EFFECTS OF SPEECH THERAPY AND PHARMACOLOGIC AND SURGICAL TREATMENTS ON VOICE AND SPEECH IN PARKINSON’S DISEASE:
A REVIEW OF THE LITERATURE

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The purpose of this review was to examine the different treatment approaches for persons with Parkinson’s Disease (PD) and to examine the effects of these treatments on speech. Treatment methods reviewed include speech therapy, pharmacological, and surgical. Research from the 1950s through the 1970s had not demonstrated significant improvements following speech therapy. Recent research has shown 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. Pharmacological methods of treatment in isolation do not appear to significantly improve voice and speech function in PD across research studies. Surgical treatment methods including pallidotomy and deep brain stimulation may be significant treatment options which improve voice and speech function in some persons with PD. Possible explanations for the differential responses to treatment are discussed. Future studies should investigate the effects of combined treatment approaches. Perhaps the combination of pharmacological, surgical and speech treatment will prove superior to treatments combining pharmacological and surgical or pharmacological and speech therapy in improving the communication abilities of persons with PD. © 2000 by Elsevier Science Inc.

Educational Objectives: The reader will be able to (1) describe the major types of treatments for persons with Parkinson’s disease, (2) specify the effects of these treatments on voice and speech functions, and (3) specify possible explanations for differential responses to treatment.

KEY WORDS: Parkinson’s Disease; Hypokinetic dysarthria; Treatment effects; Voice and speech
INTRODUCTION
Parkinson’s Disease (PD) is a progressive disease resulting from a reduction in the release of dopamine (DA) within the striatum of the basal ganglia. PD affects 1 in 1000 of the world’s population, and symptoms usually appear in both men and women after the age of 50 years (Marsden, 1996). Nearly 1.5 million people in the United States are affected by PD each year and approximately 40,000 new patients are diagnosed every year (Krauss & Jankovic, 1996; Tapper, 1997).

The diagnosis of PD is based on symptoms including tremor, rigidity or stiffness, bradykinesia, akinesia, and postural abnormalities (Manyam, 1997; Marsden, 1996; Stern & Lees, 1990; Tanner & Goldman, 1996). There is often difficulty executing simultaneous or sequential motor programs, indicating impaired motor planning (Marsden, 1982). An example of akinesia in PD is the classic masked face, in which the person with PD appears expressionless and unresponsive.

The characteristics of idiopathic PD differ from those of persons with Parkinson plus syndromes (PPS) such as multiple systems atrophy (MSA) and Shy Drager syndrome. For example, vocal fold abductor paresis and phono-tary characteristics including laryngeal stridor, glottal fry, and excess hoarseness were found to differentiate persons with Shy-Drager syndrome and PD (Bassich, Ludlow, & Polinsky, 1984; Hanson, Ludlow, & Bassich, 1983). The response to treatment also differs depending upon etiology. Limb symptoms do not improve to the same extent with Levadopa (L-dopa) in MSA as they do in PD (Fetoni, Genitrini, Monza, Soliveri, Testa, Caraceni, & Girotti, 1997). Therefore, this review of the literature on the voice and speech effects of various treatment techniques will concentrate on idiopathic PD unless otherwise stated.

CHARACTERISTICS OF PARKINSONIAN DYSARTHRIA
PD can affect speech output. In a survey of 460 patients with PD or multiple sclerosis, 70% of the persons with PD indicated that their speech was impaired after the onset of PD (Hartelius & Svensson, 1994). The speech production problem resulting from PD is known as Parkinsonian dysarthria, or hypokinetic dysarthria. Characteristics of hypokinetic dysarthria include: monotonous and reduced pitch and loudness, variable rate, short rushes of speech, imprecise consonants, and a breathy and harsh voice (Canter, 1963, 1965a, 1965b; Darley, Aronson, & Brown, 1969a, 1969b). Each of the speech production subsystems, respiration, phonation, articulation, resonance, and prosody may be affected in hypokinetic dysarthria (Swigert, 1997). The particular degree of impairment of each of these speech subsystems has a direct effect on how persons with PD convey their meaning during communication.

The respiratory system is affected in PD. During speech breathing, persons with PD have smaller rib cage volumes and larger abdominal volumes when
initiating breath groups, suggesting inadequate amounts of air reach the vocal tract during speech (Solomon & Hixon, 1993). Some persons with PD have lower oral pressures during CVC productions (Netsell, Daniel, & Celesia, 1975; Solomon & Hixon, 1993). Their ability to sustain prolonged vowel phonation has been reported to be impaired (Boshes, 1966; Canter, 1965a; Mueller, 1971). However, Fox and Ramig (1997) reported that there was no statistically significant difference in the vowel prolongation time of participants with PD and healthy comparison participants.

Persons with PD also have variations in speaking rates compared to control speakers (Boshes, 1966; Canter, 1963). Both faster than control speaking rates and slower than control speaking rates have been observed in individual persons with PD (Canter, 1963; Metter & Hanson, 1986). Such a wide range of speaking rates often makes group comparisons statistically non-significant, another frequent finding (Caligiuri, 1989; Ludlow, Connor, & Bassich, 1987; Pitcairn, Clemie, Gray, & Pentland, 1990).

Some of the discrepancies observed in these studies can be attributed to the particular speech samples used. Several studies have noted differences in the performance of persons with PD dependent upon the type of task used to assess impairments (e.g., Connor & Abbs, 1991; Ho, Bradshaw, Cunnington, Phillips, & Iansek, 1998). There may be a difference in speaking rate for persons with PD based on whether they are reading or whether they have to generate speech as in conversation or picture description (Schulz, Greer, & Friedman, 1998). Furthermore, speaking rate measures may differ depending on whether the measure includes pauses. Several studies have found persons with PD produce fewer words, spend less time producing speech per breath group, have a more rapid speech rate between pauses, and/or have longer and more frequent pauses (Hammen & Yorkston, 1996; Metter & Hanson, 1986; Pitcairn et al., 1990; Solomon & Hixon, 1993). These phenomena, in conjunction with articulatory undershoot, may give rise to the perception of “acceleration of speech” (Netsell et al., 1975). Although most research has demonstrated longer pause durations in persons with PD, some studies have not found a difference in pause durations between persons with PD and control participants (Canter, 1963; Volkmann, Hefter, Lange, & Freund, 1992).

