FACIAL EMOTIONAL EXPRESSION FOLLOWING VOICE TREATMENTS IN INDIVIDUALS WITH PARKINSON'S DISEASE

by

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Abstract

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A growing body of work has documented impairments in emotional facial expression (i.e., masked facies) in individuals with Parkinson's disease (PD). These impairments negatively impact patients' social interactions and functioning in daily life. However, little attention has been given to remediating facial emotional expression deficits in PD. Preliminary research has demonstrated that the treatment of voice using the Lee Silverman Voice Treatment (LSVT®; Ramig et al., 1995) has beneficial effects on limited aspects of facial expression in PD (Spielman et al., 2003). The present study extends the literature by examining the effects of two voice treatments on facial expression in PD in a comprehensive way, including facial mobility (FM) and three aspects of facial emotional expressivity (i.e., frequency [EF], variability [EV] and intensity [EI]). Participants included 56 posers, individuals who produced emotional and non-emotional monologues, and 18 raters, individuals who rated posers' facial expressions from video-recorded monologues. Ratings were made on a 7-point Likert scale for the four aspects of facial expression. Raters were trained to criterion, and reliability was high for each emotional expression variable (Intraclass Correlation Coefficient range .85 to .90). The study included four poser groups: 3 PD groups whose posers were randomly assigned into an LSVT, Articulation Voice Treatment (ARTIC), or a no treatment control group, and a demographically matched healthy control group (NC). Findings revealed that PD male posers displayed impaired facial

expression at baseline compared to NCs on all variables examined, although PD women did not differ from NCs for any aspect of facial expression. Treatment findings showed that patients who received LSVT were rated as having higher FM, EF, EV, and EI after treatment, four weeks later, than at baseline. This increase was not observed for the 3 other poser groups. It is speculated that LSVT improves facial expression because facial and vocal expression are emotional communication channels that exist within a larger network of emotional processing. Facial and vocal emotional expressions are linked at several levels of neural organization: cortical, subcortical, and cranial nerve. The broader clinical implications of our findings are that masked facies can be remediated using LSVT.

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CHAPTER I.

Introduction

Research Question

A growing body of work has documented impairments in emotional facial expression in individuals with Parkinson's disease (PD). These impairments negatively impact patients' social interactions and functioning in daily life. Little attention has been given to remediating these facial emotional expression deficits in PD. However, preliminary research has demonstrated that the treatment of voice using the Lee Silverman Voice Treatment (LSVT; Ramig, Countryman, Thompson, & Horii, 1995) has beneficial effects on facial expression in PD (Spielman, Borod, & Ramig, 2003). This unanticipated treatment effect is thought to occur because facial and vocal expressions are both emotional communication channels that exist within a larger network of emotional processing. Thus, facial and vocal expressions are mediated by overlapping neural substrates and are neuroanatomically linked at several levels of neural organization: cortical, subcortical, and cranial nerve. The effect of this voice treatment on facial emotional expression is thought to occur from the association between vocal and facial emotional communication channels and their shared neural substrates. Patients treated with LSVT, as compared to a control respiratory therapy, showed a significantly larger percent change in facial expressivity after treatment (Spielman et al., 2003). This preliminary research was based on a small sample size and examined only two aspects of facial expressivity: facial mobility and social engagement.

The present study will extend the literature by examining the effects of LSVT on facial expression in PD in a more comprehensive way than has been done to date. This comprehensive approach will include three aspects of facial expressivity: frequency, variability, and intensity. Evaluating these set of variables will allow us to determine which aspects of facial expression are most influenced by LSVT. Furthermore, the study will attempt to replicate preliminary findings reported in the initial study (Spielman et al., 2003).

The present study also incorporates a methodological improvement over Spielman and colleagues' (2003) study. In this previous study (Spielman et al., 2003), facial expression ratings were made from video segments of participants who spoke about a topic of their choosing or who were engaged in a clinical interview. Thus, the content of the material that the participants were discussing may not have been fully conducive to emotional facial expression. In the present study, participants were explicitly asked to recount emotionally salient happy, sad, and angry experiences, as well as a neutral episode, from their lives, and the facial expression ratings will be based on these data. Therefore, the fact that participants are instructed to speak about emotional experiences will likely elicit more facial expression from participants than the potentially non-emotional speech content collected in the previous study. The present study will also include a larger sample size of PD patients than was previously examined, and also incorporated two control groups. Results will contribute to the literature by characterizing the type of emotional facial impairments present in PD using a novel and more comprehensive way of conceptualizing facial expressivity than has been done before, by assessing emotional intensity, frequency, and variability. This knowledge may be used by researchers to further understand the mechanisms connecting vocal and facial expression, and it may be used by

clinicians to select appropriate treatment options for individuals with PD. Finally, it may be used by PD patients' family members to anticipate the manifestation of their loved one's disease process and the potential benefits of voice treatments. Informing researchers, clinicians, and family members may ultimately improve the daily life of PD patients.

Background

Role of emotion in daily functioning.

Emotional processing. The ability of humans to express emotions, that is, experiences that are uniquely private, through behavioral means represents a critical component of social functioning (Brozgold, Borod, Martin, Pick, Alpert, & Welkowitz, 1998). Behavioral expressions of emotion facilitate accurate communication of affect (Ekman, 1965) and thus enable healthy social interactions. An individual's ability to communicate emotions to others significantly affects quality of life and contributes critically to his or her ability to function fully in society (Dowding, Shenton, & Salek, 2006). The importance of facial expressions is not only necessary for healthy social interaction in our present society, but is theorized to have been important for survival of early man (Darwin, 1872). Charles Darwin theorized that the ability for humans as a species to produce pronounced facial expressions was so critical for survival that the skill was incorporated into our behavioral repertoire through natural selection (Darwin, 1872).

The behavioral manifestation and communication of feeling occurs through several communication channels, including facial expression, postural expression, and gestural expression, as well as lexical and prosodic aspects of language. While any given emotion can be expressed through several of these channels, and is often expressed concurrently in more than one channel, facial expression is arguably the primary way individuals convey emotion (Assuras,

Barry, Borod, Halfacre, & Crider, 2005; Borod & Koff, 1984). Thus, facial expression holds a unique place in an individual's repertoire of social and affective functioning.

A more comprehensive understanding of facial expression can be made once it is placed within the context of a multicomponent framework of emotional processing. Borod (1993) proposed four aspects or components of emotion: processing modes (e.g., perception, expression, experience, and physiological arousal), communication channels (e.g., facial, prosodic, lexical, gestural, and postural), emotional dimensions (e.g., pleasant-unpleasant), and discrete emotions (e.g., happiness and sadness). Emotional perception is defined as the ability to perceive or encode the facial expression of another individual whereas emotional expression is the ability to behaviorally produce or express a particular emotion. There is a growing body of work devoted to each of the aforementioned aspects of emotion and their respective elements in healthy, psychiatric, and neurological populations (for review, see Borod, Tabert, Santschi, & Strauss, 2000). Among processing modes, specifically between emotional expression and perception, clear dissociations have been demonstrated in both healthy (Fridlund, Ekman, & Oster, 1987) and brain-damaged populations (Borod, Koff, Lorch, & Nicholas, 1986). Dissociations have also been demonstrated between expression and experience (Borod, Rogers, Spielman, Halfacre, McCabe, Flanagan, & Ramig, 2008). Due to these documented dissociations, it is appropriate to examine one processing mode in isolation. Our study will focus on a single processing mode: emotional expression. With respect to communication channels, the facial channel will be the focus of the present study. However, since a strong association has been demonstrated between the expression of facial and vocal communication channels in brain-damaged individuals (Borod et al., 1990; Borod et al.,1985), a discussion of both channels of communication will be

presented.

Gender differences have long been documented in the emotional processing literature (e.g., for review, see Borod & Madigan, 2000), and these differences are found across several processing modes. There is evidence that women have an advantage over men with respect to the perception, expression, and experience of emotion. Women may form more accurate perceptions of others' facial emotional expression, (e.g., Donges, Kersting, & Suslow, 2012) are typically rated as producing more pronounced facial emotional expressions, and experience emotions with higher intensity compared to men (Huang & Hu, 2009).

Based on the multicomponent framework of emotional processing presented above, it follows that impairment in one component of emotion processing can occur despite intact functioning in another component. Thus, impairment in emotional processing can be said to occur if an individual is unable to produce emotional expression, despite otherwise preserved emotional functioning. Indeed, there is evidence that individuals who have facial expression impairments (e.g., PD patients) experience emotion normally, that is, they experience emotion with the same intensity and accuracy as healthy controls (Borod et al., 2008). For such individuals, a mismatch is created between what they feel subjectively and what they project to the outside world.

Emotional expression and social functioning. The mismatch between experience and expression of emotion may partially account for some of the problems facial expression impairments create in the daily social functioning and communication of patients with PD. The relationship between facial expression and social functioning was examined by Brozgold and colleagues (1998) in patients with PD, right-hemisphere brain damage, unipolar depression, and

schizophrenia, and demographically matched healthy adult control participants. Participants completed clinical rating scales that assessed their level of social functioning. They were later videotaped while recollecting pleasant and unpleasant emotional experiences and were urged to relive each experience with as much intensity as possible. Subsequently, raters, naïve to the purpose of the study, watched the emotional monologues and rated the amount and intensity of positive and negative emotion displayed by each participant. The ratings of social functioning and facial expression were later correlated. Results showed that PD patients exhibited less intense emotions compared to normal controls, although this difference did not reach significance. However, there was a positive relationship between intensity of facial expression and social functioning, wherein the more intense patients' facial expressions, the better their social functioning. Findings also showed that normal controls displayed significantly more positive emotion than patients with PD, schizophrenia, and right-hemisphere brain damage, and less negative emotion than all patient groups. Taken together, results from this study (Brozgold et al., 1998) suggest that patients with PD display both quantitative (reduced intensity) and qualitative (greater negative and reduced positive emotion) differences in facial expression compared to normal controls. These differences could account for the impairments patients experience in daily social functioning. This study identifies facial expression as a treatment target for rehabilitation of social functioning impairments in patients with various neurological and psychiatric disorders.

It is interesting to note that the reverse effect has also been shown experimentally, where interpersonal interactions have been shown to affect motor symptoms. In a case study of a 74 year-old male PD patient (Griffin & Greene, 1994), negative interpersonal interactions

characterized by unfavorable comments from his wife led to increased orofacial bradykinesia. These motor symptoms were later reversed when the patient was engaged in a pleasant conversation with another individual.

Impression formation. The special role of facial expression in social interaction may be mediated by impression formation. Social partners may form impressions about individuals based on the emotional intensity (EI), emotional frequency (EF), and/or emotional variability (EV) of their facial expression. EI is defined as the intensity with which a given emotional expression is produced, EF is the number of emotional expressions produced within a given period of time, and EV is the number of *different* types of emotional expressions produced during a given time period. Indeed, a link has been established between the variability of facial expression and impression formation (Riggo & Friedman, 1986). In this study, 15 non-verbal behaviors were studied, and their role on initial impression formation examined. Results indicated that variability in the facial expressions of the female participants was the cue most consistently related to positive impression ratings. This link has also been documented in patient populations. In a series of studies, Pentland and colleagues demonstrated that occupational therapists, physiotherapists (Pentland, Pitcairn, Gray, & Riddle, 1987), and senior speech therapy students (Pentland, Gray, Riddle, & Pitcairn, 1988) formed negative impressions about PD patients' mood, personality, and intelligence. PD patients were perceived as more anxious, depressed, and passive compared to a control group of patients with ischemic heart disease. Furthermore, the patients were not perceived to relate well or appear to enjoy the conversation with the interviewers, and were rated as less likable than controls. These impressions were made despite no evidence of impairments in mood, personality, or intelligence as assessed by

standardized tests. In a follow-up study, it was established that these negative impressions were related specifically to the facial expressions of the PD patients (Pitcairn, Clemie, Gray, & Pentland, 1990). The patients' facial expressions were less frequent than those of normal controls, and their smiles were rated as less genuine and perceived as false. Thus, results indicated that the facial expressions produced by patients were quantitatively as well as qualitatively different than those of the control participants, consistent with findings from Brozgold and colleagues (1998). Results from our own laboratory (Dumer, Borod, Oster, Spielman, Rabin, & Ramig, 2011) also corroborate these findings, showing that the personalities of PD patients, assessed along four dimensions (i.e., extraversion, dependence, anxiety, and likability), were perceived more negatively when compared to normal controls. A factor that may contribute to social partners' negative impression formations of individuals with reduced facial expression is the inconsistency with which individuals with masked facies are able to produce facial expression. Parkinson's patients are often able to produce a facial expression in certain conditions, for example, under voluntary control but may not be able to produce it spontaneously. It is widely accepted that the production of spontaneous facial expression is dissociable from the production of facial expression to command in PD (e.g., Smith, Smith & Ellgring, 1996). In the Smith et al. study, spontaneous facial expressions were elicited by showing PD patients and normal control emotionally salient clips from popular movies. Posed facial expressions were elicited by asking participants to mimic how they look when they feel various emotions. In both posed and spontaneous conditions, the protocol was designed to elicit expressions reflective of happiness, sadness, fear, disgust, and anger. In addition, all participants were asked to rate their experience of six emotions after viewing each video clip. Results

indicated that with respect to smile intensity, PD patients were selectively impaired in their ability to produce spontaneous facial expressions. However, although PD patients' posed smiles were lower in intensity than were those for normal controls, no significant differences were found among controls, mild PD patients, or moderate PD patients. Furthermore, PD patients' emotional experience ratings were as high as or higher than control participants, consistent with Borod and colleagues (2008). It should be noted, however, that posed expressions are not always spared in PD; there are documented posed expression impairments in PD patients (Borod et al., 1990). For the most part, however, there is a behavioral dissociation between posed and spontaneous facial expression in PD. This dissociation results from distinct neural mechanisms that will be described in further detail in subsequent sections of this paper.

