there are subtypes of PD. Researchers have hypothesized the existence of clinical subtypes of PD, including one group presenting with bradykinesia, postural instability, and gait difficulty (PIGD) and another presenting as tremor-dominant PD (Bostantjopoulou et al., 1991; Jankovic et al., 1990; Rajput et al., 1993). Therefore, there may be two different clinical syndromes within the YOPD population and different symptoms of vocal dysfunction may be associated with each syndrome. It is clear that there are eight functionally distinct and segregated cortico-striatө pallido-thalamo-cortical loops, and there is somatotopic organization, similar to that observed in primary and secondary motor and sensory cortices (Borrett et al, 1993; Obeso et al., 1997; Wichmann & DeLong, 2003). For example, there are specific "arm," "leg," "larynx" loops projecting to corresponding frontal lobe areas. These areas could be differentially affected in persons with PD and account for observed differences in limb and speech symptoms (Alexander et al., 1986). There appears to be little or no correspondence between voice and speech deficits and duration of PD, nor is there correspondence between severity of limb symptoms and voice or speech deficits. Also, the patterns of hypokinetic dysarthria observed in persons with PD are highly variable (Gamboa et al., 1997; Metter & Hanson, 1986; Schulz et al., 1999). Thus, the findings in the present study of intra-speaker group variability with

respect to deficits in phonatory offset-onset mechanisms are consistent with the reported heterogeneity of the disease process in PD.

Comparison of phonatory offset-onset mechanisms

There was a significant phonetic context effect consistent across speaker groups and speaking modes. Speech intensity level was significantly greater for the sentence stimuli involving the phonatory offset-onset aspiration gesture associated with a completely open vocal tract (V_H context) in comparison with stimuli for the aspiration gesture, associated with an oral constriction (V_S) and the laryngealization gesture, associated with a glottal constriction. This finding suggests that the presence or absence of vocal tract constriction may affect speech intensity level. The V_H phonetic context is associated with a completely open vocal tract as compared to the glottal constriction associated with the V_V phonetic context and the oral constriction associated with the V_S phonetic context. Perhaps the lack of vocal tract constriction during the intervocalic interval associated with the V_H phonetic context has the effect of increasing respiratory drive for the entire syllable, resulting in increased vowel intensity. It is known that sub-glottal air pressure and glottal resistance interact in the control of speech intensity (Dromey et al., 1995).

With respect to the measures of intervocalic spectral energy change, a significant consistent phonetic context effect across speakers groups was observed, but this effect was inconsistent across speaking modes. While a consistent effect across phonetic contexts was observed in the projected mode, the effect in the confidential and habitual modes was limited to the V_V/V_H and V_V/V_S

comparisons; no effect was observed for the V_V/V_S comparisons. Increased spectral energy change was evident in the V_V context in comparison to the V_H and V_S contexts for all speaking modes. The laryngealization gesture is associated with greater intervocalic spectral energy change than the aspiration gesture with or without oral constrictions.

Phonetic context effects were observed for spectrographic pattern codes, suggesting that the spectrographic coding system clearly differentiated the laryngealization and aspiration gestures. All tokens involving partial or complete phonatory offset-onset for the aspiration gesture were associated with codes of 5 or 6, while all tokens involving partial or complete phonatory offset-onset for the laryngealization gesture were associated with codes of 2,3, or 4. No overlap was observed between the codes assigned to different mechanisms of phonatory offset-onset. Furthermore, the code of 4 clearly identified spirantization associated with a laryngealization gesture, and provided evidence of loosely approximated vocal folds.

Effects of voice mode manipulation

A significant speaking mode effect was found for speech intensity level and was consistent across speaker groups and phonetic contexts. This finding provided experimental verification of the speaking mode tasks. There was a mean group increase of 6 dB observed in both speaker groups for the projected versus habitual modes and a mean group decrease of 6 dB between the confidential versus habitual modes. Speech intensity levels revealed that both speaker groups evidenced a Lombard effect associated with speaking under speech masking noise.

Both groups increased speech intensity levels. The finding that all 12 YOPD speakers demonstrated a Lombard effect is inconsistent with the findings reported in older-onset PD speakers by Adams and Lang (1992). Notably, these researchers did not control for hearing loss. The present study did control for hearing loss and provided evidence that speech masking is an effective facilitator for achieving a full, loud voice in persons with YOPD.

Significant speaking mode effects consistent across speaker groups but inconsistent across phonetic contexts were found for the measure of intervocalic spectral energy change. There was a consistent mode effect on mechanisms of aspiration with or without oral constriction. Higher values of intervocalic spectral energy change were found for the confidential mode, while the least amount of change was observed for the projected mode. However, there was no consistent mode effect for the laryngealization gesture. Only the V_V habitual/projected comparison demonstrated a significant mode effect in that intervocalic spectral energy change was greater for the habitual mode in comparison to the projected mode. No mode effect was observed for the habitual/confidential and confidential/projected comparisons. Increased intra-group speaker variability for the YOPD participants (related to the heterogeneity of PD), especially in the V_V phonetic context, may have limited the speaking mode effect for the V_V phonetic context to the habitual/projected comparison.

Similar changes were observed across speaker groups from the habitual to confidential modes. The speaker groups became more similar in the confidential mode. With respect to the laryngealization gesture, only the YOPD speaker group

demonstrated a high occurrence of spirantization in the habitual mode, while both speaker groups demonstrated a high occurrence of spirantization associated with the laryngealization gesture in the confidential mode. However, the YOPD speaker group maintained a 12% greater occurrence of spirantization than the HC speaker group. This finding suggests that YOPD speakers demonstrate a habitual speaking mode that is consistent with a breathy or aspirant voice similar to that used by healthy speakers when speaking in a confidential mode.