Hypokinetic dysarthria also affects the phonatory system. The majority of persons with PD experience reduced vocal loudness. For example, vocal sound pressure level (SPL) in persons with PD has been shown to be statistically significantly lower (by 2.0–4.0 dB SPL) during speech and voice tasks as compared to age-matched control subjects (Fox & Ramig, 1997). In an analysis of the speech of persons with PD before undergoing surgical intervention, over half had diminished vocal loudness, with voices ranging from weakly to barely audible (Buck & Cooper, 1956). Persons with PD also exhibit difficulty achieving low-intensity phonation without going to the level of a whisper (Boshes, 1966; Canter 1965a), and they have a reduced ability to
produce a loud voice when asked (Canter, 1965a). Interestingly, persons with PD can increase their vocal volume when conversing with persons who are greater distances from them but do so to a lesser degree than persons without PD (Ho, Iansek, & Bradshaw, 1999). This suggests that volume regulation may be reflexively intact in persons with PD but that they are still unable to voluntarily control the “gain” of their speech.

Laryngeal abnormalities in the form of bowed vocal cords and an abnormally large glottic aperture during phonation have been found to accompany those with PD (Hanson, Gerratt, & Ward, 1984; Smith, Ramig, Dromey, Perez, & Samandari, 1995). This results in incomplete approximation of the vocal cords, thus decreasing vocal loudness. Laryngeal dysfunction in the form of breathiness, hoarseness, roughness, and tremulousness often occurs in hypokinetic dysarthria (Logemann, Fisher, Boshes, & Blonsky, 1978). Laryngeal tremor and abnormalities in vocal fold phase closure and phase symmetry were frequently observed in persons with PD (Perez, Ramig, Smith, & Dromey, 1996) which may account for the perceptual findings of Logemann et al. (1978). Reduced amplitude and firing rates of one of the major vocal fold adductor muscles, the thyroartenoid, have been reported (Baker, Ramig, Luschei, & Smith, 1998; Luschei, Ramig, Baker, & Smith, 1999) which may also contribute to reduced vocal loudness frequently accompanying PD.

Buck and Cooper (1956) reported that some patients had equally affected phonation and articulation. Imprecise articulation in hypokinetic dysarthria often affects the stop consonants (Ackermann & Ziegler, 1991; Canter, 1965b; Forrest, Weismer, & Turner, 1989), but it has also been reported that 45% of persons with PD exhibit misarticulations affecting affricates and fricatives (Logemann & Fisher, 1981). One manifestation of the imprecise articulation is the impaired ability of persons with PD to perform diadochokinetic tasks such as rapid movements of the lips, tongue tip, and back of the tongue required for /pa/, /ta/, /ka/, and /pataka/ repetitions (Canter, 1965b; Connor, Ludlow, & Schulz, 1989; Hirose, Kiritani, Ushijima, Yoshioka, & Sawashima, 1981).

The resonatory system may also be affected by hypokinetic dysarthria. For instance, Logemann et al. (1978) reported that some of their subjects exhibited hypernasality. An examination of the resonatory system of persons with PD revealed that some persons with PD are perceived as hypernasal due to inadequate velopharyngeal closure (Hoodin & Gilbert, 1989). Although hypernasality has been perceived in some persons with PD, it is not considered a hallmark of the speech disorder.

Hypokinetic dysarthria also affects the prosodic aspects of speech. Prosody of speech can be defined as “... the patterned distribution of stress, intonation, and other phonatory features in speech” [(Scott, Caird, & Williams, 1985), p. 13]. Characteristics of the prosodic aspects of hypokinetic dysarthria include monoloudness, reduction of stress, and monopitch.
1965a) documented significantly higher pitch levels and reduced pitch range for persons with PD. In a study of the production and perception of speech prosody, persons with PD were found to be poorer than controls in identifying and producing angry and questioning statements (Scott, Caird, & Williams, 1984).

The patterns of hypokinetic dysarthria that exist in persons with PD are highly variable. These variations may be dependent upon disease severity, dysarthria severity, task type, co-existing conditions, and/or specific neurological substrate affected. There is a general belief that hypokinetic dysarthria severity increases with increased duration of PD and/or with increased severity of limb symptoms. However, there appears to be little correspondence between dysarthria severity and duration of PD nor severity of limb symptoms (Gamboa, Jimenez-Jimenez, Nieto, Montojo, Ortí-Pareja, Molina, García-Albea, & Cobeta, 1997; Metter & Hanson, 1986; Schulz, Greer, & Friedman, in press).

The existence of other co-occurring symptoms such as dementia and/or depression may also account for the variability observed in the speech of persons with PD. It is now recognized that there is a significant subgroup of persons with PD who may also have dementia and the prevalence of dementia in PD is approximately 15–32% (Brown & Marsden, 1984, Wallin, Jennersjo, & Granerus, 1999). This subgroup may not have been differentiated in some studies and thus could have accounted for variations in patterns of hypokinetic dysarthria. In addition, a significant number of persons with PD also suffer from depression and the degree of depression has not been found to correlate with PD severity (Poewe & Luginger, 1999). The presence of depression and/or dementia has now been recognized as possible confounding factors that may affect speech production measures as evidenced by exclusion criteria in several recent studies [e.g. (Ramig, Countryman, Thompson, & Horii, 1995a; Schulz, Peterson, Sapienza, Greer, & Friedman, 1999)].

Another possible explanation for the hypokinetic dysarthria variations and the lack of correspondence between limb and speech symptoms might be differences in the underlying neuropathophysiology of the disease across individuals and systems. For example, the basal ganglia are somatopically organized into separate “leg,” “arm,” and “face” regions in a similar manner as that of the primary sensory and motor cortices (Alexander, Crutcher, & DeLong, 1990; Alexander, DeLong, & Strick, 1986). These areas could well be differentially affected in individuals with PD and thus give rise to differences observed between limb and speech symptoms. In addition, depletion of DA may be differentially affected across various basal ganglia-thalamus-cortex motor loops thus giving rise to differences observed within limb and speech motor systems.