The inconsistency with which a patient is able to produce facial expressions is important because posed and spontaneous expressions have different effects on impression formation. For example, posed smiles have been associated with negative impressions characterized by less favorable judgments of genuineness (Krumhuber & Kappas, 2005). It is likely that observers can easily discriminate between spontaneous and posed facial expressions, as it has been documented that there are quantifiable differences between these two types of expression. For example, the amplitude and timing of movements that generate smiles are different in posed and spontaneous expressions (Schmidt, Ambadar, Cohn, & Reed, 2006). Thus, observers may falsely assume that a patient who produces posed expressions is not engaged and not enjoying a conversation due to expressions that are perceived as disingenuous and false. Social partners who may have observed an individual smiling in the past may not realize that the same individual is unable to produce a smile spontaneously under natural conditions. This differential impairment in patients' ability to

produce emotional facial expressions under different conditions may have negative interpersonal consequences for the patient, perhaps more so than if both systems were similarly impaired. The fact that patients *can* make facial expressions but appear not to do so spontaneously may reinforce a perceiver's assumption that the patient is not engaged in a conversation, is unresponsive, or is even depressed and/or anxious. This may further lead to false assumptions about the intentions, motivations, and internal state of the individual, and these assumptions could extend into and interfere with future social interactions. These may be some of the reasons observers form negative impressions of PD patients based on their facial expressions.

Impression formation is vital to social functioning because it shapes subsequent social interactions between an individual and his/her social partners (Riggio & Friedman, 1986). Social partners often form immediate first impressions of individuals based on very short interactions and limited information (Schneider, Hastorf, & Ellsworth, 1979). Despite the lack of information, however, the impressions formed may be enduring. If those impressions are based on faulty assumptions (i.e., this person is not likable or not engaging), future social interactions may suffer. Enduring effects of such impressions may result from social partners' assumptions that facial expressions reveal an individual's true personality traits and character. This hypothesis was tested and supported by Tickle-Degnen and Lyons (2004). Findings from their study indicated that novice clinicians formed inaccurate impressions about PD patients' personality traits based on their facial expressivity. Facial expressivity was assessed by three independent raters using the facial expression item of the motor exam portion of the Unified Parkinson's Disease Rating Scale (UPDRS; Fahn & Elton, 1987). This item is assessed on a 5-point Likert scale, with "0" representing normal facial expression, "1" being minimal hypomimia, "2" being

slight but abnormal diminution of facial expression, “3” being moderate hypomimia with lips parted some of the time, and “5” being masked or fixed facies with severe or complete loss of expression and lips parted ¼ inch or more. The novice clinicians inaccurately labeled patients with less facial expression as more neurotic and less extroverted than did the more experienced clinicians. These findings suggest that social partners, just like the inexperienced clinicians in the Tickle-Degnen and Lyons (2004) study, may use facial expressions to infer patients’ personality traits. As the disease progresses, facial expressions are less reliable reflections of internal dispositions (Tickle-Degnen & Lyons, 2004), and thus, this approach may lead to inaccurate attribution of personality traits. Furthermore, personality traits have been shown to be stable over time and across situations (McCrae & Costa, 2003). Because inaccurate judgment of an individual's personality may affect future social interactions, facial expression impairments may have enduring effects if they affect personality judgments.

Impression formation in the context of clinical populations may be especially important. In psychiatric and neurological populations, when inaccurate impressions are formed by health professionals, there may be negative medical consequences for the patient. It has been documented that clinicians can incorrectly perceive PD patients as depressed (Pentland et al., 1988). The potential misdiagnosis of depression presumably occurs due to the physical presentation of the patient. PD patients often present with flat affect (e.g., Borod et al., 1990), which clinicians may easily mistake for depressed mood if a full psychiatric diagnostic interview is not conducted. Furthermore, some of the somatic symptoms of depression, such as psychomotor agitation or retardation (American Psychiatric Association, 2000), are also present in PD. Misdiagnosis of a mood disorder presents a serious negative consequence to a patient as it

may set the treatment off course.

The importance of clinician impressions is also highlighted in the schizophrenia literature. In a study of patients with stabilized symptoms of schizophrenia, spontaneous facial expressivity during an interview predicted clinician-rated work and social disability (Troisi, Pompili, Binello, & Sterpone, 2007). Work and social disability were assessed by a psychiatrist naïve to the purpose of the study. Facial expression was assessed specifically with respect to prosocial behavior, defined as those behaviors that invite social interaction (e.g., smiling and moving head to the side), and prevent hostile responses (e.g., mouth corners back), among others. These were assessed by a different trained observer who viewed and rated patients' behavior during a videotaped interview. Those patients who showed fewer displays of prosocial behavior were independently rated by the psychiatrist as suffering from more functional disability than patients who showed a greater amount of prosocial behavior. These findings are particularly important because clinician-rated work and social disability have significant implications for the services individuals are granted. The comparison of patients with schizophrenia and PD is especially relevant in the context of the present discussion, as one of the prominent features of schizophrenia is flat affect, characterized by a severe lack of expressive facial behavior (Borod et al., 1990). Thus, evidence from both schizophrenia and PD indicates that impairments of facial expressivity in patient populations may have highly significant and overarching consequences for patients. PD is a neurological disorder in which facial expression impairments are especially prominent.

Motor and emotional deficits in PD.

PD epidemiology. PD is a neurodegenerative hypokinetic disorder with a paucity of spontaneous movement attributed to the degeneration of dopaminergic neurons in the substantia nigra pars compacta, one of the basal ganglia nuclei. PD is one of the most common movement disorders, affecting approximately 1% of individuals over age 65 (Blumenfeld, 2002). Based on the total number of individuals over 65 around the world, it is estimated that approximately six to eight million individuals are currently living with PD (Sapir, Ramig, & Fox, 2008), with over one million in the United States alone (Kandel, Schwartz, & Jessel, 2000). The incidence of PD is significantly higher in men than women (Van Den Eeden, Tanner, Bernstein, Fross, Leimpeter, Bloch, & Nelson, 2003). Age of onset is typically between 40-70, and the progression of the disease is most often insidious over the span of 5-15 years. PD was first described by James Parkinson in 1817, and today, its pathophysiology is one of the best understood among the movement disorders (Kandel et al., 2000). PD is not genetically determined in most cases, and its etiology is unknown (Blumenfeld, 2002).

PD signs and symptoms. PD is characterized by a constellation of four cardinal motor signs: akinesia, bradykinesia, rigidity, and tremor. Akinesia is the impaired initiation of movement that results in a loss of voluntary movement, and bradykinesia represents reduced amplitude and velocity of voluntary movement (Kandel et al., 2000). Rigidity refers to muscle rigidity, and the tremor is characteristic of the disorder, with a frequency of 4-5 per second at rest, often referred to as Parkinsonian tremor (Blumenfeld, 2002). In addition to these cardinal signs, several additional motor signs are also typical: flexed posture and postural instability that causes unsteady gait as well as generally impaired balance (Blumenfeld, 2002; Kandel et al.,

2000).

Although the motor impairments described above are central to the disorder and cause disability due to reduced mobility and independence, motor problems specific to the face also cause significant functional impairments. PD patients have characteristic masked facial expressions that appear as a reduction in spontaneous blink rate, reduced facial mobility, and decreased facial expression. This is often referred to as masked facies, hypomimia (Blumenfeld, 2002), or mimitic facial paralysis (Rinn, 1984), and has crucial implications for individuals with this disease. As discussed above, patients' reduced ability to express their feelings through facial expression can lead to interpersonal problems.

Pathophysiology of Parkinson's disease. The hallmark motor symptoms of PD result from an underlying imbalance within the basal ganglia-thalamocortical circuitry. Fluid voluntary motor action depends on the balance between two largely segregated neural pathways connecting the basal ganglia and thalamus: the direct and indirect pathways. The direct pathway facilitates movement while the indirect pathway inhibits unwanted movement. Therefore, it is only through the coordinated action of these two pathways that fluid voluntary motor action is produced. A brief description of the normal functioning of these pathways will be presented, followed by the effects of PD on their dysfunction.

The following brief discussion pertaining to the normal functions of the direct and indirect pathways of the basal ganglia is adapted from Blumenfeld (2002) and Kandel (Kandel et al., 2000). In the normal human brain, the striatum, consisting of the caudate nucleus and putamen (Kandel et al., 2000), is the main recipient of afferents to the basal ganglia. Within the basal ganglia, the direct and indirect pathways synapse serially onto different nuclei and remain

segregated until they converge onto the thalamus. All synaptic connections within these two pathways of the basal ganglia are inhibitory and mediated through GABA-ergic neurons, with the exception of one excitatory synapse in the indirect pathway. This difference generates the direct and indirect pathways' opposing effects on the basal ganglia output nuclei, and thus on the thalamus. Cortical excitation of the direct pathway results in disinhibition (i.e., excitation) of the thalamus, which facilitates movements through its connections with the motor and premotor cortex. Excitation of the indirect pathway on the other hand, results in inhibition of the thalamus and reduction in movements (Blumenfeld, 2002).

In PD, according to Blumenfeld (2002) and Kandel (Kandel et al., 2000), the degeneration of dopaminergic neurons of the substantia nigra pars compacta results in a change in the functioning of the direct and indirect pathways. This results because the dopaminergic projections from the substantia nigra pars compacta to the striatum have different effects on the direct and indirect pathways. Dopamine has an excitatory effect on striatal neurons in the direct pathway, and an inhibitory effect on striatal neurons in the indirect pathway. Reduced excitatory activation in the direct pathway leads to reduced activation of the thalamus and cortex, and a reduction in movements. A loss of inhibitory dopaminergic neurons in the indirect pathway serves to facilitate the function of this inhibitory circuit. Thus, the effect of dopaminergic degeneratation of the substantia nigra pars compacta neurons is net inhibition of movement though both direct and indirect pathways.

Emotional communication deficits in PD. Disease-related changes in the underlying circuitry of the brain lead to a host of motor deficits described above as well as impairments in facial expression and in aspects of voice and speech. Both vocal and facial expression are

important forms of communicating internal emotional states and are often activated together in times of danger. A discussion of the specific emotional communication impairments found in PD is detailed below.

Facial expression. A loss of facial expressivity in PD is one of the prominent clinical features of the disease which negatively impacts social functioning and the daily lives of PD patients. This deficit has long been described anecdotally by clinicians and family members of PD patients (Dakof & Mendelsohn, 1986). Masked facies has also been documented experimentally in numerous studies using both ecologically-valid rating systems and objective facial measurements, using standardized coding techniques. In fact, masked facies is such a common and reliable feature of Parkinsonian symptomatology that PD patients have been included in studies of spontaneous facial expression to provide minimum baseline levels of emotional expressivity against which other neurological populations are compared (Buck & Duffy, 1980). In the Buck and Duffy study, patients were shown a series of emotionally laden slides portraying familiar and unfamiliar people and scenes, and the accuracy with which naïve raters correctly identified the type of slide shown was assessed. As expected, results indicated that raters' judgments of PD patients' expressions were the least accurate when compared to those of other brain-damaged groups.