Similar changes were also observed from the habitual to projected modes, although apparent speaker group differences were maintained. Continued evidence of impairment in the YOPD speakers included (a) greater occurrence of spirantization, (b) greater occurrence of partial phonatory offset-onset, and (c) greater occurrence of no or minimal phonatory offset. In comparison with the habitual mode, both speaker groups decreased the occurrence of spirantization associated with the laryngealization gesture in the projected mode, indicating a tighter approximation of the vocal folds during phonatory offset onset. This improvement was especially noticeable for the YOPD speakers. Furthermore, there was a decrease in the occurrence of complete phonatory offset-onsets for both the adductory and abductory mechanisms produced without oral constriction observed for both speaker groups. This decrease in complete phonatory offset-onset gestures was not predicted, as it was hypothesized that increasing speech intensity and the resultant increase in vocal effort would improve mechanisms of phonatory offset-onset, particularly in an impaired speaker. Retrospectively, this hypothesis did not consider all of the effects of projected speech. One such effect

was that perceptually both speaker groups demonstrated "legato speech" in the projected mode. The term "legato" has its derivation in the music literature. It refers to a smooth connection between two adjacent tones where no break is heard perceptually between the two tones (Repp, 1997). Legato speech is defined as "the uninterrupted voicing pattern that seems to flow without break" (Boone & McFarlane, 2000, p. 13). In legato speech, there is diminished phonatory offset-onset and words run continuously together (Packman, Onslow, & Menzies, 2000). Therefore, the legato style of speaking that accompanied the voice mode change observed in both speaker groups may have accounted for the observed decrease in complete phonatory offset-onset gestures in both mechanisms produced without oral constriction. In summary, both speaker groups demonstrated a similar change in the laryngeal mode setting associated with the projected speaking mode. This mode change seemingly increased glottal closure, as observed by the decreased occurrence of spirantizations, although both speaker groups demonstrated legato speech that resulted in a decrease in occurrence of complete phonatory offset-onset gestures associated with the laryngealization and aspiration gestures produced without oral constriction.

Limitations

The present findings should be considered preliminary and their interpretation tentative. The lack of significant group differences with respect to the intervocalic spectral energy and voice spectra measures may have been related to methodological shortcomings, such as the small sample size and the intra-group variability observed for the YOPD speakers. It may be that such differences were

not detected because of decreased power (i.e., Type II error) related to small sample size. Thus, a larger sample size would have provided more power. Additionally, the large intra-group variability observed in the YOPD speaker group and speaker group differences were more evident in the habitual speaking mode.

The subject selection criteria, while controlling for "nuisance" variables, limits external validity with respect to the generalization of the present findings to the YOPD population. It is important to note that many of the YOPD speakers, who volunteered to participate in this study but were excluded, evidenced marked problems in the control of speaking rate and demonstrated inconsistently dysfluent speech. All of these persons had initiated L-dopa treatment. It has been suggested that rapid speech rate (tachyphemia) and dysfluency (palilalia) may be a side effect of the medication and not a primary symptom associated with PD (Fahn, 1995). Such speakers were excluded from the present investigation, in accordance with the participant selection criteria, because valid acoustic measures could not be obtained from dysfluent speech. Furthermore, persons with YOPD who had undergone a surgical procedure for the treatment of PD symptoms (e.g., Pallidotomy or DBS) were excluded from the present study. Therefore, the findings of vocal dysfunction in the present study cannot be generalized to post surgerical cases of YOPD.

A further confounding or nuisance variable that may have contributed to the finding of no significant group differences was that subject selection criteria did not preclude professional voice users. Potentially, this could have posed a

threat to internal validity with respect to subject selection bias or history effects. That is, the trained professional voice users may be using vocal techniques characteristic of trained singers or public speakers; they may be speaking in a different mode.

Acoustic measures were interpreted to reflect evidence of impaired phonatory offset-onset mechanisms with respect to laryngealization and aspiration gestures produced without oral constriction in YOPD speakers. No direct physiological or aerodynamic measures were obtained. Thus, the presumption of impaired voicing control in YOPD speakers awaits verification from future physiologic investigations.

A limited number of acoustic measures, presumably reflecting impaired laryngeal function, were employed in the present study, and they were focused primarily on phonatory offset-onset control. Multidimensional changes involving prosodic (e.g., intonation and speaking rate) and articulatory deficits related to speakers' overall intelligibility were not explored. For example, measures of (a) deviations from normalcy in the time variation of the acoustic pattern within the consonant and at the consonant-vowel boundary, and (b) the spectrum shape of frication noise have been found to correlate with intelligibility and perceptual ratings in speakers who have dysarthria (Chen & Stevens, 2001). Such measures could potentially identify additional differences between YOPD and HC speakers. Furthermore, dependent acoustic measures were obtained in controlled experimental tasks of sustained phonation and reading tasks, which may not reflect vocal dysfunction associated with conversational speaking in YOPD speakers.

Clinical Implications

The finding of speaker differences with respect to self-perceived voice and speech symptoms and the negative impact of PD on quality of life indicates the need to assess vocal dysfunction in the YOPD population to identify those persons in need of voice intervention. Measures of self-report of voice symptoms and speaker intelligibility provide sensitive and efficient voice assessment screening tools for persons with YOPD. None of the individuals with YOPD in the present study who reported a mild or moderate voice handicap had previously sought voice treatment, whereas after participation in this study, two participants enrolled in therapy. All PD participants who reported a mild to moderate voice handicap indicated in the pre-experimental interview that they did not have voice problems. However, following completion of the Visual Analogue Scale and Voice Handicap Index, they became aware of the subtle communication problems they were experiencing that negatively impacted on their daily living. This suggests the need for routine and early assessment for vocal dysfunction in all persons with YOPD. Such assessment should include simple instruments such as the (a) Voice Handicap Index, that assesses the impact of the disorder on daily living, and (b) Visual Analog Scale, that provides a quick assessment of voice symptoms and speech intelligibility. Additional insight and information might be provided by having a spouse or significant other complete the Visual Analog Scale. Also, perceptual evaluation of L-DDK tasks may be sensitive measures for identifying vocal dysfunction. Acoustic measures of intervocalic spectral energy change and spectrographic patterns of phonatory offset-onset mechanisms were shown in the

current investigation to be valid assessment tools for the identification of impairments of dynamic voicing control.

The findings of this study that speech masking facilitated an effective speaking mode change in YOPD speakers suggest that the use of commercially available speech maskers can be used in a clinical setting to promote a full, clear or projected mode. However, a word of caution is needed. Four YOPD speakers experienced an exacerbation in limb tremor during the masking condition. At the end of the experimental protocol, they indicated that although speech masking facilitated a stronger voice, they would not be interested in using the masker as a component of a behavioral voice therapy program. Therefore, the use of speech masking may not be appropriate for all YOPD speakers, particularly those who have symptoms that are predominately tremor.