Clearly, studies employing larger sample sizes with participants who exhibit a wide range of speech and limb symptoms with well-controlled inclu-
sion criteria are needed to aid in defining similarities and differences in the patterns of hypokinetic dysarthria. As will become apparent, there are discrepancies in the literature regarding the efficacy of different forms of treatment for the speech system in persons with PD. Such differences in treatment response may also be attributable to differences such as dysarthria severity, task used to assess treatment, co-occurring conditions, and/or specific neurological substrate affected as discussed above.

TREATMENT APPROACHES FOR PARKINSON’S DISEASE

The purpose of this study was to examine how different treatment approaches affect the voice and speech of persons with PD. Treatment methods include speech therapy, pharmacologic, and/or surgical. Recent speech therapy methods include treatments that utilize various therapeutic devices, treatment that targets prosodic aspects of speech and the Lee Silverman Voice Treatment (LSVT). Pharmacological treatment methods include those that replace DA and DA agonists; surgical treatment methods include thalamotomy, pallidotomy, fetal cell transplantation (FCT), and deep brain stimulation (DBS).

SPEECH THERAPY IN PARKINSON’S DISEASE

Between the 1950s and 1970s, there was not much confidence in the effectiveness of speech therapy for persons with PD. Morley (1955) suggested that the goal of rehabilitating the speech mechanism in dysarthric adults was to regain or obtain effective movements of the articulators used for speaking. However, he felt that this approach did not apply to individuals with paralysis agitans (PD) since it is a progressive and irreversible disease (Morley, 1955). Sarno (1968) also believed that individuals with PD did not improve with treatment. In observing the treatment of over 300 persons with PD who had various speech treatments (controlling intensity, improving articulatory mobility, improving articulation), Sarno concluded that “Often they [the patients] are impressively improved during the treatment session only to revert to the pathologic patterns immediately after” [(Sarno, 1968), p. 274)]. Furthermore, some believed that because persons with PD’ speech continually deteriorates, PD patients would always need treatment (Allan, 1970).

Some of the recent research has focused on the use of therapeutic devices for treating those with hypokinetic dysarthria. These devices include a voice amplifier, delayed auditory feedback (DAF), a wearable intensity biofeedback device, and a masking device. Voice amplification increases vocal loudness thus relieving any anxiety a person with PD may have if he or she was previously not audible to others. Another benefit of voice amplification was to increase self-monitoring of speech intensity (Greene & Watson, 1968). Intelligibility was not directly assessed, although the authors stated that intelligibility
was improved. Instead of focusing on one aspect of speech production, the goal of the DAF device was to improve the patients’ overall intelligibility (Downie, Low, & Lindsay, 1981). Two out of 11 subjects showed marked improvement in speech intelligibility. These two subjects demonstrated a “festinating speech difficulty” [(Downie et al., 1981), p. 852]. One of these patients continued to wear the DAF device for two years and still showed improved speech intelligibility. In a three-month study of two persons with PD, increases in relative vocal loudness and fundamental frequency as well as a marked reduction in speech rate were reported with use of the DAF device (Hanson & Metter, 1983).

In a case study, a microcomputer-based wearable biofeedback device was used to generalize a patient’s vocal loudness outside the clinic (Rubow & Swift, 1985). The device provided the person with PD with information about intensity that was usually only available during treatment in the clinic. Acoustic and perceptual analyses were performed on his voice pre- and post-treatment, showing generalization of clinic improvement to his daily life while wearing the device. This suggests that biofeedback, or behavioral modification, is effective in treating individuals with PD as long as the device is worn.

The masking device has been used to improve persons with PD’ vocal loudness (Adams & Lang, 1992). This device is based on a phenomenon known as the “Lombard effect,” in which most individuals increase their vocal loudness when speaking in the presence of masking noise. A significant increase in vocal loudness was demonstrated in 10 out of 10 persons with PD under the masking condition compared to speaking without masking noise. There was no report of the results of a follow up evaluation or whether this vocal loudness level generalized outside of the clinical setting. These results further indicate that persons with PD can reflexively increase their vocal intensity, as was demonstrated by Ho et al. (1999) but that under “normal” conditions, they cannot voluntarily control the “gain” in their volume.

Today, many individuals with reduced vocal loudness resulting from PD seek speech therapy from a certified speech-language pathologist. In the 1980s, speech therapy for persons with PD was initially centered on the prosodic aspects of speech, sometimes including other areas of speech (i.e., respiration, articulation, etc.); today it focuses on the voice itself (i.e., vocal loudness). The research on speech therapy during the 1980s and 1990s refutes the pessimism of Morley (1955), Sarno (1968), and Allan (1970).

One early report of successful speech therapy techniques targeted respiratory exercises (Erb, 1973). Three persons with PD were given several breathing exercises along with oral speech and non-speech exercises for 30 minutes three times per week. All three were subjectively said to improve in intelligibility but improvement was noted to be inconsistent over time. Prosodic exercises were initially used in the realm of speech therapy for persons with PD (Scott & Caird, 1983). Scott and Caird (1983) examined the effects of pro-
sodic exercises as well as their visual reinforcement via a Vocalite machine. Subjects significantly improved speech intelligibility and prosody for up to three months after treatment. The visual reinforcement device, however, appeared to only greatly benefit those with the most severe speech disorder. The long-term effects of intensive speech therapy focusing on prosody were also examined (Robertson & Thomson, 1984). Therapy targeted respiration, pitch variation, vocal loudness, articulation, strength and speed of the articulators, rate of speech, intonation and stress patterns, and communication intelligibility. Results revealed improvement in almost every aspect of speech (respiration, phonation, intelligibility, prosodic aspects of stress, intonation, and rate), as well as the ability to maintain these improvements for up to three months following the intensive treatment.

The effects of less intensive speech therapy on persons with PD were also examined (Johnson & Pring, 1990). In lieu of giving therapy to each patient for 35–40 hours over 2 weeks (Robertson & Thomson, 1984), 10 hours of treatment was given over 4 weeks. Therapy focused on improving two prosodic aspects of speech: vocal loudness and pitch, during spontaneous speech and reading tasks. Patients demonstrated objective improvement in these areas, indicating that therapy was beneficial. No follow-up measures were noted. Three prosodic aspects of speech in a woman with Parkinsonian dysarthria were also examined (Le Dorze, Dionne, Ryalls, Julien, & Ouellet, 1992). These aspects included linguistic modulation of fundamental frequency, mean fundamental frequency, and rate of speech. The results of the study revealed that the patient benefited from speech therapy in that she had more normal prosody and greater speech intelligibility. It was also noted that the patient’s improvement was maintained 10 weeks post-treatment.