The first comprehensive examination of emotional processing in psychiatric and neurological populations using the same battery of tests was undertaken by Borod and colleagues (1990). To our knowledge, this study also provided the first experimental data for PD patients producing posed facial expressions. PD patients were compared with schizophrenic, depressed, right brain-damaged, and healthy adults on tasks of emotional expression and perception in both

facial and vocal channels. For the expression tasks, performance was rated for accuracy and intensity by naïve raters, and results indicated that PD patients voluntarily expressed emotion significantly less accurately than normal controls.

In a series of studies, Katsikitis and Pilowsky (1988, 1991) documented facial expression deficits in PD patients using a computer program that employs mathematical models of the face to quantify facial expression. This computerized approach provides 12 quantitative measures of facial expression and is fully automated (Pilowsky, Thornton, & Stokes, 1985). In the first study, PD patients and a control group viewed cartoons, and their smiles were measured quantitatively (Katsikitis & Pilowsky, 1988). Results revealed that while smiling, PD patients opened their mouths to a lesser extent compared to healthy controls. Furthermore, PD patients smiled less overall while watching cartoons when compared to healthy controls. In a follow-up study of PD patients, patients with major depression, and normal controls (Katsikitis & Pilowsky, 1991), participants were presented with "amusing slides," and expressions were measured using the Facial Expression Measurement program described above (Pilowsky et al., 1988). Results indicated that both depressed and PD patients smiled significantly less often than healthy controls. The same result emerged in a more recent study (Simons, Pasqualini, Reddy & Wood, 2004) in which PD patients and normal controls were engaged in conversation and watched video clips and their facial expressions were measured using both a behavioral rating system (based on observation) and the Facial Action Coding System (FACS; Ekman & Friesen, 1978). Results indicated that across measurement techniques and experimental conditions, PD patients displayed reduced spontaneous facial expressivity compared to controls.

Although facial expression impairments have been documented in PD (Borod et al.,

1990; Buck & Duffy, 1980; Katsikitis & Pilowsky, 1988, 1991; Simons et al., 2004; Smith et al., 1996), it is important to be mindful of the high incidence of depression in this population, which is estimated at approximately 40% (Raskin, Borod, & Tweedy, 1990), with some variability reported in the literature (Zgaljardic, Borod, Foldi, & Mattis, 2003). It has also been documented that unipolar depression is associated with reduced facial expression (Jaeger, Borod, & Peselow, 1986). However, while the high incidence of depression in PD patients may play *some* role in the facial expression impairments documented in the literature (Katsikitis & Pilowsky, 1988), there are several reasons to believe that depression cannot fully account for a reduction in facial expression in PD.

One line of evidence emerges from the studies of facial expression discussed above. For example, Smith and colleagues (1996) found that while PD patients' Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996) scores were significantly higher than those for normal control participants, covarying for depression did not significantly affect the facial expression results. This indicates that higher levels of depressive symptoms in PD patients did not account for corresponding reduced facial expressivity in the patients examined (Smith et al., 1996). Furthermore, Katsikitis and Pilowski (1991) showed that despite PD patients having higher depression scores than controls, no significant correlations emerged between the depression scores and quantitative measures of facial movements for the PD or control groups, only for the depressed group. This indicates that for the PD patients in this study, higher depression symptomatology was not significantly associated with facial impairments.

Another line of evidence suggesting that depression does not *fully* account for a reduction in facial expression in PD emerges from symptomatology of various subtypes of PD patients.

The first group of PD patients of interest is those not diagnosed with depression. It has been documented that the phenomenon of masked facies is also found in PD patients who are not depressed (Rinn, 1984). Thus, in this particular group, masked facies is not related to depression and is presumably connected to the PD disease process. Furthermore, masked facies is also seen in patients who have suffered strokes, tumors, and lesions of the basal ganglia but are not depressed (Rinn, 1984), suggesting that masked facies is related to neuropathology rather than to depression. Further evidence emerges from the phenomenon of hemiparkinson's disease. In this condition, motor symptoms are lateralized to one side of the body and may be transient or may persist for years (St. Clair, Borod, Sliwinski, Cote, & Stern, 1998). For these PD patients, masked facial expressions appear on only one side of the face, and this presentation is highly inconsistent with affective symptoms of depression that normally present on both sides of the face (Rinn, 1984).

Volitional versus spontaneous innervation of the face. As discussed previously, PD patients are often able to make emotive facial expressions to command, but are impaired in their ability to produce spontaneous emotional facial expressions (Heilman, Blonder, Bowers, & Crucian, 2000; Smith et al., 1996). In this document, emotional facial expressions will be referred to as spontaneous expressions and the act of making facial expressions to command will be referred to as posed expressions. Spontaneous expressions are considered to be involuntary, and posed expressions are considered volitional. This difference in functioning across posed and spontaneous modes results from distinct neural networks that subserve these different types of facial expressions. The neuroanatomical correlates of this dissociation are well documented and have been used diagnostically in the field of neurology for over 70 years (Borod & Koff, 1984;

Karnoush, 1945; Monrad-Krohn; 1924; Rinn, 2007; Thompson, 1985). Deficits in posed expression typically arise from cortical lesions of the motor strip and corticobulbar projections, whereas deficits in spontaneous expression typically arise from dysfunction of subcortical regions, most often of the basal ganglia. In his review of mechanisms for producing facial expression, Rinn (1984) argued that the dissociation can be understood when the motor system is examined through progressively functionally differentiated evolutionary stages. In the primitive brain, consisting of the basal ganglia and other structures, the behavioral repertoire of an animal was limited to highly patterned automatic behaviors. These evolved into what we now conceptualize as emotional reactions. Emotional reactions, when seen as survival mechanisms (such as fear for the flight response and anger for the fight response), were governed by these primitive brain regions and were devoid of conscious thought. This line of thinking is echoed in Charles Darwin's writing, which argued that emotions are functional products of evolution through natural selection (Darwin, 1872).

Rinn (1984) went on to argue that an important organizational principle in evolutionary neuroanatomy is that primitive regions of the brain and their corresponding functions are not replaced over time but rather work in concert with more developed regions. Therefore, as the cortex developed and gave rise to conscious thought and decision-making, emotional responses were no longer automatic responses meant only to preserve survival of the organism but were modulated by higher cortical regions and became more fine-tuned (Rinn, 1984). When understood through this evolutionary perspective, the neuroanatomical dissociation between the two types of facial expression becomes clear. Impulses for posed expressions originate in the cortical motor strip and, therefore, are controlled by conscious thought. These movements then

travel to the facial nucleus through the pyramidal tract and are contralaterally innervated (Borod, Haywood, & Koff, 1997; Borod & Koff, 1984; Rinn, 1984). Impulses for spontaneous (i.e., emotional) facial movements arise from the phylogenetically older extrapyramidal motor system and are comprised predominantly of subcortical nuclei, mainly the basal ganglia, and parts of the frontal and prefrontal cortices (Borod et al., 1997; Borod & Koff, 1984; Rinn, 1984). Spontaneous expression is thought to be bilaterally innervated (Borod et al., 1997) and to have a much more complicated neuroanatomical circuitry than posed expression (Borod & Koff, 1984).

The neuroanatomical distinction between volitional and emotional facial expression can be demonstrated using several lines of evidence from clinical phenomena and surgical interventions. The first line of evidence emerges from a double dissociation between these behaviors, whereby either can be impaired by neurological damage while the other is preserved. The first example is illustrated in the case of PD and referred to as emotional or mimetic paralysis, or reverse facial paralysis (Borod & Koff, 1984). This condition results in patients' reduced ability to produce spontaneous emotional facial expressions, whereas volitional expressions are relatively preserved (Rinn, 1984, 2007). Emotional facial paralysis has been documented in patients with dysfunction to several subcortical structures, including the basal ganglia (De Jong, 1979; Tschiassny, 1953), thalamus (De Jong, 1979; Ross & Mathiesen, 1998; Tschiassny, 1953), hypothalamus (De Jong, 1979), and substantia nigra (Tschiassny, 1953). The opposite scenario has been demonstrated in patients with lesions of the cortical motor strip or the projections from the motor strip to the brainstem, generally called the corticobulbar projections (Rinn, 1984; Rinn, 2007). These lesions lead to volitional facial paralysis that is experienced on the hemiface contralateral to the lesion (Borod & Koff, 1984; Borod et al., 1997). This

hemiparalysis occurs only in response to commands to express certain expressions. These same patients are able to smile bilaterally when there is an internal cue to perform that behavior, that is, the expression is performed spontaneously.

Another line of evidence has emerged from a surgical intervention called facial nerve anastomosis (Rinn, 2007). In this procedure, hemi-paralysis of the face is corrected surgically by grafting intact nerve fibers onto the facial nerve (Rinn, 2007). Successful outcomes of this surgery result in patients who are able to volitionally control facial muscles, although they never regain spontaneous facial expression. This procedure and its outcome further suggest distinct neuroanatomical pathways for posed and spontaneous facial expression.

The present study explores only spontaneous facial expression in PD patients. Thus, any dysfunction in spontaneous expression will be thought to result from dysfunction in the subcortical structures described previously.

Voice and speech. Emotional deficits in PD are not isolated to the facial communication channel, but include the vocal channel as well. Impairments in an individual's voice have been shown to reduce social interactions, thus reducing the possibility of emotional communication (Miller, Noble, Jones & Burn, 2006). Nearly 90% of patients with idiopathic PD have speech and voice symptoms (Logemann, Fisher, Boshes, & Blonsky, 1978; Ramig, Fox, & Sapir, 2008). Speech is comprised of several subcomponents, including respiration, phonation, resonance, and articulation. The speech disorder in PD is comprised of a constellation of symptoms collectively known as hypokinetic dysarthria (Sapir et al., 2008). PD patients experience hypophonia (Ho, Bradshaw, & Iansek, 2008), or reduced volume of the voice, although they typically have no awareness that they are speaking softly (Fox, Ramig, Ciucci, Sapir, McFarland, & Farley, 2006).

In fact, when they are cued to speak at a normal amplitude to improve their intelligibility, they often perceive their speech as too loud (Liotti, Ramig, Vogel, New, Cook, Ingham, & Fox, 2003). PD speech is also characterized by dysphonia, defined as poor voice quality, and hypoprosodia (Mobes, Joppich, Stiebritz, Dengler, & Schroder, 2008), defined as a reduction in voice pitch inflections or a monotone voice. Patients also have a reduced range of articulatory movements (Sapir, Spielman, Ramig, Story, & Fox, 2007), that is, hypokinetic articulation. Finally, speech can be hesitant (Sapir et al., 2008) whereas articulation is rushed when performing repetitive movements (Moreau, Ozsancak, Blatt, Derambure, Destee, & Defebvre, 2007). These vocal, speech, and prosodic symptoms impede social interactions and, as a result, negatively affect emotional communication (Miller et al, 2006; Pell, Cheang & Leonard, 2006). Miller and colleagues (2006) interviewed patients with PD and examined their feelings and attitudes regarding changes in their voice and speech. PD patients expressed concerns regarding their interactions with others, their intelligibility, and their ability to participate in a conversation. These concerns negatively impacted self-concept and directly affected socialization, with some patients expressing apprehension at the prospect of social interaction, and others completely withdrawing socially.

Rehabilitation of emotional processing impairments. Rehabilitation of speech and voice in PD has received considerable attention and has been approached by several different types of voice therapies (Schulz & Grant, 2000). However, the effectiveness of all such approaches has been limited in terms of long-term success except for the Lee Silverman Voice Treatment (LSVT; Ramig et al., 2004). LSVT will be discussed in detail below and further described in Appendix A.

Rehabilitation of emotional facial expression. Little attention has been devoted to the rehabilitation of facial emotional expression impairments in PD. The only known study, to date, that was primarily aimed at improving facial mobility in PD patients was conducted by Katsikitis and Pilowsky (1996). There are only two known studies that have addressed facial movement in PD; however, one study (Katsikitis & Pilowsky, 1996) focused exclusively on the motor aspects of facial movement (i.e., mobility) whereas the other (Speilman et al., 2003) addressed the emotional processing aspects of facial movement (i.e., emotional expression). Using an orofacial treatment (e.g., brushing muscles or applying ice to muscles), Katsikitis and Pilowsky (1996) documented improvements in PD patients' facial mobility, most notably around the mouth region. However, this study was targeted at the mobility of facial muscles (i.e., motor changes) and did not specifically address facial expression or any aspect of emotional processing. The authors noted that their findings could be used to guide other rehabilitation programs that specifically target communication of emotional states through facial expression.