The high occurrence of vocal dysfunction in persons with YOPD suggests that intervention should be implemented soon after diagnosis of PD. Symptomatic therapy should be initiated to teach techniques that maximize voice quality, and achieve the best full, clear voice as possible. This study demonstrated that YOPD speakers are capable of manipulating their speaking mode. Speech masking noise was shown to be an effective facilitator for eliciting a projected speech mode. This facilitative technique could provide an effective and efficient treatment method for training YOPD speakers to speak in a projected mode. Such treatment might be more effective and efficient for the YOPD population than LSVT™. The LSVT™ program requires intensive (i.e., four 60-minute treatment sessions per week over a 4 week period). Young-onset patients who are engaged in full time employment

might be reluctant to pursue a time-intensive treatment program that requires daily treatments. Furthermore, it is not certain whether young-onset patients would require such intensive treatment. Future research is needed to investigate the efficacy of using speech masking as a facilitator in training a full, clear voice characteristic of a projected mode in persons with YOPD.

Theoretical and Research Implications

Findings from the current study of voice control problems in YOPD speakers cannot be explained by a loudness control hypothesis. Rather, these findings may be explained by a bradykinesia hypothesis. Impairment of adducting and abducting movements of the vocal folds by bradykinesia could account for impaired phonatory offset-onset control as well as the presumed loosely approximated vocal folds that would result in the weak breathy voice characteristic of PD speakers during conversational speech.

The current study established the existence of vocal dysfunction in YOPD using subjective clinical measures and acoustic measures of vocal function. Electromyographic studies of intrinsic laryngeal muscles would be useful in better delineating the mechanism of impairment underlying the phonatory offset-onset control problems in YOPD speakers. Aerodynamic techniques (e.g. airflow and air pressure) would be particularly useful in delineating impaired valving of airflow at the glottis. Airflow and air pressure measures should provide additional insights into the impairments of phonatory offset-onset control in YOPD speakers during the performance of both L-DDK and speech tasks. Such measures would also

provide new insights into the effects of speech mode manipulation on the mechanisms of phonatory offset-onset control.

Other measures of voice and speech should be used in the further investigation of vocal fold dysfunction in the YOPD population. For example, acoustic measures of segment and pause durations, as well as fundamental frequency variation measures associated with prosody should be investigated in YOPD speakers. This information might identify dysfunction and mechanisms more specific to speaker intelligibility.

Perceptual corroboration of changes in speaker intelligibility associated with the speech mode manipulations in the present study would have been useful. It is not certain if impaired phonatory offset-onset control indeed affected speaker intelligibility. In the present study, HC speakers, in addition to the YOPD speakers, produced a lower occurrence of completely laryngealized phonatory offset-onsets in comparison to the number of complete aspiration phonatory offset-onset gestures. Umeda (1978) noted that the use of complete glottal stops varied across three healthy speakers. Interestingly, listeners judged those speakers who used frequent glottal stops to mark word boundaries as having greater fluency and clarity in comparison with speakers who produced fewer glottal stops.

Conclusion

This is the first comprehensive study of vocal dysfunction in an exclusively young-onset group of PD speakers, not contaminated by aging effects. Pending replication of this preliminary study with a larger sample size, the following conclusions are offered:

1. Unlike HC speakers, YOPD speakers reported voice symptoms of reduced loudness, tremor, hoarseness, and monotone, as well as decreased speech intelligibility. As anecdotally reported, these symptoms may in some persons precede the onset of limb symptoms. These voice symptoms have significant psychosocial consequences for persons with YOPD that place emotional, functional, and physical restrictions on their daily lives. Regardless of aging effects, PD is associated with perceived vocal dysfunction.

2. YOPD speakers did not demonstrate deficits during sustained phonation with respect to mean Fo, speech intensity level, and voice spectra measures related to voice quality parameters of roughness, hoarseness, and harshness. Both speaker groups demonstrated evidence of breathiness as measured acoustically by the soft phonation index (spectral tilt).

3. YOPD speakers demonstrated a slower syllable repetition rate for the adductory but not abductory L-DDK task, suggesting either impairment or use of a compensatory mechanism for producing adductory phonatory offset-onset. Also, a greater number of breath pauses were observed in the YOPD speakers for the abductory L-DDK gesture, suggesting inefficient valving of the pulmonary airflow through the glottis that resulted in a rapid depletion of expiratory airflow.

4. Unlike HC speakers, YOPD speakers appear to show deficits in phonatory offset-onset mechanisms for the control of both the laryngealization and aspiration gestures associated with an open vocal tract. However, consistent phonatory offset-onset control was observed for the aspiration gesture associated with an oral constriction, suggesting that the effect of intraoral air pressure as a passive mechanism of phonatory offset-onset control was intact.

5. Voicing control appears to be impaired in YOPD speakers for both mechanisms of laryngealization and aspiration not associated with oral constriction. However, voicing control

is not impaired in phonetic contexts associated with oral constriction where the effects of oral air pressure provide a passive mechanism of devoicing.

6. Intra-speaker group variability was observed for YOPD speakers. Five speakers were clearly impaired with respect to voicing control, and they accounted for a large proportion of spectrographic pattern codes of partial or no phonatory offset-onset. Six speakers demonstrated near normal or mild impairment for some gestures. One speaker, who was a professional voice user, demonstrated superior phonatory offset-onset control for the laryngealization gesture, as compared with the mean and 95% confidence interval for the HC speaker group. However, this speaker reported a moderate voice handicap related to vocal symptoms of hoarseness, monotone, and decreased speech intelligibility. The presence and degree of vocal dysfunction varies in the YOPD population.

7. Experimental manipulation of speaking mode suggested that YOPD speakers use a habitual postural setting of the larynx that is similar to that used by HC speakers when speaking in a breathy or aspirant mode. Speech masking facilitated an

effective speaking mode change in YOPD speakers, suggesting that it might be a useful behavioral treatment technique for promoting a projected voice mode in YOPD speakers.

8. Persons with YOPD are often unaware of the psychosocial consequences of their vocal dysfunction. Therefore, they should be routinely screened for vocal dysfunction using simple, routine questionnaires (VAS and VHI) and a clinical task of L-DDK.