As stated previously, the current primary focus in speech therapy for individuals with hypokinetic dysarthria is vocal loudness. The most recent and efficacious therapy of this type is known as the Lee Silverman Voice Treatment (LSVT), which was developed by Ramig et al. (Ramig, Bonitati, Lemke, & Horii, 1994). The LSVT focuses on increasing both respiratory effort and vocal fold adduction. The combination of these entities is necessary in order to increase vocal loudness in individuals with PD (Ramig & Dromey, 1996). The five essential concepts of the LSVT include focusing on loudness, using increased effort, having an intensive treatment regimen, calibration, or knowing and accepting the amount of effort needed to increase vocal loudness consistently, and quantification, or measuring the patient’s performance to increase motivation (Ramig, Pawlas, & Countryman, 1995b). Patients are given vocal loudness exercises such as maximum duration of sustained vowel phonation, maximum fundamental frequency range, and maximum functional speech loudness drill (Ramig et al., 1995b). The LSVT has been proven to be a successful long-term (12 months post-treatment) form of treatment in large numbers of participants (45) for increasing vocal loudness when compared to ther-
apy targeting respiratory effort only (Ramig, Countryman, O’Brien, Hoehn, & Thompson, 1996; Ramig, Countryman, Thompson, Horii, 1995a). Not only does LSVT improve vocal loudness, but it also decreases the negative impact of PD on communication (Ramig et al., 1995a). The LSVT also has a positive impact on intelligibility, pitch variability, phonatory stability, rate, and vocal fold adduction (Dromey, Ramig, & Johnson, 1995; Ramig et al., 1995a; Smith et al., 1995).

Although speech therapy may not have been effective in the past, current speech therapy techniques have proven efficacious in contributing to greater speech intelligibility in persons with PD. The successful speech therapy treatments, it should be noted, were performed on persons with PD who were also receiving pharmacologic treatment(s). For example, the participants in the LSVT studies were reported to be optimally medicated with dopamine replacement medications (L-dopa).

**PHARMACOLOGICAL TREATMENT METHODS**

Many different drugs have been developed to treat PD. Some of these medications enhance (DA agonists) or replace (L-dopa) the DA that is no longer present in the brain of persons with PD. Adjunctive treatments to these medications include: anticholinergic agents, monoamine oxidase-B (MAO-B) inhibitors, and catechol-O-methyl transferase (COMT) inhibitors (Calne, 1994; Tolosa & Valldeoriola, 1994).

Anticholinergic agents are the oldest form of pharmacotherapy for PD. They act to reduce tremor by blocking the action of acetylcholine (ACh), which is predominant due to the deficiency of DA (Stern & Lees, 1990). The goal in turn is to create a balance between both neurotransmitters. Two common anticholinergics are trihexyphenidyl (Artane) and benztpine (Cogentin). Little improvement has been noted in the speech of persons with PD following the use of anticholinergics. When comparing Artane to a placebo, objectively, little significant effect on overall speech was found, but subjectively, maximum intensity speech range and speaking rate “... showed a tendency toward significant improvement” [(Brumlik, Canter, De La Torre, Mier, Petrovick, & Boshes, 1964), p. 431]. Little improvement in articulation followed the administration of anticholinergic agents (Critchley, 1981).

MAO-B inhibitors such as selegiline (Deprenyl) inhibit the degradation of DA and may prolong the antiParkinsonian action of L-dopa. COMT inhibitors such as tolcapone (Tasmar) also increase the length of time that L-dopa is effective (Jankovic & Marsden, 1993). Selegiline has been shown to improve speech in both subjective and objective measures of articulation and respiration (Shea, Drummond, Metzer, & Krueger, 1993). Articulatory improvements were noted in rate and range of oral motor diadochokinesis; respiratory improvements were noted in measures of vital capacity and words per exhala-
tion during speech reading (Shea et al., 1993). On the other hand, deprenyl was shown to have no consistent effect on acoustic measures of speech in persons with early PD who were not taking L-dopa (Stewart, Winfield, Hunt, Bressman, Fahn, Blitzer, & Brin, 1995).

L-dopa, first introduced in 1968, acts to replenish DA levels in the brain. Combining L-dopa with carbidopa produces Sinemet the principle medication for treating PD. Carbidopa prevents L-dopa from converting into DA before crossing the blood-brain barrier. Short-term side effects include dyskinetic and involuntary movements, orthostatic hypotension, and nausea (Marsden & Parkes, 1977). An effect termed the “on-off” phenomenon occurs after several years of DA replacement treatment. After years of L-dopa therapy, the motoric improvements of the “on” period begin to wane and become shorter in duration, and the person with PD becomes “disabled” due to the prolonged reappearance of Parkinsonian symptoms during the “off” period (Marsden & Parkes, 1977). Many patients become akinetic and experience postural instability during the “off” period. These “on-off” swings gradually become more rapid and violent, increasing the patient’s need for L-dopa. This may be due to the inability of L-dopa to convert to DA or the limited space available for DA storage and release by diminishing dopaminergic neurons (Rabey, 1995).

Findings appear to show a general trend in improvement of speech production through the use of L-dopa. Though speech improvement was not found to be as dramatic as limb symptoms, subjectively, “... there appeared to be a trend in the direction of improved speech during L-dopa therapy” [(Rigrodsky & Morrison, 1970), p. 142]. This trend was observed subjectively in spontaneous speech as well as in oral reading by evaluating overall speech adequacy, clarity of articulation, normalcy of nasal resonance, and temporal aspects of speech (rate, pauses, and rhythm). During the oral reading task, rate, pauses, and rhythm were observed as the most improved (Rigrodsky & Morrison, 1970). In another study, the speech of most persons with PD became more intelligible after L-dopa treatment, primarily as a result of improved vocal loudness (Mawdsley & Gamsu, 1971). Although the rate of speech did not show change in this study, the tendency for a more regular distribution of both speech time and pauses was noted after L-dopa treatment, thus increasing intelligibility (Mawdsley & Gamsu, 1971).