The field of cognitive rehabilitation is anchored by the two rehabilitation principles of remediation and compensation (Raskin, Bloom & Borod, 2000). Remediation refers to restoration of a previously intact ability whereas compensation focuses on finding alternative ways to adapt to loss or trauma without attempting to restore it directly (Raskin et al., 2000). Emotional impairments that occur in neurological disorders and acquired brain injury have traditionally been treated pharmacologically or via cognitive or behavioral therapy (Raskin et al., 2000), which are largely geared to remediation.

Although remediation approaches have had some clinical utility, compensatory

approaches may be especially beneficial in the rehabilitation of emotional facial expression. Utilization of compensatory mechanisms is ideal in the case of emotion, given that we conceptualize emotion as being multidimensional (Borod, 1993), consisting of several communicational channels, each distinct yet interconnected. In the presence of a discrete impairment, improved emotional functioning may be facilitated through reliance on a preserved channel of emotional communication. For example, it has been suggested that individuals with unilateral frontal lobe lesions who have deficits in prosodic emotional perception could be trained to place greater emphasis on preserved facial emotional perception (Hornak, Rolls & Wade, 1996). In this way, one preserved channel of emotion communication can be used to compensate for another to further the overall goal of improving affective functioning. In a similar vein, there is anecdotal evidence that in PD, treatment of one communication channel (i.e., vocal expression) can improve another channel (i.e., facial expression). These observations and subsequent preliminary research (Speilman et al., 2003) have suggested that there may be an association between the facial and vocal emotional communication channels in PD.

Lee Silverman Voice Treatment. To our knowledge, no treatment has directly targeted several communication deficits in PD including speech, voice, and facial expression impairments. LSVT is the only treatment that targets voice and speech and also indirectly improves facial expression. LSVT is a speech treatment developed by Ramig and colleagues (Ramig et al., 1988) that has been used extensively with PD patients to improve speech and several other impaired domains affected in PD, such as swallowing, limb gesture, and limb function. Although LSVT was developed as a voice therapy to improve aspects of voice and speech, anecdotal evidence from patients and their spouses has suggested that facial expressions

improve as well (Speilman et al., 2003). LSVT has a narrow treatment focus—to maximize the loudness with which an individual produces speech to optimize phonatory effort. This treatment focus is guided by several important principles, which are important to follow carefully to produce the underlying neural plasticity necessary for the treatment to succeed. The principles include *intensity, complexity, saliency of practice, functional use,* and *timing of interventions*. These guiding principles translate into five specific tasks that are practiced during every training session (Fox et al., 2006). Details of the treatment process are included in Appendix 1. The theoretical basis for LSVT's effect on facial expression is that this therapy targets more primitive aspects of voice production (i.e., respiration and phonation), which reside within phylogenetically older brain regions that are shared with facial emotional expression.

Articulation Therapy. Articulation Therapy (ARTIC), developed by Ramig and colleagues (Spielman, Dumer, Borod, Oster, Rabin, Halpern, & Ramig, 2012), is another voice treatment that may also affect facial expression. ARTIC was designed as a control treatment to LSVT and is structured in exactly the same way as LSVT with respect to a number of factors, including number of sessions and length of treatment, patient contact with clinicians, and type and amount of feedback given by therapists. However, ARTIC therapy focuses on a different treatment target. Instead of maximizing the parameter of voice amplitude, as in LSVT, ARTIC focuses on articulatory effort. Patients are asked to repeat overarticulated vowel, consonant, and vowel consonant sounds. The effects of ARTIC on facial expression are unknown at this time. However, the theoretical basis for speculating that ARTIC voice therapy would have less of an effect on facial expression than LSVT is that it targets the highest level component of voice production (i.e., articulation) compared to LSVT which targets primitive aspects of voice

production (i.e., respiration and phonation). This high level component of voice production is necessary for producing speech, a function unique to human beings. When considered from an evolutionary perspective, it is logical that primitive aspects of voice production (e.g., phonation expressed as mating calls and other species-specific calls) found in the animal kingdom exist within older phylogenetic regions of the brain (i.e., subcortical regions) that exist in birds, non-human primates, and other animals. However, articulation, an aspect of voice production specific to human beings is controlled by the neocortex, a region of the brain unique to humans. Since ARTIC exerts its influence on the cortex, it can be expected that it would have a lesser effect on facial expression since it is not speculated to engage the subcortical network of emotional processing.

Mechanisms underlying LSVT effectiveness on facial expression.

Emotion as a system. One line of evidence connecting emotional facial and vocal communication is derived from the concept of emotion as an integrated system with several components that work in concert to produce an emotional response. This concept has been advanced by several researchers, such as Porges (Porges, 2001), who proposed the "social engagement system," which includes facial expression and vocal expression among the additional components of gaze, head turning, and listening. Similarly, Borod (1993) proposed a multicomponent framework of emotional processing, with facial and vocal (prosodic/intonational) expression representing two of five emotional communication channels in addition to lexical (verbal), gestural, and postural. Kaiser and Scherer (1998) also conceptualized facial and vocal expression as linked "components of a coordinated organism-wide response system."

There is strong empirical evidence from behavioral studies to support these theoretical claims. Studies have consistently shown positive correlations between facial and prosodic emotional expression in both healthy adult populations (e.g., Borod et al, 1985; Malatesta, Davis, & Culver, 1984; Mehrabian & Wiener, 1967) and neuropsychiatric populations (e.g., Borod et al., 1985, 1990). Given the strong association between facial and prosodic emotional expression in behavioral studies, it would be important to examine the neural underpinnings of emotion and voice to see if there might be evidence to support the behavioral data that associate voice and face in emotion.

Neural correlates of emotion. The neural correlates of emotion are widely distributed throughout the brain (Derryberry & Tucker, 1992) but there are several anatomical structures and circuits that have been implicated more specifically in emotional facial expression. These structures include the amygdala, basal ganglia, thalamus, and anterior cingulate cortex. The connectivity among these structures reveals that they either operate within neural circuits that function in a coordinated fashion or are connected neuroanatomically. The following discussion will review the circuitry among these regions.

Of the four structures identified above as important for emotional facial expression, three, the basal ganglia, thalamus, and anterior cingulate cortex, can be analyzed more meaningfully as a group instead of as separate structures. In their seminal review, Alexander, Delong, and Strick (1986) delineated five functionally segregated parallel circuits connecting the basal ganglia and the cortex. The circuits include the anterior cingulate circuit, the dorsolateral prefrontal circuit, the lateral orbitofrontal circuit, the motor circuit, and the oculomotor circuit. All five circuits have a similar structure, with each circuit receiving distinct inputs from the cortex, remaining

segregated in its path through the thalamus and projecting back onto the same cortical region (Alexander et al. 1986). More specifically, each circuit engages distinct regions of the same five areas: the cortex, thalamus, and three basal ganglia nuclei (i.e., striatum, pallidum, and substantia nigra). Although all five circuits are closed loop systems, there are open connections within the circuits from distinct anatomical regions that integrate information from functionally related brain regions.[1]

Alexander and colleagues (1986) suggest that it is more appropriate to examine the functions of basal ganglia-thalamocortical circuits than to isolate functions of specific basal ganglia nuclei. This idea is mirrored by Derryberry and Tucker (1992), who suggest that emotional processes are not circumscribed to specific areas of the brain but are highly distributed. Based on this circuitry, it is a reasonable assumption that the anterior cingulate cortex, thalamus, and basal ganglia are functionally related and are all involved in emotional processing. Indeed, there is empirical support to corroborate this claim as dysfunction in each of these structures leads to impairment in emotional facial expression.

The basal ganglia structures are intimately involved in emotional facial expression, specifically for patients with PD. Trosch and colleagues reported about a 15-year old boy with a

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[1] When these five circuits were originally described, the formulation was made primarily based on anatomical and physiological data as the functional specificity for only the motor and oculomotor circuits had been firmly established (Alexander et al., 1986). Within the past two decades, evidence from normal and psychiatric populations has shed light on the functional role of the remaining three circuits: the dorsolateral prefrontal circuit, the lateral orbitofrontal circuit, and the anterior cingulate circuit (Tekin & Cummings, 2002). These circuits have been associated with a diverse array of cognitive and affective functions, ranging from executive and memory functions to emotional processing, with the latter including affect and mood, personality, and behavior.

focal basal ganglia infarction (head of the caudate nucleus, putamen, and anterior limb of internal capsule) that resulted in impaired emotional facial expression (i.e., spontaneous expression) and preserved volitional facial expression (i.e., posed expression; Trosch et. al., 1990). Based on these findings, the authors speculated that the striatum (i.e., caudate and putamen) may have a causal role in producing the masked facies symptoms found in PD. Another recent case study reported the same clinical profile following an isolated striatocapsular infarction (Michel, Derkinderen, Laplaud, Daumas-Duport, Auffray-Calvier, & Lebouvier, 2008). However, the basal ganglia do not function in isolation but as part of larger circuits. As such, a description of all the basal ganglia-thalamocortical circuits will be presented here, with particular emphasis on the anterior cingulate circuit.

The anterior cingulate cortex (ACC) circuit is one of the most important basal ganglia-thalamocortical circuits for the production of emotional facial expression. Patients with epilepsy who have seizure foci in the ACC commonly display emotional changes such as laughing or crying during and between seizures (Devinsky, Morrell, & Vogt, 1995). Further, electrical stimulation of the ACC evokes emotional responses, such as fear, pleasure, and agitation (Meyer et al., 1973). The anterior cingulate circuit originates in the anterior cingulate cortex, Brodmann's area 24. From there, neurons project onto the ventral striatum (Selemon & Goldman-Rakic, 1985), which consists of the ventromedial caudate, ventral putamen, nucleus accumbens, and the olfactory tubercle (Mega & Cummings, 1994). The ventral striatum is also known as the “limbic striatum” (Alexander et al., 1986; Tekin & Cummings, 2002) due to the extensive projections it receives from limbic structures, including the amygdala, hippocampus, and entorhinal and perirhinal cortices (Alexander et al., 1986). A discussion of the striatum’s importance in

emotional facial expression has already been described above. From the ventral striatum, projections continue to the rostromedial globus pallidus and rostrodorsal substantia nigra (Haber, Lynd, Klein, & Groenewegen, 1990) and then onto the ventral anterior nucleus of the thalamus. As in all the circuits, the loop is closed via thalamic projections back to the origin in the anterior cingulate cortex (Giguere & Goldman-Rakic, 1988).

Dysfunction in the ACC circuit is generally associated with lack of motivation in patients. Bilateral anterior cingulate lesions cause akinetic mutism, a condition characterized by a waking state with prominent apathy, lack of motor initiative, lack of facial expression and verbalization, and reduced response to external commands. Patients with PD who have neuropathology in the subcortical components of the anterior cingulate circuit often manifest apathy (Tekin & Cummings, 2002), and though apathy is not the underlying cause of masked facies and other motor disturbances in PD, the overlap in symptomatology between PD and akinetic mutism is striking, particularly with respect to the lack of facial expression and reduced spontaneous movement.

Two additional basal ganglia-thalamocortical circuits, the dorsolateral prefrontal circuit and the orbitofrontal circuit, are less involved in facial emotional expression; however, they still have roles in emotional functioning, which will be described below. A more detailed account of these circuits can be found in Appendix B. The dorsolateral prefrontal circuit sends minor afferents to the limbic system, perhaps the anatomical mechanism by which this primarily cognitive circuit influences emotional processing. Reduced metabolic activity in the orbitofrontal circuit, specifically in the orbitofrontal cortex and caudate nucleus, has been observed in PD patients who have been diagnosed with depression (Mayberg et al., 1990). Dysfunction in this

circuit is associated with personality changes that include emotional lability and behavioral disinhibition. Patients may respond inappropriately to social cues and lack sensitivity in interpersonal interactions.

There is evidence from case studies and autopsy reports (Nothnagel, 1910) that the thalamus is crucial for the communication of spontaneous facial expression (i.e., emotional expression) but not facial expression that is regulated by cortical voluntary control (i.e., posed expression; Hopf et al., 1992; Ross & Mathiesen, 1998). Ross and Mathiesen provided a report about a 50-year old man with a primary lymphoma in the left thalamus who showed significant weakness of the lower right side of the face during emotional facial expression (i.e., spontaneous smiling) with preserved functioning for posed smiling. Similarly, Hopf and colleagues reported about seven patients with lesions resulting in impairment of emotional facial expression, four of whom sustained lesions in the thalamus. Of note is the fact that 6 of the 7 patients sustained lesions to one or more structures within the basal-ganglia thalamocortical circuitry.