Appendices

Appendix	Title
A	Interview: Participant Selection Criteria for Speakers
B	Advertisement to Recruit Participants
C	Consent Form for Speakers
D	Summary of Clinical Tasks and Dependent Measures
E	Summary of Dependent Measures for Sentence Stimuli
F	Counterbalancing of Speaking Mode Conditions and Fairy Tale
G	Fairy Tale
H	Mean and 95% confidence interval for the depression, dementia, and dysarthria severity rating scores for both speaker groups
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J	Summary of Auditory Masking Noise Threshold and Masking Level (dB SPL) Use for YOPD and HC Speakers

Appendix A

INTERVIEW: PARTICIPANT SELECTION CRITERIA FOR SPEAKERS

NAME: AGE: GENDER: RACE:

FOR ALL SPEAKERS:

CRITERIA	YES	NO
Smoked within last 5 years		*
Major surgery of chest, head, or neck		*
Hx of speech and/or voice problems (unrelated to YOPD)		*
Hx of respiratory or laryngeal disorders (unrelated to YOPD)		*
Hx of language or learning problems		*
Bilateral hearing sensitivity poorer than ➢ 25 db for frequencies .5 kHz, 1 kHz, 2 kHz ➢ 30 db for 4 kHz		*
Hx of substance abuse		*
Native speaker of American English	*	
Significant depression ➢ Hamilton Depression Score < 18		*
Normal cognition ➢ Score > 27 on Mini-Mental Status Exam.	*	
Completed high school	*	

FOR YOPD SPEAKERS:

CRITERIA	YES	NO
Neurologic disease other than PD		*
Onset of PD prior to age 21 or after 49 years		*
Moderate or severe dysarthria ➢ Dysarthria Severity Rating Score < 8		*
Articulatory disturbance		*
Moderate disturbance in speech rhythm or rate		*
Stages I – III (Hoehn & Yahr, 1967)	*	
Native speaker of American English	*	

FOR HC SPEAKERS:		
Clinician-judged as soft-spoken		*
Neurologic disease		*

*criteria met for inclusion in study

INTERVIEW QUESTIONS

- Do you every have any problems with your voice? Describe.
  - Soft
  - Breathy
  - Hoarse
- Have you ever had any voice problems (prior to onset of PD)?
- Is your voice today representative of it's typical nature? (better, same, worse)
- What voice requirements do you have for your
  - Job
  - At home
  - Hobbies, social activities
- Have you noticed any changes in
  - Pitch
  - Pitch range
  - Ability of raise your loudness
  - Strain or effort to talk, vocal fatigue
- Are your voice symptoms variable or consistent throughout the day?
  - Time of day
  - Particular speaking situations
- Does your voice tire out more easily now (than before PD)
- Do you have any problems swallowing
  - Liquids
  - Solids
- Do you have any problems with drooling?
- Do you ever experience symptoms of
  - Heartburn
  - Lump in your throat
  - Pain in your throat, particularly in the morning
- Currently, do you consider yourself to be a soft-spoken person
  - Do others
  - If so, were you always soft-spoken or did become evident at the onset of PD
- Have you had any surgery to your chest, neck, or head?
- Do you have any neurological problem (other than PD)?
- Did you ever have speech therapy?
- Have you ever experienced disfluent speech or stuttering?
- Do others notice that your speech is ever slurred?
- Have you smoked any cigarettes within the past five years?
- Do you have any problems with your hearing?
- Do you have any respiratory or breathing problems?
- Do you have any allergies? If so, how are they treated?
- Did you first learn to speak American English?
- Do you currently have any medical problems other than PD?
- Have you ever had a major hospitalization? For what?
- What medications are you currently taking and for what reason?

Appendix B

Advertisement to Recruit Participants

- ✓ Did you first notice your symptoms of Parkinson's disease between the ages of 21 and 55 years of age?
- ✓ Are you age 63 or younger?

THEN YOUR HELP IS NEEDED IN A STUDY OF SPEECH PROBLEMS ASSOCIATED WITH YOUNG ONSET PD!

- All of the currently published studies of voice and speech problems associated with Parkinson's disease (PD) and the treatment techniques that have been developed are based on research that has used primarily older-onset persons.
- There are no published studies that have investigated the voice and speech problems associated with persons who have young-onset PD.
- There may be a need to develop additional treatment techniques that are better suited for people who have experienced a younger-onset of this disease.

May I phone you about asking for your assistance? Name:_____
Phone: _____

Would you please volunteer to have your speech recorded?

- The purpose of this study is to identify the voice and speech symptoms associated with idiopathic Parkinson's disease in persons who first noticed parkinsonian symptoms or signs between the ages of 21 to 55 years.
- This is the first study that will help to understand the specific problems that are associated with young-onset PD.
- The results of this study will help to develop further research aimed at identifying effective, efficient voice treatment techniques for the young-onset population.
- This study is being conducted by Celia Bassich, as a part of her doctoral dissertation at the University of Maryland College Park (UMCP). Ms. Bassich is also a licensed speech pathologist, LSVT™ certified, and is a full-time Assistant Professor at Towson University. The Institutional Review Board at UMCP (IRB No. 01127) and the Institutional Review Board at Towson University (IRB No. 02-A079) have approved this study.
- This study **does not** involve the use of drugs or the use any difficult or painful procedures. Participants are asked to attend one session, either at Towson University or UMCP. Sessions are scheduled at a time that is convenient for you, including Saturdays or evenings. Free parking is available at both locations. You will be asked to read a five-minute fairy tale that was developed by the investigator to study your voice characteristics while reading aloud under 3 different speaking conditions. The speaking conditions are using your conversational voice, using a more quiet voice, and reading aloud while listening to some background noise. You will also be asked to complete questionnaires that include items concerned with your overall health, voice and speech characteristics, mental status, and depression. **All information collected in this study is confidential. Your name will not be disclosed to anyone at any time.**

WHO DO I CONTACT?

- If you have further questions or would like to participate, you can contact **Celia Bassich** by either email or phone:
  - Email address is cbassich@towson.edu

Phone: **410 704-2437**

Appendix C

Department of Hearing and Speech Science
University of Maryland
College Park, Maryland 20742

Consent Form for Speakers (Revised June 2001)

Project Title	Acoustic and Perceptual Study of Vocal Dysfunction in Young-onset Parkinson's Disease
Purpose	The purpose of this project is to identify the voice symptoms associated with idiopathic Parkinson's disease in persons who developed parkinsonian symptoms or signs between 21 and 40 years. The results will be used to formulate a cohesive theory of vocal dysfunction in young-onset Parkinson's disease (YOPD). Such formulation will guide further research aimed at understanding the nature of the laryngeal deficits and to identify effective, efficient voice treatment techniques for this population.
Procedures	I understand that I will be asked to attend a two-hour session at the Voice and Fluency Lab, UMCP. My voice will be audio recorded while I say some phrases aloud and read a fairy tale three different times, speaking in my normal conversational voice, speaking in a hushed or quiet voice, and while listening to background noise. I will be given a hearing screening test, and I will complete questionnaires which include items related to my overall emotional and physical health, mental status (including depression), voice, and speech characteristics.
Confidentiality	I understand that all information collected in this study is confidential, and my name will not be disclosed at any time.
Risks	I understand there are no known risks associated with any procedures involved in this experiment. *The background noise I* will be given falls well below the minimum standards specified by Federal regulations and will not reach a level that is uncomfortable for me.