Physiological studies have concentrated on measuring labial kinematics and muscle recordings as a function of L-dopa medication. In a study of labial kinematics related to speech intelligibility, after the administration of L-dopa, labial movement tracings showed a shorter period of time between the initiation of labial movement and speech, and increased speed and symmetry of labial activity (Nakano, Zubick, & Tyler, 1973). Nakano et al. (1973) also reported that 16 out of 18 persons with PD subjectively felt that their speech had improved as compared with procyclidine or placebo. Labial pressures in speech and non-speech tasks also tended to improve following L-dopa admin-
istration (Cahill, Murdoch, Theodoros, Triggs, Charles, & Yao, 1998). In another study, electromyographic recordings of labial muscles revealed tonic hyperactivity before taking L-dopa, which decreased after taking this medication (Leanderson, Meyerson, & Persson, 1971). This suggests that L-dopa normalized the neuromotor control of labial muscular activity, which may have contributed to the subjective improvements observed in six out of seven patients’ hypokinetic dysarthria (Leanderson et al., 1971). Although labial rigidity may continue to decrease throughout the drug cycle, Caligiuri and Abbs (1986) found that movements of lips did not change in a parallel fashion. This may mean that while rigidity improved, reduced motor drive to labial muscles may still have been a factor preventing improvement in labial movement.

Short-term and long-term effects of L-dopa on speech production have also been examined. Short-term L-dopa therapy “has a favorable influence upon” Parkinsonian dysarthria in the form of improved voice quality, pitch variation, and articulation as measured subjectively [(Wolfe, Garvin, Bacon, & Waldrop, 1975), p. 277]. After four years of L-dopa treatment, subjective observations revealed that 75% of persons with PD either maintained or improved over their initial improvement in speech when comparing results to those variables examined in the short-term study (Wolfe et al., 1975).

Other and more recent studies have not shown a positive impact of L-dopa on speech production. For example, subjective improvement was not noted in speech following L-dopa therapy (Quaglieri & Celesia, 1977) and there have been reports from neurologists of “peak-dose dysphonia” noted (Critchley, 1976). More recent studies have shown no difference in acoustic measures in persons with PD in the “on” or “off” state. Persons with PD had lower intensity, lower variability of fundamental frequency and intensity, and greater degrees of whisperiness and harshness in both the “on” and “off” states as compared to healthy control subjects and these measures did not change between the “on” and “off” state (Daniels, Oates, Phyland, Feiglin, & Hughes, 1996). Additionally, Poluha, Teulings, and Brookshire (1998) demonstrated no significant change in acoustic measures of vowels in ten persons with PD across their L-dopa drug cycle. Likewise, Solomon and Hixon (1993) failed to find significant differences in speech breathing measures as a function of the L-dopa drug cycle. Finally, no differences in vocal stability measures (amplitude and frequency perturbation) nor in indirect vocal fold movement measures (electroglottograph) were demonstrated across changes in drug cycles in two persons with PD (Larson, Ramig, & Scherer, 1994).

Such discrepancies in voice and speech function reported following L-dopa treatment might be attributable to participant related differences across studies. For example, studies may differ in other medications that participants may have been taking and/or there may be differences in dysarthria severity of participants in various studies. Measures of vocal intensity (Schulz et al., in press) and speech duration (Schulz et al., 1998) have been found to differ as a
function of dysarthria severity. The studies reviewed did not account for possible differences in dysarthria severity.

Dopamine agonists enhance DA levels in the brain. These include apomorphine, bromocriptine (Parlodel), lisuride, pergolide (Permax), cabergoline, quinpirole, ropinirole (Requip), and pramipexole (Mirapex). DA agonists are often taken during the “off” periods to enhance the supply of L-dopa or to prolong the effect of DA (Tolosa & Valldeoriola, 1994). For example, as assessed by the Unified Parkinson Disease Rating Scale (UPDRS), Mirapex improved the motor function of persons with PD during “on” and “off” periods as well as decreased the time spent in and reduced the severity of “off” periods (Lieberman, Ranhosky, & Korts, 1997). DA agonists may also be used to delay the beginning of L-dopa therapy (Piccoli & Riuggeri, 1995). Although they all have different pharmacological properties, all DA agonists stimulate D2 receptors (Calne, 1994). D2 receptors are highly specialized DA receptors in the brain. DA agonists have been shown to improve motor symptoms in persons with PD; however, their effects on speech have not been reported.

**SURGICAL TREATMENT METHODS**

Surgical intervention is another form of treatment for individuals with PD. The following types of surgery have been performed in order to reduce Parkinsonian symptoms: thalamotomy, pallidotomy, transplantation (i.e., fetal cell), and deep brain stimulation.

**Thalamotomy**

The surgical procedure known as thalamotomy consists of lesioning the ventralis intermedius (VIM) of the ventrolateral thalamus (Grossman & Hamilton, 1993). This is accomplished with a technique known as stereotactic surgery, in which “... a thin probe is delicately inserted into the brain through a hole in the skull” [(Stern & Lees, 1990), p. 36]. This method has produced a significant reduction of contralateral tremor and rigidity for persons with PD (Tasker, Lang, & Lozano, 1997) and is used to treat severe drug-resistant Parkinsonian tremor (Tasker, Siqueira, & Hawrylyshyn, 1983).

Speech has not been shown to improve postoperatively after VIM thalamotomy, it has shown some evidence of deterioration after the procedure and as PD progresses (Tasker et al., 1983). Those persons with PD who had thalamotomy were more dysarthric than those who had not had surgery (Quaglieri & Celesia, 1977). Unilateral operations of the thalamus in the individual’s dominant hemisphere were more likely to produce speech disturbances such as dysarthria, monotonous voice, slow speech (Jenkins, 1968), and decreased vocal loudness and articulation difficulties (Allan, Turner, & Gadea-Ciria, 1966) than operations in the non-dominant hemisphere. Initiation of speech, mainte-
nance and control of speech, fluency, and vocal loudness were disturbed in VIM thalamotomy in either hemisphere (Petrovici, 1980), and diminished loudness, dysarthria, and dysphasia were present after lesioning the ventral lateral thalamus (Bell, 1968).