The one structure that is often associated with emotion, specifically the expression of fear, is the amygdala (LeDoux, 2003). Work of LeDoux, corroborated by many others, has identified the amygdala as a critical structure in conditioned fear responses and places the amygdala as a central structure responsible for emotional processing. The amygdala sends projections to the brainstem to direct behavioral expressions of fear and sends projections to the cortex to facilitate the subjective experience of fear (LeDoux, 2003). The amygdala's projections to the brainstem synapse onto the trigeminal (Barnett, Evans, Suq, Perlman, & Casse, 1995) and facial cranial nerves (CN), perhaps, providing the emotional input necessary for producing facial expression (CN V) and affecting sensory innervation to the face, mouth, and tongue (CN VII).

Neural correlates of emotion: Cortical influences. Whereas subcortical structures are critically important for emotional processing, cortical lesions, especially frontal lesions, have also been shown to adversely affect production of spontaneous and/or posed facial expression (Blair, 2003; Borod et al., 1985, 1986, 1990; Kazandjian, Borod, & Brickman, 2007; Kolb & Milner, 1981; Laplane, Talairach, Meininger, Bancaud, & Orgogozo, 1977; Weddell, 1994). This is consistent with the argument that emotional processing is not isolated to a discrete region but is distributed throughout the brain (especially within the basal ganglia-thalamocortical circuitry). There is also a long-standing tradition in the field of emotion research that considers the right hemisphere as dominant in producing facial emotional expressions (Borod et al., 1986; Feyereisen, 1986; for reviews, see Borod, Bloom, Brickman, Nakhutina, & Curko, 2002; Borod, Zgaljardic, Tabert, & Koff, 2001).

Cortical control of facial expression is evidenced through several reports of frontal lesions resulting in disordered facial expression. Laplane and colleagues (1977) documented a reduction in facial motor activity on the contralateral hemiface, particularly during posed facial expression associated with damage to the supplementary motor area. Kolb and Milner (1981) examined the effects of frontal, temporal, parietal, and occipital lesions on patients' ability to produce complex facial movements through imitation. Though participants were not asked to imitate emotional facial expressions, many of the facial movements included in the study were clearly necessary for the production of emotional facial expressions. For example, two of the movements in the protocol were raised brows and knitted brows, movements involved in the production of surprise and anger, respectively. The results of the study were striking. Among all groups of patients, the only one that showed significant impairment in producing facial

movements was the one that had sustained injury to the frontal lobe. The participants in that group exhibited significantly higher numbers of intrusions, omissions, sequencing errors, and total facial movement errors when compared to other patient populations. However, patients with temporal, parietal, and occipital lesions did not differ in their ability to produce facial movements when compared to control participants. Similarly, Weddell (1994) evaluated the effects of anterior, posterior, thalamic, and hypothalamic lesions on posed and spontaneous facial expression and found impoverished facial expressions only in individuals with anterior (i.e., frontal) lesions. Laterality of the anterior lesions was not a significant predictor of performance; however, a there was a trend for patients with right hemisphere damage to produce fewer emotional expressions than those with left hemisphere damage.

Neural correlates of voice production. One of the primary brain regions associated with vocalization is the periadqueductal gray (PAG). There is a great deal of evidence that the PAG is critical for vocalization; its stimulation produces species-specific calls in many animals including in rats, cats, dogs, guinea pigs, bats, amphibians, reptiles, and several types of monkeys (Jurgens & Zwirner, 1996; Ploog, 1981). Despite its critical role in vocal production, a study that used a pharmacological blockade of the PAG in the squirrel monkey showed that the PAG was not necessary for all vocalizations. When the PAG was blocked, non-verbal emotional vocalizations were abolished but vocal fold control necessary for speech was preserved (Jurgens & Zwirner, 1996). This study provided evidence for two distinct pathways supporting vocal control, one for speech and one for non-verbal emotional vocalizations.

The neural organizational principles of voice production are strikingly similar to those of facial expression. Just as there are two distinct neural pathways mediating different types of

facial expression, posed and spontaneous, there are two distinct neural pathways responsible for vocal control, one for speech and one for non-verbal emotional vocalizations. In both the facial and vocal systems, volitional behaviors (e.g., posed expression and intentional speech) are mediated by neocortical pathways whereas the automatic behaviors (e.g., spontaneous expression and non-verbal vocalizations) are emotional in nature, and recruit subcortical and limbic pathways.

The neocortical pathway supporting learned vocal patterns (i.e., speech) starts in the cortex, specifically in the larynx area of the facial motor cortex and controls vocal fold movement and speech. The limbic pathway supporting non-verbal emotional vocalizations begins in the anterior cingulate cortex (ACC), projects to the periaqueductal grey (PAG) of the midbrain, and eventually synapses on the laryngeal motorneurons via several nuclei (Jurgens & Zwirner, 1996). Devinsky and colleagues (1995) proposed that the ACC can be subdivided into affective and cognitive components and that the vocalization region is part of the affect component. They proposed that vocalizations evoked from this region have emotional content and express internal emotional states.

Neural correlates underlying LSVT effectiveness. Neural substrates of emotion and voice are described above. It is plausible that LSVT exerts its influence on facial expression by affecting some of these regions. However, many of the subcortical regions (e.g., basal ganglia) that are important in emotional processing and voice production are compromised in PD. Despite this neuropathology, LSVT is efficacious. What mechanism(s) could account for the efficacy of this treatment?

Functional neural reorganization appears to be key in mediating the positive behavioral

outcomes associated with different kinds of interventions in PD (Fox et al., 2006). Previous imaging studies of motor-limb function in PD have identified several regions with abnormal functioning pre-treatment that normalized following successful treatment with pharmacological and neurosurgical interventions (Samuel, et al., 1997; Rascol et al., 1994). These regions include cortical premotor areas, primarily the supplementary motor area (SMA), and the cerebellum, among others. Whereas pharmacological and neurosurgical interventions have been successful for motor-limb symptoms, the only treatment that has proved consistently efficacious for speech and voice symptoms in Parkinson's patients is LSVT (Schulz & Grant, 2000). Liotti and colleagues (2003) examined whether the voice improvements that result from LSVT occur due to the same type of functional reorganization in the brain seen following other interventions. Using positron emission tomography (PET), they examined phonation and speech before and after LSVT in PD patients with hypophonia compared to control participants. This study was the first to examine the neural mechanisms underlying successful LSVT treatment. Beyond this primary goal, they also sought to show that the treatment-related gains following LSVT were not simply the result of increased loudness, but rather the product of functional reorganization in the brain resulting from extended loudness practice.

Participants were tested on three behavioral tasks: overt paragraph reading, sustained phonation of the phoneme /ah/, and a control condition where participants were asked to rest with their eyes closed. PD patients were scanned before and after a standard course of LSVT consisting of 16 sessions over a one-month period, while control participants participated in the paragraph reading and rest conditions only. For normal controls, activations were seen primarily in the left posterior cerebellum, and notably, no SMA activation was detected.

For PD patients two important findings were uncovered about the mechanisms of action of LSVT. First, neural activation patterns revealed a shift from primarily cortical activation before treatment to primarily subcortical activation after treatment. Second, pre-treatment activations were mostly bilateral, whereas post-treatment activations were overwhelmingly clustered to the right hemisphere.

Many regions of activation were observed in both pre-treatment and post-treatment, however, for the purpose of clarity, only activations unique to each time point will be summarized. For PD patients, pre-treatment activations were observed bilaterally in the motor strip (M1 mouth region) and premotor cortex (right SMA and bilateral inferior lateral premotor cortex, including Broca's area 44). Post-treatment activations were primarily in portions of the left cerebellum, right anterior insula, right head of the caudate, right putamen, and right and left dorsolateral prefrontal cortex (DLPFC). Though discrete right hemisphere activations were seen pre-treatment (primarily right SMA), the overwhelming right hemisphere activations post-treatment were consistent with the authors' conclusion that right hemisphere activations in post-treatment results point to "evidence of a site of action of voice treatment on substrates of emotion regulation and representation" (Liotti et al., 2003, pp. 439) since the right hemisphere is specialized for emotional processing. This interpretation is corroborated by the post-treatment activation in right anterior insula, a region that receives significant input from the amygdala and is associated predominantly with emotion.

It is significant to note that the neural pattern of activation in PD patients observed after LSVT was similar to the activation seen in the normal controls at baseline (primarily cortical activation). This result was interpreted by the authors as reflecting a treatment-related

normalization of neural activation. In PD, subcortical circuits are compromised, and, therefore, it might be expected that pre-treatment activations would be primarily cortical due to natural neural reorganization that occurs following trauma to discrete regions in the brain (Johansson, 2000). After effective voice treatment, however, many subcortical structures (i.e., mainly basal ganglia) were engaged in this task, possibly reflecting experience-dependent (i.e., repetitive practice during treatment) neural reorganization. The authors argued that this may reflect recruitment of other intact frontal-subcortical circuits (Alexander et al., 1986) and, thus, normalization of neural activation as a result of treatment.

Although pharmacological, surgical, and behavioral treatments of movement-related symptoms in PD all result in functional reorganization in the brain, an important difference exists in the mechanism by which treatments exert their effect. In the case of LSVT, reorganization is an experience-dependent process whereas in pharmacological and surgical treatments, neural plasticity is not experience-dependent. The importance of experience-dependent neural plasticity is also evidenced in basic animal studies showing morphological and neurophysiological changes following extended exercise (Van Praag, Christie, Sejnowski, & Gage, 1999; Van Praag, Kempermann, & Gage, 1999).

Facial and vocal communication channel connectivity.

Evidence from cranial nerve connectivity. The connectivity between facial and vocal channels for emotional communication is revealed upon closer examination of the neural innervation of the face as well as their shared neural substrates. The brain innervates the muscles of the face through motor neurons whose circuits have two parts, upper and lower (Rinn, 1984), which collectively make up the corticospinal tract, sometimes referred to as the pyramidal tract

(Kandel et al., 2000). Upper motor neurons carry motor impulses from the motor strip in the cortex to the brain stem or spinal cord, where they synapse onto lower motor neurons, which then carry the impulses to muscle (Blumenfeld, 2002). The muscles of facial expression are innervated by the lower motor neuron tract (i.e., the seventh cranial nerve, also referred to as the facial nerve; Rinn, 1984). Once the facial nerve enters the face, it separates into five main branches that are responsible for most facial movements (Rinn, 1984). Thus, different branches innervate the lower, middle, and upper portions of the face (Rinn, 1984). Although the facial nerve is not the only cranial nerve that innervates the face, it is the only one primarily devoted to manipulation of the facial musculature to produce facial expression (Blumenfeld, 2002). It also has a role in tear production, salivation, taste, and other somatosensory functions. The other cranial nerve that innervates the face is the fifth or trigeminal nerve, which has the role of innervating the jaw and facilitating chewing, as well as providing sensory innervation to the face, mouth, and tongue (Blumenfeld, 2002). Although the primary functions of these two cranial nerves are distinct, there is evidence of connectivity between them. Both anatomical (Carpenter & Hanna, 1961) and electrophysiological (Tanaka, Yu, & Kitai, 1971) studies have demonstrated direct connections between the trigeminal and facial nuclei. Early anatomical studies in cats indicated that a few fibers from the spinal trigeminal nucleus project onto the facial nucleus bilaterally (Carpenter & Hanna, 1961). Furthermore, stimulation of cat peripheral trigeminal afferents leads to activation of facial motor neurons (Tanaka et al., 1971). The connections between the facial and trigeminal nerves may partially underlie the connectivity between the facial and vocal emotional communication channels. These connections may be one of the mechanisms by which treatment for a voice disorder, that is, the Lee Silverman Voice Treatment,

affects facial expressivity.

Evidence from neural substrate overlap. The association between facial and vocal communication channels also likely results from common cortical and subcortical regions responsible for their regulation. There is evidence that the anterior cingulate cortex (Devinsky et al, 1995), periaqueductal grey, thalamus, and basal ganglia regulate both facial and vocal expression. The connection between face and voice, and thus the mechanism of action in voice treatment, specifically LSVT, on facial expressivity appears to occur at several levels, including cortical, subcortical, and cranial nerve innervations of the face. There is also evidence for facial and vocal system connectivity from observations of neural coupling between orofacial muscles *and* laryngeal and respiratory systems (McClean & Tasko, 2002), such that facial muscles move in a coordinated fashion with the production of speech sounds.