Freedom to withdraw and ask questions	I understand that the experiment is not designed to help me personally, but that the investigators hope to learn more about the vocal symptoms in people with young-onset Parkinson's disease. I understand that I am free to ask questions or to withdraw from participation at any time without penalty.
Name, address and phone number of principal investigator	Dr. Gerald N. McCall , Ph.D. Department of Hearing and Speech Sciences 0100 LeFrak Hall College Park, Maryland 20742
Name and phone number of graduate student investigator	Ms. Celia Bassich-Zeren, M.A., CCC-SLP 301 405-8362
Signature of Participant *Date*	_____ _____

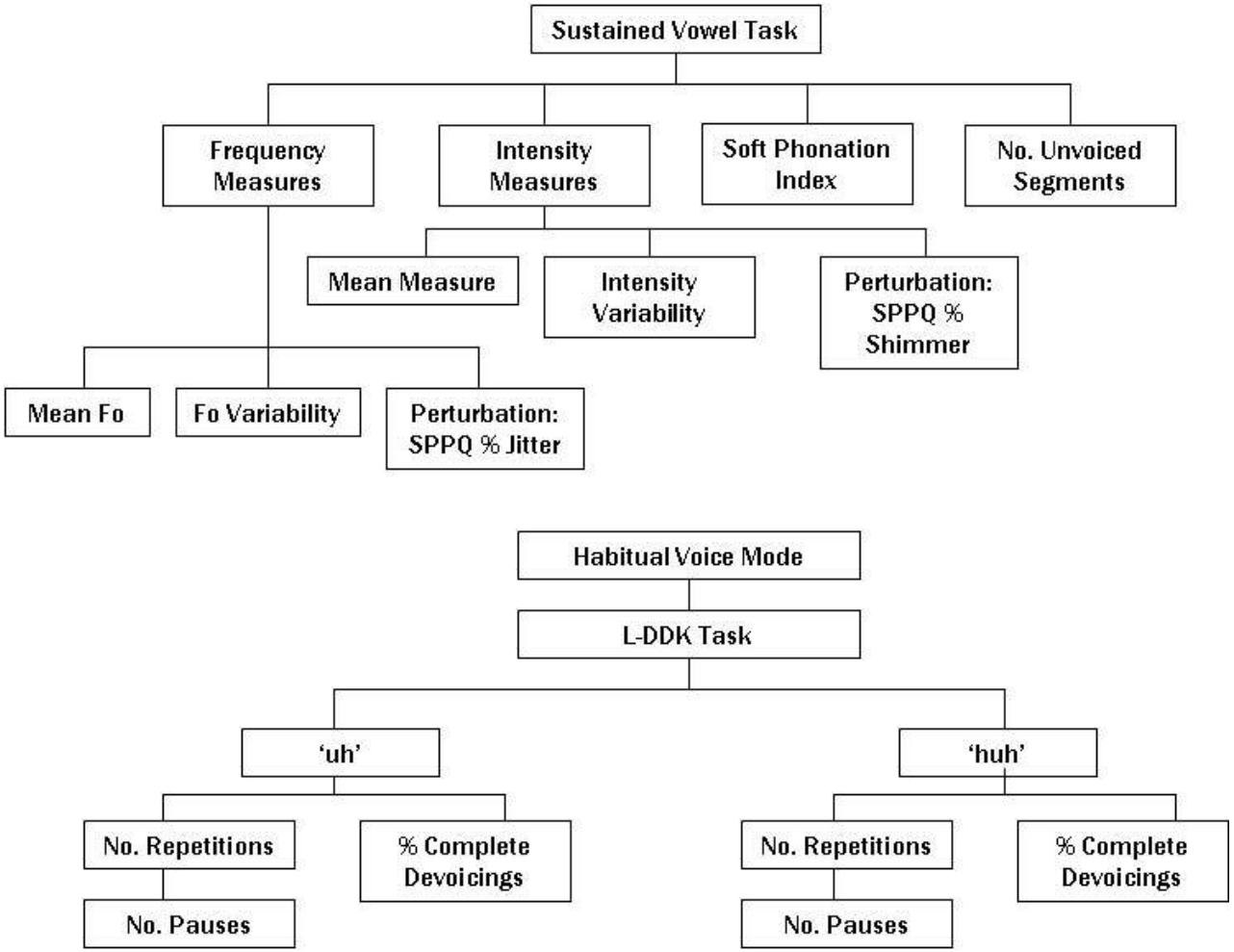
Attachment to Speaker Consent Forms for Speakers Tested At Towson University

This study was initially approved by the Institutional Review Board at University of Maryland, College Park (IRB No. 01127, June 26, 2001 and renewed July 1, 2002 to 31, 2003). In order to provide a more convenient testing location for participants who live in the Baltimore area, the study has also been approved by the Institutional Review Board (02-A079) (Rooms 242, 244 Enrollment Services Building, Towson University, 8000 York Road, Towson, MD 21252). Both institutions at College Park and Towson have fully equipped facilities for conducting the testing for the speech recordings and listening experiments.

Participants may contact Dr. Pat Alt at the Towson University Institutional Review Board (410 704-4221) or the Principle Investigator of this study (Celia Bassich-Zeren, 410 704-2437) if they have any questions about the study.