Bilateral thalamotomy is performed to relieve bilateral tremor and rigidity (Grossman & Hamilton, 1993). Speech problems resulting from bilateral thalamotomy include persistent worsening of dysarthria (Tasker et al., 1983), word blocking, slow speech, and hypophonia (Matsumoto, Asano, Baba, Miyamoto, & Ohmoto, 1976). Jenkins (1968) reported that persons with PD were less likely to have speech disturbances in bilateral operations whose less impaired side was performed first; Allan et al. (1966) reported that bilateral operations in which the left hemisphere lesion was performed before the right hemisphere lesion more commonly produced dysarthria than vice versa. L-dopa and carbidopa therapy failed to significantly improve speech of persons with PD who had recently undergone unilateral or bilateral thalamotomy (Quaglieri & Celesia, 1977).

**Posteroventral Pallidotomy**

The surgical procedure known as posteroventral pallidotomy (PVP) involves lesioning the globus pallidus internus (GPI) of the basal ganglia. Under normal conditions, DA is found in high concentrations in the corpus striatum. For persons with PD, DA input into the corpus striatum is depleted, resulting in overactivity of the GPI, which is inhibitory to the thalamus and brainstem (Eller & Dan, 1997). Lesioning the GPI thus causes the release of inhibition to the thalamic and brainstem motor centers. This lesion may improve all major Parkinsonian symptoms, including bradykinesia (Grossman & Hamilton, 1993; Laitinen, Bergenheim, & Hariz, 1992). Another benefit of PVP is that anti-Parkinsonian medications can be reduced. During the late stages PD, PVP significantly decreases L-dopa-induced movement disorders and “off”-periods (Lang, Lozano, Montgomery, Duff, Tasker, & Hutchinson, 1997).

Few investigators have examined the effects of PVP on Parkinsonian dysarthria. After undergoing neurosurgical intervention in the form of anterior choroidal occlusion or chemopallidectomy, the speech of persons with PD did not subjectively improve (Buck & Cooper, 1956). They suggested that “... improvement in speech should not be a prime goal in selecting patients for surgical treatment of Parkinsonism” (Buck & Cooper, 1956), p. 1290. One study showed that bilateral PVP resulted in similar speech ratings on the UPDRS as did unilateral pallidotomy (Iacono, Lonser, & Kuniyoshi, 1995).

More recently, Barlow et al. (Barlow, Iacono, Paseman, Biswas, & D’Antonio, 1998) reported on labial force production and stability and aerodynamics following bilateral PVP. They found that 45-55% of their 11 subjects had significantly reduced labial force instability and peak and average rate of la-
bial force recruitment during non-speech tasks. Additionally, some of their subjects exhibited translaryngeal airflow and intraoral pressures that were more like control subjects during consonant vowel consonant (CVC) syllable repetitions. Interestingly, the distribution of voice onset time (VOT) remained unchanged in one of these patients. They concluded that bilateral PVP might reflect a “. . . global rescaling of neural inputs or concomitant adjustments in muscle stiffness among muscle subsystems of the vocal tract” [(Barlow et al., 1998), p. 150].

Research is presently being conducted on the effects of voice and speech following PVP. Preliminary findings indicated that four out of six patients demonstrated positive changes in either phonatory or both phonatory and articulatory measures post unilateral PVP surgery (Schulz et al., 1999). Some subjects demonstrated greater intensity, more syllables per second, longer extended vowel duration, and longer syllable vowel duration post-surgery (Schulz et al., 1999). Analysis of the vocal intensity changes following unilateral PVP in a greater number of participants (25) with a greater range of hypokinetic severity has recently been completed (Schulz et al., in press). Results revealed that the greatest improvements in vocal intensity were observed in those participants who were rated as having mild hypokinetic dysarthria prior to surgery. Mildly dysarthric Parkinson’s patients may benefit most from unilateral PVP perhaps due to less overall destruction of the basal ganglia sensorimotor control circuits involved in oral facial functions, thus increasing the chances to observe improvements post-surgery. This would suggest that PVP be performed while speech symptoms are still relatively mild to derive the greatest benefit in vocal SPL. Although further research is necessary on this surgical procedure in order to determine the full effects on speech production, these studies demonstrate the potential beneficial effects of this treatment for persons with PD. In addition, if future studies follow the trends demonstrated by Schulz and her colleagues, beneficial PVP surgical effects may be dependent upon the severity of hypokinetic dysarthria.

Fetal Cell Transplantation (FCT)

Transplantation refers to the placement of fetal dopaminergic cells within the caudate or putamen of the basal ganglia of persons with PD. This surgical technique is still considered experimental. It is based on the theory that grafts of dopaminergic cells can survive and secrete DA into the striatum of individuals with PD. Fetal cell nigral graft survival was demonstrated by autopsy findings of graft survival and striatal innervation in humans (Olanow, Kordower, & Freeman, 1996). For this reason fetal nigral transplants are considered the “gold” standard donor tissue [(Kordower, Goetz, Freeman, & Olanow, 1997), p. 45]. Embryonic mesencephalic tissue transplants lead to the survival of dopaminergic neurons and improvements for the majority of the
recipients (Wenning, Odin, Morrish, Rehncrona, Widner, Brundin, Rothwell, Brown, Gustavii, Hagell, Jahanshahi, Sawle, Bjorklund, Brooks, Marsden, Quinn, & Lindvall, 1997). These improvements were observed in rigidity, hypokinesia, reduction of L-dopa dosage, and reduction of “off” time (Wenning et al., 1997). It also appears that variations of fetal graft volume have an impact on performance of persons with PD during motor tasks (Kopyov, Jacques, Lieberman, Duma, & Rogers, 1997). For instance, those who received higher volumes of fetal grafts had significantly improved UPDRS scores in all areas than those who received lower volumes (Kopyov et al., 1997). Improvements five years after implantation of fetal ventral mesencephalic tissue in the caudate nucleus included improved motor function and the persons with PD had less severe and more “on” time with less intense and shorter periods of dyskinesia (Hauser, Freeman, Snow, Nauert, Gauger, Kordower & Olanow, 1999; Lopez-Lozano, Bravo, Brera, Millan, Dargallo, Salmean, Uría, & Insauti, 1997).