Aims and Hypotheses

Based on the evidence outlined above, we expected that LSVT therapy, which targets basic and primitive aspects of voice production (i.e., respiration and phonation), would improve facial expression in PD by activating the phylogenetically older subcortical regions that vocal and facial expression share. We further expect that our control treatment, ARTIC, which targets more advanced aspects of voice production (i.e., articulation), would improve facial expression to a lesser extent than LSVT because it activates newer cortical regions not associated with emotional processing. Based on these assumptions, we outlined the following aims and hypotheses for the study:

Aim 1. We proposed to examine emotional facial expression and non-emotional facial mobility of PD patients in comparison to NCs. Analyses were conducted on

pre-treatment data only, and the PD patient group included all three PD subject groups participating in the study. Analyzing pre-treatment baseline data only will allow us to examine facial expression across groups irrespective of treatment effects.

  - **Hypothesis 1:** We hypothesize that all 3 PD patient groups will exhibit reduced facial emotional expression (i.e., EF, EV, and EI) and facial mobility (FM) as compared to NCs (see Methods for detailed definitions of DVs). This is predicted based on substantial evidence from the literature showing facial expression impairments in PD (e.g., Borod et al., 1990; Brozgold et al., 1998); Buck & Duffy, 1980; Katsikitis & Pilowsky, 1988, 1991; Simons et al., 2004; Smith et al., 1996).
  - **Hypothesis 2:** Based on previous empirical findings, we hypothesize that PD patients will display reduced EF specifically in the happy monologues (e.g., Pitcairn et al., 1990) and increased EF specifically in the sad monologues (e.g., Brozgold et al., 1998).

- **Aim 2**: Our second aim was to compare potential changes in facial emotional expression (i.e., EF, EV, and EI) and facial movement (i.e., FM) over two time points, in our four groups (i.e., LSVT, ARTIC, no treatment PD, and NC).
  - **Hypothesis 1**: LSVT will differentially improve EF, EV, and EI as compared to ARTIC, no treatment PD, and NC. This hypothesis is made based on preliminary findings showing improvements in aspects of facial expression following LSVT (Spielman et al., 2004). From a theoretical

perspective, this hypothesis emanates from the fact that LSVT focuses on respiration, a more primitive or basic aspect of speech that relies on older subcortical structures within the basal ganglia. LSVT affects facial expression because it engages the basal ganglia, a set of structures involved in both emotional processing and basic aspects of voice production.

  - **Hypothesis 2:** ARTIC would improve EF, EV, and EI as compared subject groups that received no treatment (i.e., NC group and no treatment PD group), but to a lesser degree than LSVT. From a theoretical perspective, this hypothesis was offered because ARTIC is a voice therapy that focuses on articulation, a higher order level of vocal processing required for speech production that relies on cortical control. Since ARTIC exerts its influence on the cortex, it was expected that it would have a lesser effect on facial expression since it does not engage the subcortical networks involved in emotional processing.

- **Aim 3**: Our third aim focused on the 3 parameters of emotional expression: EF, EV, and EI. For this aim, we proposed to examine how the voice treatments (i.e., LSVT and ARTIC) specifically affected each of the facial expression parameters (EF, EV, and EI) and facial movement (FM). As these parameters are a novel way of examining facial expression, this analysis will be done on an exploratory basis with no *a priori* hypotheses proposed.
- **Aim 4:** Our fourth aim was to examine the relationships among the DVs (EF, EV,

and EI). Since it is the first time these three variables have been examined in this patient population, it would be of interest to understand the relationships among them.

- **Hypothesis 1**: It is hypothesized that the four DVs will have some degree of positive association.

CHAPTER II.

Methods

Participants

Two sets of participants were involved in this study: posers and raters.

Posers. The first set of participants, the posers, consisted of 56 PD patients and normal healthy controls from the laboratory of Dr. Lorraine Ramig at the University of Colorado-Boulder. A subset of participants from the poser group received voice treatments and was tested at the University of Colorado-Boulder. Four groups of posers were examined, PD patients receiving LSVT (LSVT), PD patients receiving an articulation therapy (ARTIC), PD patients not receiving any treatment (no treatment PD) and demographically matched healthy controls (NC). For participants with PD, illness severity was assessed using the Hoehn and Yahr scale (Hoehn & Yahr, 1967), which is a 5-point scale indicating early (i.e., Stage 1) to late (i.e., Stage 5) stages of the disease. The four groups of posers were recruited from the Denver, Colorado area through physician referrals, support groups, and senior centers. Each participant was paid $10 for undergoing the screening process and $30 for participating in the study. Each participant signed an informed consent form approved by the Colorado Multiple Institutional Review Board at the University of Colorado.

LSVT (LSVT; Ramig et al., 1995) is a voice treatment that primarily focuses on the amplitude of speech, that is, on maximizing the loudness with which individuals produce speech. The standardized treatment includes 16 sessions and lasts 4 weeks. The fundamental exercise in LSVT involves patients saying “ah” loudly for as long as they can. Patients were later instructed to sustain “ah” for 5 seconds in the highest and lowest pitch that they could produce. As patients

progressed through treatment, they were first asked to use their loud voice to produce words or phrases in Week 1, to sentences in Week 2, to reading paragraphs in Week 3, and finally, to engaging in conversation during Week 4. LSVT also emphasizes the carry-over of skills learned in the therapy to everyday life. Treatment outcomes were measured by several voice criteria, although the most important measure was of vocal loudness, measured as sound pressure level (Sapir et al., 2007).

ARTIC is the control voice treatment that focuses on articulatory effort (Speilman et al., 2012). ARTIC treatment was designed to have the same structure as LSVT, that is, the treatment is completed in 16 sessions over the course of 4 weeks. The tasks comprising this treatment include many repetitions of overarticulated consonant combinations (e.g., /t-k/ and /n-g/), consonant vowels (e.g., "pa," "ta," and "ka"), and consonant vowel consonants (e.g., "tat" and "kak"). As in the LSVT treatment, patients produced words, phrases, sentences, and paragraphs, and engaged in conversation while focusing on the enunciation of their words as opposed to the loudness of their speech as done in LSVT. The same treatment outcomes were used following ARTIC and LSVT.

The following inclusion criteria were used to admit PD posers and healthy NCs into the study: age range between 45-85; PD severity within Stages I-IV; absence of dementia (as measured by a Mini-Mental Status Examination score below 27; Folstein, Folstein, & McHugh, 1975); and any level (mild, moderate, or severe) of a speech or voice disorder. Exclusion criteria for the study consisted of the following: severe depression as measured by a Beck Depression Inventory-II score above 25 (Beck et al, 1996), dementia, cancer of the head or neck, and history of surgery or pathology in any of the following systems: gastrointestinal, laryngeal, neurological

(excluding PD symptoms and treatment), or speech-language. History of pathology or disease was evaluated by specialists in each of the areas listed above, including otolaryngologists, neurologists, and speech-language pathologists. Additional exclusionary criteria included participation in any intensive speech treatment within the last 2 years, history of cigarette smoking within the last 4 years due to effects of smoking on aspects of voice production, severe temporomandibular joint (TMJ) disorder, or pregnancy. An audiological screening was conducted to confirm that none of the participants had hearing loss that exceeded expectations based on age. Demographic information about the posers is displayed in Table 1.

Raters. The second set of participants was raters. Eighteen undergraduate and graduate students were recruited from Queens College of the City University of New York to serve as raters. Raters were recruited in three cohorts, with 6 raters per cohort (50% female), using flyers posted throughout the Queens College campus. Raters were the individuals who rated videos of facial expressions obtained from the posers. This research protocol and rater consent forms were approved by the Queens College Institutional Review Board. Raters were reimbursed at the rate of $9/hour for attendance at training sessions and rating sessions. Inclusion criteria for raters in this study were Caucasian individuals between the ages of 20-35 who were right-handed and native English-speakers. Native English-speakers were defined as individuals who learned to speak English by the age 7. Handedness was assessed using verbal report provided by the raters. Caucasian individuals were recruited due to the fact that the posers were almost exclusively Caucasian because of the geographical area (i.e., Boulder, Colorado) in which the experimental data were collected. Of note, recent literature has demonstrated an effect of ethnicity on the perception of emotional expressions and has labeled this effect as the “in-group advantage”

(Elfenbein, Beaupre, Levesque, & Hess, 2007). This has been described in the literature as follows, "individuals can more easily and accurately understand emotional expressions originating from members of their own cultural groups rather than expressions originating from members of a different cultural group" (Elfenbein et al., 2007). Therefore, to ensure the accuracy and validity of the facial ratings, we recruited men and women who were Caucasian. Exclusionary criteria for the raters included any reported history of neurological or psychiatric disorder, substance abuse, learning disability, and/or head trauma.

Procedures

Poser Procedures. The posers were brought into a sound-resistant booth (Industrial Acoustics Company) and were seated in a dental examining chair with a headrest. They generated emotional and non-emotional monologues that were videotaped and sent to Dr. Joan Borod's Emotion Laboratory at Queens College. The video data were captured using a Canon XL1S mini DV video-camera. The camera was positioned approximately nine feet away from the poser, and the video images consisted of the neck, face, and head of the poser. Posers produced emotional and non-emotional monologues describing salient happy, sad, angry, and neutral life events, using procedures from the New York Emotion Battery (NYEB; Montreys & Borod, 1998; Borod, Welkowitz, & Obler, 1992). This process was not automated, and an experimenter was always present to give posers instructions and to video record the session. Posers were given the following paraphrased instructions from the NYEB for producing the emotional monologues: "please speak for approximately three 90-second intervals describing experiences when you felt intense sadness, happiness, or anger." In addition to the 3 emotional monologues, posers were asked to recount a non-emotional experience (i.e., going to the

supermarket). A trip to the supermarket was chosen for the neutral monologue due to its assumed emotional neutrality.

All posers produced these monologues at two time points; for the two treatment groups (i.e., LSVT and ARTIC), these procedures took place before and after treatment. For the two non-treatment poser groups (i.e., NCs and no treatment PDs), the two sets of monologues were performed with the same time interval between them as was the case for the treatment groups, that is, approximately 4-5 weeks.

Poser voice treatment procedures. Three professional speech-language pathologists served as the treating clinicians for both ARTIC and LSVT. All three clinicians were expert level therapists, defined as those clinicians who are judged as experienced enough to train novice clinicians to perform the therapy by the creators of the therapy. All treating clinicians were female, and they treated PD patients in both the ARTIC and LSVT poser groups. Each PD patient received therapy for all 16 sessions from only one treating clinician. The treating clinician was never present during the pre-treatment or post-treatment video recording of the experimental monologues; the only person with whom each poser interacted during the video recording sessions was a lab technician who was not otherwise involved in the study.

Rater Procedures.

Rater cohorts. Experimental poser video data were divided into three cohorts, and each cohort was rated fully before the next set of cohort ratings began. Each cohort consisted of four poser groups: LSVT, ARTIC, no treatment PD, and NC. The posers in each group were carefully selected, where poser demographics, including age, education, degree of illness severity for the PDs (assessed according to the Hoehn and Yahr [1967] stage), and gender, were matched as

closely as possible across the four poser groups and across the three cohorts. In each poser cohort, 6 raters rated all of the data from the PD patients and controls (poser numbers varied by cohort), including all four variables (FM, EF, EV, and EI), and both time points (pre-treatment and post-treatment). For each cohort, a new set of 6 raters was recruited and admitted into the study according to the inclusion criteria described above. For each cohort, raters rated the variables in the same order, with FM first, followed by EF, EV, and EI. FM was always rated first because it represented facial activity devoid of emotional content and therefore had to be introduced before any emotion ratings took place.

Training procedures. The training procedures used in the present study are based on the rater training system developed by Canino, Borod, Madigan, Tabert, and Schmidt (1999). The experiment consisted of two phases, the training phase and the experimental rating phase. The training phase was conducted with all raters from a given cohort present to facilitate the interactive nature of the training. Once the raters for Cohort I completed the study, Cohort II raters, followed by Cohort III raters, were trained. Each training session included three parts: *exemplar, conferencing, and inter-rater reliability phases.* Each of these training phases will be described in detail below. The goal of each training session was to train the raters to reliably rate each given variable on a 7-point Likert scale, from minimal (i.e., a score of 1) to maximal (i.e., a score of 7). For example, for the non-emotional facial mobility variable, raters were trained to rate a face that is not moving at all with a rating of 1 and a highly mobile face with a rating of 7. The goal of the experimental rating phase was to collect experimental data on the following variables: facial mobility (FM), emotional frequency (EF), emotional variability (EV), and emotional intensity (EI). During the training phase, raters received extensive training on how to

make the facial ratings. To that end, all raters first participated in a group training session for each variable of interest performed with as many raters as possible present and with one to two experimenters leading the session. If all six raters were not able to attend the same training session due to scheduling conflicts, additional training sessions were held to accommodate all the raters. When this scenario occurred, raters' responses from previous training sessions were shared with the raters that were present to simulate a single group session. Training sessions were both instructive and interactive and assessed the reliability of the raters as a group. For each group of raters, four training sessions were held, one for each dependent variable that was examined: FM, EF, EV, and EI.