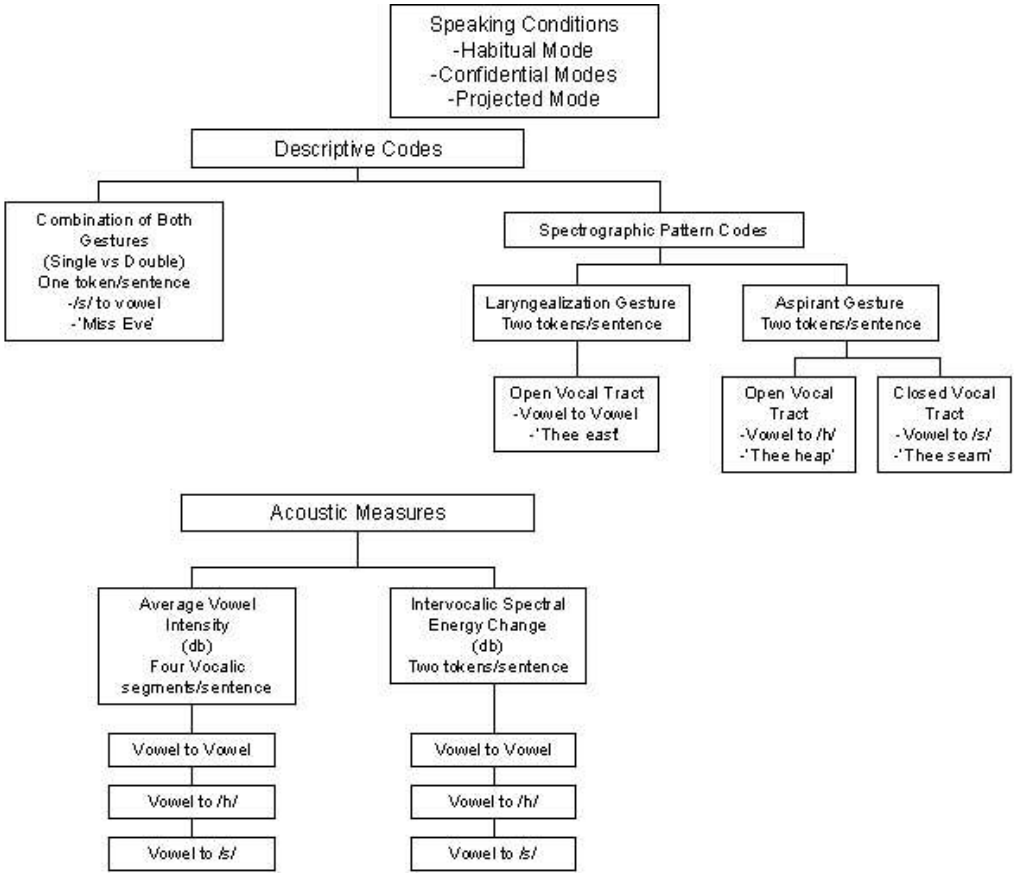
Appendix D

Summary of Dependent Measures for Clinical Tasks



Appendix E

Summary of Dependent Measures for Sentence Stimuli



Appendix F

Counterbalancing of speaking mode conditions and Fairy Tale Version (vowel order)

Speakers	First Condition	Second Condition	Third Condition
PD01, PD02 HC10, HC11	A	B	C
PD06, PD11 HC06, HC02	B	C	A
PD03. PD10 HC01, HC03	C	A	B
PD04, PD09 HC04, HC09	A	C	B
PD05, PD07 HC05, HC07	B	A	C
PD08, PD12 HC08, HC12	C	B	A

- A – Habitual speaking mode
- B – Confidential speaking mode
- C – Projected speaking mode

There were three versions of the fairy tale one for each speaking mode. The vowel order for each version was as follows:

- Version 1: /i/, /u/, /e/, /o/, /ae/, /a/
- Version 2: /a/, /ae/, /i/, /u/, /o/, /e/
- Version 3: /o/, /e/, /a/, /ae/, /u/, /i/
-

The versions of the fairy tale were randomized across speakers.

Appendix G

The Precise Words for the Water of Life
(Fairy -tale - version 1)

There was once a king who was so ill that it was thought impossible his life could be saved. He had three sons, and they were all in great distress on his account, and they went into the castle gardens and wept. An old man came up to them and asked the cause of their grief.
They told him that their father was ill, and nothing could save him.
The old man said, "There is only one remedy which is the Water of Life. If your father drinks of it he will recover, but it is very difficult to find because you must know the precise words."
The eldest Prince thought, "If I bring this water I shall be the favorite, and I shall inherit the kingdom!" So he asked the old man, "Tell me how to find it so I may save my father's life?"
The old man answered, "You must travel to a faraway land where there is a Troll guarding the Water. As you approach, the Troll will ask if you saw a young maiden in the meadow and you should nod your head. When he inquires as to what the maiden was doing, you must reply,

'Miss Eve was eating this eave.'

The Troll will then ask, "What dost thou foresee of me?" You must reply with these three predictions.

First, dear Troll, thee heap will be heeding thee heat.
Then, dear Troll, thee east will be eating thee eve.
And finally, dear Troll, thee seam will be seeking thee scene.

If you answer with these precise words then you shall have access to the Water of Life and can save your dear father's life."The Prince thought, "This is quite simple!" So he set off, and when he had ridden some distance he came upon a Troll standing in the road who cried, "Excuse me young man, did you see a maiden in the yonder meadow?" The Prince nodded and said,
"Dear Troll, please let me pass so that I may find the Water of Life and save my father's life.
The Troll inquired, "What was the fair maiden doing in the meadow?" The Prince thought hard but could not recall the old man's precise words. He decided to make up his own words thinking that the Troll was dull and would not know the difference." He replied,

"Miss Oot was oozing this oop."

The Troll then asked, "And what dost thou foresee of me? The Prince quickly recited,

"First, dear Troll, thee hoop will be hooting thee hoot.
Then, dear Troll, thee ooze will be oozing thee oot.
And finally, dear Troll, thee suit will be soothing thee soup."

The Troll reddened with anger, "You have tried to deceive me with your imprecise words and for this you shall be banished forever from the Water of Life!"
The Prince fell to his knees, and begged for mercy. "Please, dear Troll, I am weary and answered without thinking. Please give me another chance. Please, Sire, for the sake of my father's life!"

The Troll softened. "I shall give you one more chance. What was the maiden doing in the meadow?"

The Prince thought hard but could not recall the precise words. Attempting again to deceive the Troll he replied,

"Miss Aide was aching This ape."

The Troll raised his voice, "And what do you foresee of me?"

Continuing with his deceitful response, the Prince replied,

"First, dear Troll, thee hake will be hating thee Hague.
Then, dear Troll, thee aide will be aching thee ape.
And finally, dear Troll, thee safe will be saving thee sage.

Hearing a second deceitful response, the Troll was enraged and made an evil vow. Soon afterwards, the Prince came to a gorge in the mountains, and the farther he rode the narrower it became, until he could go no further. His horse could neither go forward nor turn around for him to dismount. So there he sat, jammed in the gorge.

The sick King waited a long time for him and when he failed to return, the second son saw this as an opportunity to inherit the kingdom and begged his father for permission to find the Water

of Life. The King reluctantly granted permission and warned him to remember the precise words for the Water of Life.