Since this technique is still new and experimental, its effects on speech production have just begun to be investigated. The effects of FCT on speech and voice production suggested that FCT had a greater positive effect on limb motor than speech motor symptoms of persons with PD (Baker, Ramig, Johnson, & Freed, 1997). Pre- and post-FCT surgery results revealed variable, inconsistent, and unremarkable changes in phonatory and articulatory measures for each person with PD. Measures included phonatory variables of jitter an shimmer, semitone standard deviation of voice fundamental frequency, speech acoustic variables of vowel duration, extent of glide, voice onset time, spirantization, as well as the listener’s perception of speech intelligibility, articulatory precision, and voice quality.

Deep Brain Stimulation (DBS)

Deep brain stimulation (DBS) refers to the electrical stimulation of the thalamus, the subthalamic nucleus (STN), or the GPi for treatment of Parkinsonian symptoms. DBS is performed in lieu of ablation techniques such as pallidotomy in order to duplicate the results of pallidotomy with a decreased risk of permanent neurologic deficit in persons with PD (Pahwa, Wilkinson, Smith, Lyons, Miyawaki, & Koller, 1997).

Chronic high-frequency unilateral thalamic stimulation is effective in reducing both essential and Parkinsonian (resting) tremor (Koller, Pahwa, Busenbark, Hubble, Wilkinson, Lang, Tuite, Sime, Lazano, Hauser, Malapira, Smith, Tarsy, Miyawaki, Norregaard, Kormos, & Olanow, 1997). The role of thalamic participation in speech production has been questioned. Basic motor speech functions such a speaking rate and articulation accuracy are suggested to be organized asymmetrically at the level of the thalamus, as found by thalamic stimulation (Mateer, 1978). Stimulation of the ventral-oral nucleus of
the thalamus produced silencing and slowing of speech (Schaltenbrand, 1975). Electrical stimulation of the left ventrolateral thalamus also caused alterations in object naming in the form of anomia, perseveration, and anarthria (Ojemann & Ward, 1971). No studies investigating speech production in persons with PD who have had thalamic stimulation have been reported.

Stimulation of the STN excites the globus pallidus, so if there is excess inhibition from the globus pallidus, then the STN works against this inhibition. Bilateral electrical stimulation of the STN improves akinesia and rigidity in persons with PD (Limousin, Pollak, Benazzouz, Hoffmann, Le Bas, Broussole, Perret, & Benabid, 1995). Its effect on speech production has yet to be determined.

Pallidal stimulation improved L-dopa-induced dyskinesias and to a lesser extent bradykinesia and rigidity (Tronnier, Fogel, Kronenbuerger, & Steinvorth, 1997; Gross, Rougier, Guehl, Boraud, Julien, & Bioulac, 1997). A subjective assessment of speech disturbances has shown general improvement during high-frequency stimulation of the GPi (Gross et al., 1997). No objective studies have reported the effects of pallidal stimulation on voice and speech production in PD.

OTHER TREATMENTS/COMBINED TREATMENTS

Clearly all of the current successful voice/speech therapy studies have been in effect, combined treatment studies. That is, all of the persons with PD that participated were optimally medicated. In another study, the combined effect of intensive speech therapy following bilateral thalamotomy was reported (Countryman & Ramig, 1993). A person with PD who had previously undergone bilateral thalamotomy was given the LSVT program. They had significant improvements in vocal functioning (e.g., increased intensity, increased vocal steadiness, increased harmonics to noise ratios, etc.) were measured immediately post treatment. However, they were unable to maintain these improvements at six and 12 months following treatment. The authors speculated that this lack of maintenance was due to lack of continued practice and/or a progression of their PD.

One treatment method that has not received much attention in the literature is augmentation of the vocal folds by injection of collagen, gelfoam, or Teflon. This method increases the bulk of one or both vocal folds, thus reducing the gap between the vocal folds often observed in persons with PD. In theory, this results in better vocal fold closure and thus improvements in vocal intensity and vocal quality. One recent study investigated the effects of vocal fold augmentation with percutaneous collagen injection (Berke, Gerratt, Kreiman, & Jackson, 1999). The self reported response (to a telephone survey) of 35 idiopathic persons with PD and glottal insufficiency was reported to be favorable in 75% compared to 16% unsatisfactory responses. Favorable responses
were found to correlate moderately with the duration of the dysphonia improvement. Clearly this treatment method offers promise for reducing the dysphonia common to persons with PD and this method could also be combined with voice therapy for perhaps longer duration of improved voice.

Another treatment method recently reported involves transcranial electromagnetic stimulation (Sandyk, 1997). In this case study of a young onset PD patient who reportedly had a severe speech impairment with severe stuttering, pulsed electromagnetic stimulation transcranially, subjectively improved above the improvement observed following drug treatment. This person’s speech was said to improve 20–30% following the addition of sertraline (a serotonin reuptake inhibitor) to his dopaminergic medications. Following four years of regular weekly administration of transcranial electromagnetic stimulation, greater and more consistent improvement in his speech was observed (Sandyk, 1997). The author attributes the improvement in speech to be the result of a facilitation of serotonergic transmission in conjunction with the sertraline. To date, the speech impairments of persons with PD have not been localized to a reduction of serotonin transmission, additionally, the weekly administration of this treatment method is not feasible for the majority of persons with PD.

**CONCLUSION**

Parkinsonian dysarthria, or hypokinetic dysarthria, affects five speech subsystems: respiration, phonation, articulation, resonance, and prosody. According to Darley et al. (1969a, 1969b), characteristics of hypokinetic dysarthria include monotonous pitch and loudness, reduced stress, variable rate, short rushes of speech, imprecise consonants, and breathy and harsh voice. All of these characteristics reduce overall speech intelligibility.

The purpose of this paper was to examine the different treatment approaches for persons with PD and to report the effects of these treatments on speech. Treatment methods included speech therapy, pharmacologic, and surgical.