Each training session focused on one specific variable, but the following elements were reviewed in all training sessions. Raters were instructed to disregard head movements and movements that appeared to be random (e.g., excessive blinking, head shaking, and body tremor) and to attend only to the movements of facial muscles. They were also asked to disregard an individual's unique facial features (e.g., age wrinkles, which could be interpreted as movement). Raters were instructed to attend to a number of parts of the face and the possible movements that can occur, that is, forehead can be wrinkled and vertical lines can be seen between the eyes; eyebrows may be raised, lowered, and drawn together; upper and lower eyelids may be open or closed to varying degrees; eye balls may display shifting of gaze; nose (i.e., sides and bridge) may be wrinkled; cheeks may be flattened, stretched, and raised and rounded; naso-labial fold may have different degrees of depth; mouth may be open or closed to varying degrees; corners of the mouth may be drawn back and up; jaw may be lowered or moved from side to side; and the lips may be non-contracted or at rest, contracted, or pressed. Facial features of common

emotional expressions (i.e., happiness, interest, surprise, sadness, disgust, anger, and fear) will be described. For the variables that contain emotional elements, raters were instructed that emotional expressions can occur in full form (e.g., all parts of the face contribute to an expression of happiness) or in partial form (e.g., only the eyes express a particular emotion without the accompanying expression in the mouth or eyebrow regions).

The following are definitions for each of the dependent variables in the study. *FM* refers to facial mobility or how much movement the face produces. Mobility is defined here as the amount and extent of muscular movement of the face that is not emotional in nature. *EF* is defined as the frequency of emotional expressions observed during each video segment. Frequency of emotion can refer to the number of distinct emotional expressions observed (i.e., a smile indicating happiness followed by trembling lips indicating sadness) or the number of the same emotional expression repeated throughout the segment (i.e., several smiles separated by neutral expressions). *EV* is defined as the variability of facial expressions observed in each video segment. EV is the number of different emotional expressions observed in a segment (e.g., sadness followed by anger followed by surprise) or the number of changes *within* the expression of one emotion (e.g., laugh followed by a smile followed by another laugh). It is emphasized to raters that one emotional expression sustained throughout the segment would receive a low rating with respect to variability. *EI* refers to the intensity or different degrees of amplitude with which the emotional expressions are conveyed. Raters were instructed to evaluate intensity only for the portions of the segment that were emotional, even if emotion was conveyed in a very small part of the segment.

Training materials.

Rating scale. At the beginning of each training session, raters received a training packet that included a copy of the Likert scale, definitions of the variables, and specific guidelines for recognizing facial muscle movements associated with specific emotions. The Likert rating scale ranged from "1," minimal or no EF, EI, EV, or FM, depending on the variable being rated, to a score of "7," maximal EF, EI, or EV. Each rater had access to this packet as a reference during the actual rating of experimental data.

Baseline faces. Raters were also given a packet of baseline faces, specific for each poser, that they were able to reference at any time during the rating of the experimental data. These baseline faces included a color printed picture of an emotionally neutral face for each poser from the experimental data. The baseline faces were created by two experimenters observing a random selection of monologues produced by each poser, frame by frame, and selecting the most representative and emotionally neutral snapshot of each poser participant. The rationale for creating the baseline faces was that many participants have facial age features or Parkinsonian facial features that are present even at rest (i.e., at baseline) that do not represent true emotional expression. Therefore, it was be important for raters to keep the baseline face for each poser in mind when making their ratings. The following sections will include details of the three phases of each training session, based on procedures developed in Dr. Borod's laboratory (Canino et al., 1999): *exemplar, conferencing, and interrater reliability.*

Exemplar training phase. The exemplar phase was designed to be instructional. During this first phase of training, the experimenter introduced and described the 7-point Likert scale used to make each rating. The experimenter presented a series of 15-second video clips showing

individuals with PD and healthy adults recounting emotional and non-emotional monologues. For each video clip, the experimenter provided the rating for the clip and gave specific reasons for each rating. For example, for the FM variable, a rating of "1" was given when no facial movement was observable. A total of 14 exemplar video clips were shown, with two examples for every point on the Likert scale. The clips were presented in increasing order, starting with a rating of "1" and ending with a rating of "7."

Preparation of training segments. Exemplar video segments (14 for each variable) were carefully selected using quasi-random sampling, thus ensuring that exemplars were balanced with respect to emotion, gender, and poser group. They were selected by 3 to 5 lab members before the study began. Lab members were blind to information about the poser in each segment. After viewing each segment, each lab member independently made his or her rating, and segments were only chosen as exemplars if all lab members agreed on the rating, within 1 point of each other. When small disparities occurred, the final rating for the segment was based on a majority vote. The same procedure was used to select conferencing training segments.

Conferencing training phase. The conferencing phase was designed to be interactive. During this second phase of training, the experimenter presented 12 quasi-randomly selected segments, and each rater independently rated the video segment according to the 7-point Likert scale. The experimenter explained that the goal of the ratings during this phase of training was for the raters to reach a consensus. After the raters independently made their ratings, each rater's rating was shared with the group so that the raters would be exposed to others' opinions about the same segment. The experimenter then determined if the raters achieved a sufficient level of consensus in their rating of the segment, defined as variability within two points of the scale

across all raters. If sufficient consensus was not reached, the experimenter led a discussion and tried to determine whether outlier ratings represented an idiosyncratic rating approach. Each rater was asked to explain why he or she gave their rating, and the experimenter reiterated that the goal of the training was for the raters to make ratings in a meaningfully predefined way and to reach consensus. After the discussion, the segment was viewed again, and each rater independently made a second rating of the segment.

Inter-rater reliability training phase. The last phase of training, that is, the inter-rater reliability phase, was designed to assess the reliability of the raters as a group. Raters were presented with 40 randomly selected segments, and each rater made his or her rating independently. After all ratings were made, the experimenters calculated the inter-rater reliability for the group using average one-way random Intra-Class Correlation (ICC; Shrout & Fleiss, 1979). If there was at least 75% agreement among raters' responses (ICC $\geq$.75), raters were qualified to begin rating the experimental data.

Experimental rating phase. Following each training session, raters performed ratings independently over the course of 2-3 weeks. All ratings were performed in the same room on one of two Dell computers. However, each rater used the same computer throughout the study. All rating sessions were supervised by a lab member to ensure that distractions were eliminated and that raters maintained adequate effort and attention to the ratings throughout the sessions. The average length of a rating session was approximately 2 hours.

Raters viewed short video clips of faces of the four poser groups (LSVT, ARTIC, no tx PD, and NCs) to make their ratings. The video clips were 15 seconds in duration. These 15-second clips were extracted from emotional and non-emotional monologues of approximately

1.5-3 minutes in length that were videotaped at two testing times, as described above. The monologues were segmented into 15-second clips using Corel Video Studio ProX 3, and those clips were presented to raters using a video-stimulus presentation software programmed in Microsoft Access v. 2007, designed specifically for this study. Video clips were presented to each rater randomly, such that each rater rated the series of video clips for each variable in a different order than each of the other raters. This procedure was designed to prevent order effects with respect to rater group, poser group, monologue type (i.e., emotion), and gender. Video clips were presented to the raters without audio to prevent the influence of extraneous variables (e.g., the prosodic or lexical/semantic characteristics of the monologues) on raters' ratings.

After viewing each 15-second video clip, raters made their rating of 1-7 on the computer screen using the mouse. Raters had the opportunity to view each segment up to 3 times, with no time limit imposed for each viewing. When raters made their rating, the Access software program automatically saved the response into a database, and these data were analyzed at a later point. When the ratings for a particular variable were completed by all 6 raters, the training session for the next variable took place.

Data Analytic Plan

Normality assessment. Before conducting statistical analyses, distributions for each variable in the study were examined and checked for normality using the Shapiro Wilk's test. Variables that deviated significantly from the normal curve, as measured by a significant Shapiro Wilk's test, were arithmetically transformed to conform to a normal distribution.

Poser group equivalence assessment. To ensure that the 4 Poser groups were equivalent with respect to age, education, and stage of disease, a one-way ANOVA, with Poser group (4) as

the between-subjects variable, was performed for each of the following demographic variables: age, education, gender, pre-treatment BDI-II score, pre-treatment MMSE score, stage of PD, and time since diagnosis of PD. For any statistically significant differences, post-hoc comparisons using the Tukey HSD test were performed. Group equivalence with respect to gender was analyzed using chi square.

Reliability analyses. Two sets of reliability analyses were conducted: one set for the training sessions and one for the experimental data. During training sessions, inter-rater reliability was assessed using intra-class correlation (Shrout & Fleiss, 1979). Raters needed to reach a reliability of ICC $\geq .75$ before they proceed to rating the experimental data. After experimental ratings had been made, reliability analyses will be conducted for each variable separately (i.e., FM, EI, EF, and EV) and for every cohort (i.e., I, II, and III), yielding twelve ICC coefficients for the experimental ratings.

Aim 1. For Aim 1, we examined facial expression and facial mobility of PD patients in comparison to NCs using the methodology and rating parameters described in the Methods section. We hypothesized that all 3 PD patient groups included in the study would exhibit reduced facial expression (i.e., EF, EV, and EI) as compared to NCs. Our second hypothesis was that PD patients would display reduced EF in the happy monologues, based on work documenting a reduced number of smiles in PD patients compared to controls (Pitcairn et al., 1990), and increased EF specifically in the sad monologues (e.g., Brozgold et al., 1998).

The statistical approach that was used to examine this aim was to conduct a two-way repeated measures analysis of covariance (ANCOVA) (2 x 4), with Poser Group (PD and NC), and Emotion (Happy, Sad, Angry, and Neutral) as variables. If results supported Hypothesis 1

(i.e., all 3 PD patient groups included in the study exhibited reduced facial expression), we expected to find a significant main effect for Poser Group. If the results supported Hypothesis 2 (i.e., reduced EF in the happy monologues and increased EF in the sad monologues for PD patients), we expected to find a significant Poser Group X Emotion interaction. This analysis was conducted on the baseline data only (i.e., on time-point 1) to isolate the facial expression characteristics of PD and NC groups irrespective of treatment effects. The PD Poser Group consisted of the three PD groups in our sample: LSVT, ARTIC, and no treatment PD. Poser Group was the between-subjects variable, and Emotion was the within-subjects variable. This ANOVA was repeated three times for each of the facial expression rating parameters: EF, EV, and EI, as well as for FM.

Aim 2. For Aim 2, we compared potential changes in facial emotional expression (i.e., EF, EV, and EI) and facial movement (i.e., FM) over two time points in our four groups (i.e., LSVT, ARTIC, no treatment PD, and NC). Our first hypothesis was that EF, EV, and EI would be most improved by LSVT as compared to the ARTIC treatment and as compared to the no treatment PD and NC groups. The second hypothesis was that EF, EV, and EI would improve following ARTIC treatment but to a lesser degree than after LSVT.

The statistical approach that was used to examine this aim was to conduct a three-way (4 x 2 x 4) repeated-measures ANCOVA, with Poser Group (LSVT, ARTIC, no treatment PD, and NC) as the between-subjects variable, and Time (pre-treatment and post-treatment) and Emotion (Happy, Sad, Angry, and Neutral) as within-subjects variables. If the results showed that Hypotheses 1 and 2 were supported (i.e., facial expression would be most improved after LSVT, followed by ARTIC), we expected to find a significant Poser Group X Time interaction. This 3-

way ANOVA was conducted three times for each of the facial expression rating variables (EF, EV, and EI) and for FM.

Aim 3. In Aim 3, we focused on the 3 parameters of emotional expression: EF, EV, and EI. In this aim, we proposed to examine how the voice treatments (i.e., LSVT and ARTIC) affected each of the facial emotional expression parameters (i.e., EF, EV, and EI) and facial movement (i.e., FM). As these parameters were a novel way of examining facial expression, this analysis was done on an exploratory basis.

The statistical approach that was used to examine this aim was to conduct a four-way repeated-measures ANCOVA, with Poser Group as the between-subjects variable (LSVT, ARTIC, no treatment PD, and NC), and Time (Pre-treatment and Post-treatment), Emotion (Happy, Sad, Angry, and Neutral), and Facial Rating Variable (EV, EI, and EF) as the within-subjects variables.