The second Prince started on the same road as his brother. He eventually came upon the same Troll, who stopped him and asked if he had seen a young maiden in the meadow. The second Prince nodded his head whereupon the Troll inquired, "What was the fair maiden doing in the meadow?" The Prince thought hard but could not remember the old man's precise words. Hoping to deceive the Troll he answered,

"Miss Ode was owning this oak."

The Troll then asked, "And what dost thou foresee of me?" Whereby the Prince replied,

"First, dear Troll, thee host will be hosing thee home.
Then, dear Troll, thee oath will be owning thee oak.
And finally, dear Troll, thee soap will be sewing thee soak."

The Troll reddened with anger and threatened to banish the Prince forever, but softened when the Prince fell begging to his knees and gave him a second chance. The Troll asked again,

"What was the maiden doing in the meadow?"

The Prince again tried to deceive the Troll and replied,

"Miss Att was adding this act."

The Troll asked loudly, "And what dost thou foresee of me?"

The Prince continued with his deceitful response,

"First, dear Troll, thee hat will be hashing thee hat.
Then, dear Troll, thee act will be adding thee ad.
And finally, dear Troll, thee sax will be sacking thee sash."

The Troll recognized his deceitful response and cast a spell over him. And the second Prince, too, got into a narrow gorge like his brother, where he could neither go backwards nor forwards. This is what happens to the deceitful.

As the second son also stayed away, the youngest one offered to go to find his brothers and to bring the Water of Life back to save his father. Reluctantly, the King granted permission but gave him a medal for protection, instructing him to wear it close to his heart.

When the youngest Prince met the Troll, he too could not recall the old man's precise words. Rather than deceive the Troll, he replied,

"I am searching for the Water of Life, because my father is dying. An old man has instructed me, but I fail to remember his precise words. I also wish to find my two brothers so they can return home to inherit my father's kingdom."

The Troll asked, "What words do you recall?"

The Prince replied, I know that you would inquire about a young maiden in the meadow, and I was to reply,

"Miss Odd was oxing this ox."

But those are not the precise words. Then, you would ask,

"What dost thou foresee of me? And I should answer,

"First, dear Troll, thee hop will be hopping thee hot.
Then, dear Troll, thee ox will be oxing thee odd.
And finally, dear Troll, thee sod will be sopping the sock.

But the precise words I have failed to recall.

"As you have spoken honestly to me, and not been deceitful like your brothers, I will help you to recall the precise words. Rub the medal that lies close to your heart and listen."

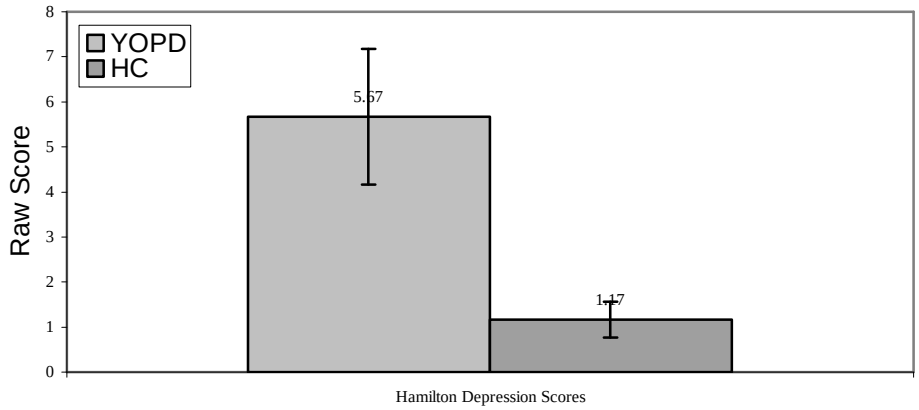
The young Prince did so at once and soon heard the old man's voice reciting the precise words. The Prince repeated them aloud. The Troll smiled and said,

"As you have recited the precise words I will lead you to the Water of Life. So you can save your father. But, as for your two brothers, they have met their fate because of their deceit and will never return home!

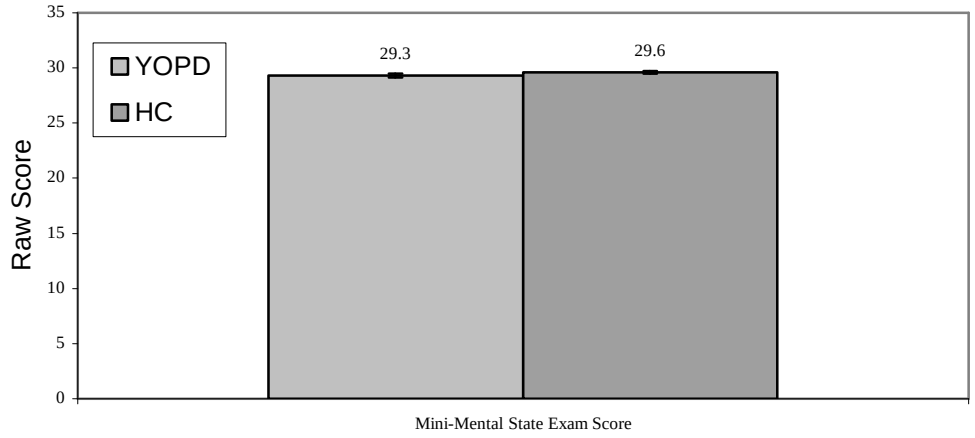
Appendix H

Mean and 95% confidence interval for the depression, dementia, and dysarthria severity rating scores for both speaker groups.

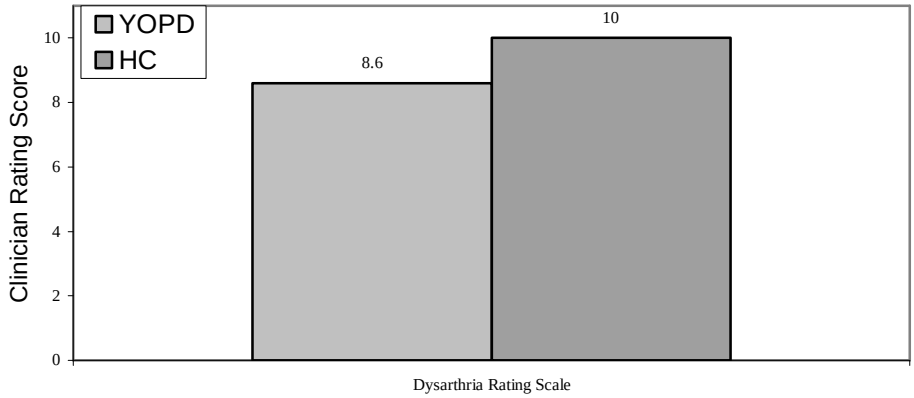
Mean and 95% confidence interval for the Hamilton Depression scores for YOPD and HC speakers



Mean and 95% confidence interval for the Mini-Mental State Examination scores for YOPD and HC speakers



Mean and 95% confidence interval for the Dysarthria Severity Rating for YOPD and HC speakers



Appendix I

Table I (1). Means and standard deviations for Visual Analog Scale measures for YOPD and HC speakers.