Several types of drugs are used in treating persons with PD. L-dopa may be more effective than anticholinergics, MAO-B and COMT inhibitors, and DA agonists for improving speech. Sinemet treatment contributes to greater speech intelligibility through increased vocal loudness and more regular distribution of phonation and pauses (Mawdsley & Gamsu, 1971). Improved voice quality, pitch variation, and articulation with both short- and long-term use have also been observed (Wolfe et al., 1975). On the other hand, Daniels et al. (1996) demonstrated that there are no significant differences between vocal performance measures during the “on” or the “off” state.

Literature on the surgical treatments for PD suggest that these methods primarily improve limb motor symptoms of PD. Unilateral thalamotomy pro-
duced the deterioration of speech in the form of monotonous voice, slow speech (Jenkins, 1968), and diminished vocal loudness (Allan et al., 1966; Petrovici, 1980). Bilateral thalamotomy produced the same results, including word blocking as well (Matsumoto et al., 1976). There are not many reports supporting consistent improvement in specific subsystems of the speech mechanism following pallidotomy. A preliminary study indicated positive changes in some persons with PD in either phonatory or phonatory and articulatory measures post-surgery (Schulz et al., 1999). In addition, positive changes in vocal intensity have been reported in persons with mild hypokinetic dysarthria following unilateral PVP (Schulz et al., in press). Also, Barlow et al. (1998) found that following bilateral PVP, persons with PD exhibited greater force stability in their perioral system, and laryngeal resistance became more like control values. Though it was not reported, this evidence could correlate with increasing speech intelligibility. The study of the effects of speech after FCT revealed a greater effect on the limb motor system than the speech motor system (Baker, Ramig, Johnson, & Freed, 1997). No literature has been published regarding the effects DBS of the GPi, STN, nor the thalamus bilaterally on speech, but unilateral thalamic stimulation produced silencing and slowing of speech in persons with PD (Schaltenbrand, 1975) and anomia, perseveration, and anarthria (Ojemann & Ward, 1971). It therefore appears that these surgical procedures have little positive effect on speech production. The suggestion by Buck and Cooper (1956) still appears to hold true: “. . . improvement in speech should not be a prime goal in selecting patients for surgical treatment of parkinsonism” (p. 1290).

Speech therapy (in persons who are optimally medicated) appears to be the most effective method of treatment for improving voice and speech production in persons with PD. The therapeutic devices used in speech therapy include the voice amplifier, DAF, the wearable intensity biofeedback device, and a masking device. Overall, these devices increased speech intelligibility, but were primarily used in a clinical setting (with the exception of the wearable biofeedback device), so generalizing persons with PD speech outside of the clinic has not been fully demonstrated. Though the biofeedback device indicated improvement in quality of life (Rubow & Swift, 1985), the person with PD had to wear the cumbersome device in all situations in order to be understood, which may not be desired by others.

Although today treatment focusing only on prosody has largely been abandoned, improvements have been noted in the following aspects of speech: intelligibility (Scott & Caird, 1983; Robertson & Thomson, 1984), phonation or vocal loudness (Robertson & Thomson, 1984; Johnson & Pring, 1990), respiration, prosodic aspects of stress, and intonation (Robertson & Thomson, 1984), and rate of speech (Robertson & Thomson, 1984; Le Dorze et al., 1992), and pitch (Johnson & Pring, 1990; Le Dorze et al., 1992). A noted drawback in using this speech therapy method is that there is no literature sup-
porting maintenance of improvement past three months. It would be beneficial to obtain data on more long-term effects of these methods of therapy.

The LSVT appears to be more beneficial to persons with PD speech than any other treatment method. It has proven to be efficacious both in the short- and long-term. Literature revealed that those who had undergone the LSVT (who were optimally medicated) had either maintained or improved their vocal loudness for 12 months after treatment (Ramig et al., 1996). Increasing vocal loudness has contributed to greater intelligibility in the speech of persons with PD and has decreased the negative impact of PD on communication (Ramig et al., 1995a).

This review has attempted to detail the effects of various treatment approaches on the speech of persons with PD. Neither pharmacological nor surgical methods of treatment alone appear to significantly improve voice and speech function in PD. Currently, speech therapy in combination with optimal pharmacologic intervention has proven to be the most efficacious therapeutic method for improving voice and speech function. Future studies should investigate the effects of other combined treatment approaches. Perhaps the combination of pharmacological, surgical, and speech treatment will prove to be the superior approach for improving the communication abilities of persons with PD.

REFERENCES


**CONTINUING EDUCATION**

**Effects of Speech Therapy and Pharmacologic and Surgical Treatments on Voice and Speech in Parkinson’s Disease: A Review of the Literature**

**QUESTIONS**

1. Which of the following has been proven to be the most efficacious speech therapy treatment for Parkinson’s disease?
   a. Biofeedback training
   b. Respiratory training
   c. Phonatory and respiratory training
   d. Prosodic training
   e. None of the above have been proven efficacious

2. Which of the following procedures has been shown to make voice and speech worse in persons with PD?
   a. Bilateral thalamotomy
   b. Unilateral pallidotomy
   c. Fetal cell transplantation
   d. Deep brain stimulation
   e. Injection of collagen into the vocal folds

3. Pallidotomy alleviates limb symptoms of PD by:
   a. Increasing inhibitory outflow from the basal ganglia to the thalamus and cortex
   b. Increasing excitatory outflow from the basal ganglia to the thalamus and cortex
   c. Decreasing inhibitory outflow from the basal ganglia to the thalamus and cortex
   d. Decreasing excitatory outflow from the basal ganglia to the thalamus and cortex
   e. Eliminating the basal ganglia from participating in movement
4. Which of the following statements is true regarding speech and voice production following L-dopa treatment?
   a. Findings show a general trend for improvement in voice and speech function
   b. Subjective observations show improvement in voice quality, pitch variation, and articulation
   c. No difference in acoustic measures in the “on” and “off” medication state
   d. Reduced tonic activity of labial muscles following administration of L-dopa
   e. All of the above are true statements

5. Which of the following is not an explanation for discrepancies observed in voice and speech function following various medical treatments in persons with PD?
   a. Methodological differences between studies such as subject inclusion criteria, dysarthria severity, other co-occurring conditions.
   b. Differences between limb motor and speech motor substrates.
   c. Differences in tasks used to assess voice and speech functions.
   d. Differences in treatment durations.
   e. Differences in treatment responses due to neurophysiologic differences between limb motor and speech motor substrates.