Voice Attributes

YOPD	Loudness	Shaky	Scratchy	Monotone
M	31.6	22.7	30.5	39.8
SD	21.1	19.8	16.9	30.6
HC				
M	11.7	8.7	11.7	8.6
SD	11.11	9.4	9.5	14.3

Speaker Intelligibility

YOPD	Slurs	Mumbles	Understandability
M	24.3	28.0	25.4
SD	16.1	22.5	20.8
HC			
M	9.5	11.2	10.5
SD	7.1	13.1	12.3

Conversational Pragmatics

YOPD	Participates	Initiates (Starts)
M	19.2	27.7
SD	23.7	22.7
HC		
M	7.8	18.3
SD	9.6	16.9

Table I (2). Means and standard deviations for the Voice Handicap Index measures for YOPD and HC speakers.

VHI Component Scores

YOPD	Total Score	Functional Score	Physical Score	Emotional Score
M	23.9	8.25	10.2	5.5
SD	17.4	6.8	5.1	6.5
HC				
M	6.6	2.1	3.7	0.8
SD	7.2	2.6	3.8	2.3

I (3). Means and standard deviations for the SF-12 Health Survey *z* score measures for YOPD and HC speakers.

YOPD	Physical Component	Mental Component
M	-0.37	-0.17
SD	1.00	1.12
HC		
M	0.39	0.48
SD	0.48	0.51

Table I (4). Means and standard deviations for the Multidimensional Voice Profile (MDVP) voice spectra measures for YOPD and HC speakers.

F_o and F_o Perturbation Measures

YOPD	Mean F_o (Hz)	F_o Variability (Hz)	Percent Jitter	Smoothed Pitch Perturbation Quotient
M	156.1	2.1	0.94	0.81
SD	46.9	0.8	0.52	0.3
HC				
M	135.2	1.6	1.1	0.96
SD	43.4	0.5	0.9	0.52

Intensity and Intensity Perturbation Measures

YOPD	Mean Intensity (dB)	Intensity Variability (dB)	Percent Shimmer	Smoothed Amplitude Perturbation Quotient
M	97.0	6.25	2.56	3.71
SD	3.8	2.2	1.14	1.88
HC				
M	96.7	8.2	2.01	3.04
SD	4.2	2.9	0.60	0.91

Soft Phonation Index and Unvoiced Segments

YOPD	Soft Phonation Index	No. of Unvoiced Segments
M	39.1	0.44
SD	21.2	0.03
HC		
M	35.6	0.03
SD	19.8	0.1

Table I (5). Means and standard deviations for the laryngeal diadochokinesis (L-DDK) measures for YOPD and HC speakers.

Number of syllable repetitions per second

YOPD	Adductory L-DDK	Abductory L-DDK
M	3.8	3.9
SD	0.6	0.2
HC		
M	4.4	4.1
SD	0.9	0.5

Percent of complete phonatory offset-onsets

YOPD	Adductory L-DDK	Abductory L-DDK
M	53.5	60.4
SD	40.9	35.0
HC		
M	65.3	70.8
SD	33.2	38.1

Number of pauses

YOPD	Adductory L-DDK	Abductory L-DDK
M	0	.67
SD	0	1.0
HC		
M	0	0
SD	0	0

Table I (6). Means and standard deviations for the average intensity level measure for three phonetic contexts and three speaking modes for YOPD and HC speakers.

V_V Phonetic Context

YOPD	Habitual Mode	Confidential Mode	Projected Mode
M	99.8	92.7	105.4
SD	4.1	3.4	1.7
HC			
M	97.9	93.7	104.8
SD	2.9	3.0	4.4

V_H Phonetic Context

YOPD	Habitual Mode	Confidential Mode	Projected Mode
M	99.8	93.1	105.9
SD	4.0	3.3	1.8
HC			
M	98.8	94.1	105.2
SD	3.5	2.9	4.3

V_S Phonetic Context

YOPD	Habitual Mode	Confidential Mode	Projected Mode
M	99.5	92.5	105.5
SD	3.7	3.5	1.7
HC			
M	97.9	93.7	104.6
SD	2.7	2.9	4.2

Table I (7). Means and standard deviations for the intervocalic spectral energy change measure for three phonetic contexts and three speaking modes for YOPD and HC speakers.

V_V Phonetic Context

YOPD	Habitual Mode	Confidential Mode	Projected Mode
M	35.9	34.9	30.0
SD	14.4	11.2	14.7
HC			
M	40.8	39.5	38.8
SD	9.3	5.7	11.0

V_H Phonetic Context

YOPD	Habitual Mode	Confidential Mode	Projected Mode
M	20.6	24.6	16.3
SD	6.2	5.3	5.6
HC			
M	26.3	28.0	19.7
SD	2.6	3.3	5.6

V_S Phonetic Context

YOPD	Habitual Mode	Confidential Mode	Projected Mode
M	23.3	25.8	21.5
SD	3.7	4.0	2.9
HC			
M	24.8	26.2	23.0
SD	2.0	1.9	2.3

Appendix J

Table J. Summary of auditory masking noise threshold and masking level (dB SPL) used for YOPD and HC speakers.

Participant Pair	YOPD Noise Threshold (dB)	HC Noise Threshold (dB)	YOPD Masking Level (dB)	HC Masking Level (dB)
01	NN*	6	89	72
02	18	10	74	66
03	10	4	64	74
04	4	5	66	71
05	9	4	61	56
06	5	20	71	78
07	6	8	70	70
08	6	12	76	76
09	2	2	80	64
10	4	8	80	70
11	28	12	66	70
12	6	12	60	80
Mean	8.9	8.6	71.4	70.6
SD	7.6	5.0	8.7	6.5

*Not Noted

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