Aim 4. For Aim 4, we proposed to examine the relationship between the DVs. Since it was the first time these four variables were examined in this patient population, it was of interest to understand the relationship among them. It was hypothesized that the four DVs would have some degree of positive association.

Two statistical approaches were employed to address this hypothesis. The first approach involved calculating Pearson product-moment correlation coefficients to create a correlation matrix showing the relationships among all DVs. The second approach was to conduct a 3-way repeated-measures ANCOVA, with Gender as the between-subjects variable, and the Facial Rating Variable (EI, EF, and EV) and Emotion (Happy, Sad, Angry, and Neutral) as the within-subjects variables.

CHAPTER III.

Results

Preliminary Analyses

Poser group characteristics. Demographic information for the 4 poser groups is presented in Table 1. Posers were carefully matched across the 4 poser groups (LSVT, ARTIC, no tx PD, and NCs) on several demographic variables, including age, education, gender, and disease stage. Several other variables were also collected for all posers, including ethnicity, time since PD diagnosis, pre-treatment BDI-II scores, and pre-treatment MMSE scores. Five one-way ANOVAs comparing demographic variables revealed that there were no statistically significant differences between the four poser groups for age F (3, 52) = 0.620, p = .605, education F (3, 49) = 0.287, p = .835, or MMSE scores F (3, 52) = 1.844, p = .151, and no statistically significant differences between the three PD groups on stage of disease according to the Hoehn and Yahr scale, F (2, 38) = 1.247, p = .300, and time since PD diagnosis F (2, 37) = 0.365, p = .696.

A sixth ANOVA was conducted comparing transformed BDI-II scores for PDs and NCs assessed at baseline. Since the distribution of BDI-II scores was positively skewed and not normally distributed, as expected, we transformed the BDI-II distribution using the natural log transformation and used this set of transformed BDI-II scores for this and all subsequent analyses. See data transformation discussion below. Mean scores on the BDI-II were generally in the normal to mild range for clinical depression, however, results revealed statistically significant differences between NCs and PDs, F (1, 50) = 1.45, p = .002. The transformed BDI-II mean for the NC group (M = 0.80, SD = .90) was significantly lower than the transformed mean for the PD group (M = 1.83, SD = .78), indicating that PD patients showed higher symptoms of

depression than NCs. To further explore this difference, we examined all four poser groups, and an additional ANOVA was conducted comparing transformed BDI-II scores across all poser groups. Results revealed a significant overall group difference F (3, 52) = 6.90, p = .001, with the mean for NC (M = 0.80, SD = .90) differing significantly from all three PD groups: No Tx PD (M = 1.61, SD = .73), ARTIC (M = 1.77, SD = .73), LSVT (M = 2.13, SD = .87). Due to the statistically significant difference between poser groups on the BDI-II score, the effects of this variable were removed from the analyses by covarying transformed BDI-II scores from all subsequent analyses. These results are summarized in Table 1.

A chi square analysis revealed no significant difference in gender between poser groups (χ^2 [3, n = 56] = 0.827, p = .84). A closer examination of the gender distribution in our sample showed that while there were no gender differences across poser groups, there was an unequal number of men and women in each of the PD groups. In all three PD groups there were almost twice as many men as women. This is not surprising given that the incidence of PD is significantly higher in men than women (Van Den Eeden et al., 2003). This gender distribution illustrates that our sample is representative of the PD population. However, this pattern was not observed in the NC group, where there were an equal number of men and women. This unequal gender distribution in some poser groups but not others would serve as a confounding factor in the analyses proposed for this study. Therefore, it was decided to include gender as a factor in all analyses to control for this possible confound. Gender was also included in the analyses given the large literature showing gender differences in emotion processing (Borod et al., 2001; Borod & Madigan, 2000).

Rater group characteristics. Raters were carefully selected for this study and an equal number of men and women were selected for each cohort and matched with respect to education. Demographic information for the raters is presented in Table 2.

Assessing normality. The Shapiro Wilk's test of normality was conducted to assess the normality of the variables in the dataset. A criterion of $p < .05$ was selected as the level of significance needed to identify distributions that differed significantly from the normal distribution. Shapiro Wilk's tests were conducted separately for each poser group. Results showed that many of the distributions were non-normally distributed. See Table 3 for normality assessment results.

Data transformation. In order to normalize the data to meet the assumptions of the parametric statistics planned for the data analyses, a natural log transformation was performed on all the variables in the dataset. The Shapiro Wilk's test of normality was repeated on the transformed data to determine whether the transformation successfully normalized the majority of the variables in the dataset. Results showed that of the 128 variables examined, non-significant Shapiro Wilk's tests emerged in 121 cases, indicating that 121 variables were normally distributed after the data transformation. Only seven variables, or 5% of the data, remained non-normally distributed, a level expected by chance. See Table 4 for normality assessment results following data transformation. One covariate, the Beck Depression Inventory (BDI) score, was used throughout all the aims. As expected, the distributions for this variable were non-normally distributed for both NCs and PDs. The same transformation procedure, the natural log transformation, was applied to this variable. The resultant normality of the variable was demonstrated by a non-significant Shapiro Wilk's statistic.

Inter-rater reliability. A series of one-way random-model Intra-Class Correlations (ICC) was conducted to assess inter-rater reliability among our raters. Four sets of reliability analyses were conducted; the first two sets occurred during training sessions for each variable, the third was performed on the experimental ratings for each variable in each cohort, and the final analyses were performed on the entire dataset for each variable separately. During training sessions, raters were first presented with videos of facial expressions representing each point on the Likert scale, and then were asked to rate additional videos with feedback from the group (i.e., conferencing stage) and independently (i.e., inter-rater reliability stage). In total, 24 ICC analyses were performed during training sessions (4 Variables X 3 Cohorts X 2 Rating Sessions per training). Results are displayed in Table 5. Results showed that agreement between our raters was high. In addition, 12 ICC analyses were performed on the experimental data collected (4 Variables X 3 Cohorts), and 4 ICC analyses were performed on the entire dataset, one for each variable. Results for the reliability analyses of the experimental ratings are displayed in Table 6.

Analyses to answer aims.

Aim 1: Assessing differences between PDs and NCs. To assess differences between male and female PDs and NCs, a series of 2 X 4 X 2 mixed-model analyses of covariance (ANCOVAs; Group X Emotion X Gender) were conducted with repeated measures for Emotion, and BDI-II score as a covariate, on the pre-treatment data (i.e., baseline data). Monologue type included three emotional monologues (happy, sad, and angry) and one non-emotional monologue (neutral). The ANCOVAs were conducted separately for each of the facial variables (i.e., FM, EF, EI, and EV). The results of Aim 1 are summarized in Table 7.

Assessing facial mobility in PD and NC individuals. Results showed that there was no

significant main effect for Group, Pillai's Trace, F (1, 51) = 2.34, p = .13, however this result was superseded by the significant Group by Gender interaction. There was a significant interaction between Group and Gender, Pillai's Trace, F (1, 51) = 7.48, p = .009, indicating that the pattern of results for PDs and NCs differed as a function of gender. PD men displayed significantly lower EF than PD women while NC men and women performed similarly. There was a significant main effect of Emotion, Pillai's Trace, F (3, 49) = 2.72, p = .05. The emotion with the highest mean was Angry (M = 1.05, SE = .05), followed by Happy (M = 1.04, SE = .05), Neutral (M = .98, SE = .05), and finally Sad (M = .95, SE = .05). There was also a significant main effect of Gender F (1, 51) = 4.09, p = .05, with women (M = 1.10, SE = .07) displaying higher scores on FM than men (M = .91, SE = .06).

Assessing emotional frequency in PD and NC individuals. The interaction between Group and Gender emerged as a trend, Pillai's Trace, F (1, 51) = 2.98, p = .09. The interaction showed that while NC men and women performed similarly, PD men displayed significantly lower EF than PD women. There was no significant main effect for Group, Pillai's Trace, F (1, 51) = 1.66, p = .20, however this result was superseded by a trend for a Group by Gender interaction. The main effect for Emotion was not significant, Pillai's Trace, F (3, 49) = 0.98, p = .41. However, the pattern of means across the emotions was the same for FM and EF, with the highest mean for Angry (M = .99, SE = .05), followed by Happy (M = .99, SE = .06), Neutral (M = .96, SE = .06), and finally Sad (M = .90, SE = .06). There was no significant main effect of Gender F (1, 51) = 1.50, p = .23, but the means for men and women followed the expected pattern; women (M = 1.02, SE = .08) displayed more EF than men (M = .89, SE = .07).

Assessing emotional intensity in PD and NC individuals. There was a trend towards a

significant Group by Gender interaction, Pillai's Trace, F (1, 51) = 3.38, p = .07. The interaction showed that as in FM and EI, PD men displayed lower EI than PD women, but NC men and women performed similarly. There was no significant main effect of Group, Pillai's Trace, F (1, 51) = .33, p = .56, however this result was again superseded by the trend for the Group by Gender interaction. There was a trend toward a significant main effect of Gender F (1, 51) = 3.75, p = .06, and, again, the means for men and women followed the expected pattern; women (M = .94, SE = .08) displayed more EI than men (M = .74, SE = .07).

Assessing emotional variability in PD and NC individuals. There was a trend for the interaction between Group and Emotion, Pillai's Trace, F (1, 51) = 3.64, p = .06. The interaction showed the same pattern of results for all the variables examined, with NC men and women showing similar displays of facial expression and PD patients showing reduced expression compared to PD women. The main effect of Group was not significant, Pillai's Trace, F (1, 51) = 1.66, p = .20, however, this result was again superseded by the trend of the Group by Gender interaction. Despite not reaching significance, the means for PDs and NCs emerged in the expected direction, where NCs (M = .91, SE = .09) were rated as having significantly more EV than PDs (M = .77, SE = .05). The main effect for Emotion was significant, F (3, 49) = 2.98, p = .04, with Angry (M = .89, SE = .04), followed by Happy (M = .86, SE = .05), Neutral (M = .82, SE = .05), and finally Sad (M = .79, SE = .05). There was a significant main effect of Gender F (1, 51) = 3.91, p = .05, and, again, the means for men and women followed the expected pattern; women (M = .93, SE = .07) displayed more EV than men (M = .75, SE = .06).

Aim 2: Treatment effects. The goals of our study were focused on facial emotional expression following ARTIC and LSVT, and thus our treatment outcomes are measured in

intensity, frequency, and variability of emotional expression. The main treatment outcome of the larger study was of vocal loudness, measured as sound pressure level (SPL). All 13 PD posers who received LSVT displayed marked improvements in SPL. In comparison, results from the ARTIC group showed no overall change in SPL level after treatment. Both untreated PD and NC groups displayed lower SPL at the time of the second measurement compared to baseline.

To assess treatment effects of facial emotional expression over time (pre-treatment, post-treatment) among the four poser groups (NC, no tx PD, ARTIC, and LSVT) for the four emotional monologue types (Angry, Happy, Neutral, and Sad) and gender (men and women), we conducted a series of 2 x 4 x 4 x 2 mixed-model ANCOVAs, with repeated measures for Emotion, and the BDI-II score as a covariate. The results of Aim 2 are summarized in Table 8. We also examined LSVT treatment responses in individual posers. Those data are displayed in Figure 3.

Treatment effects in facial mobility. No significant 3-way interaction emerged between Time, Poser Group, and Gender, Pillai's Trace, F (3, 46) = 2.01, p = .13. However, the 2-way interaction between Time and Poser Group, Pillai's Trace was significant, F (3, 46) = 2.62, p = .03. This finding indicates that significant differences were found between the treatments for FM over time, but these did not differ as a function of gender. The data for the Time by Poser Group interaction are displayed in Figure 2. Results revealed that while FM in NCs and no Tx PD did not change over time, FM increased to a small degree for ARTIC, and increased significantly for the LSVT group. Both Emotion and Gender main effects were significant, F (3, 44) = 6.37, p = .001, F (1, 46) = 1.94, p = .002, respectively. For Gender, women (M = 1.11, SE = .07) were rated as significantly more facially mobile than men (M = .83, SE = .05). Significant differences

were also found for the emotions, with Happy (M = 1.02, SE = .04), followed by Angry (M = 1.00, SE = .04), Neutral (M = .94, SE = .04), and finally Sad (M = .92, SE = .05). The main effect for time emerged as a trend, F (1, 46) = 3.23, p = .08, with a lower rating for pre-treatment (M =.96, SE = .04) than post-treatment (M = .98, SE = .04).

Treatment effects in emotional frequency. As in FM above, no significant 3-way interaction emerged for EF for Time, Poser Group, and Gender, Pillai's Trace, F (3, 46) = 1.78, p = .16; however, the 2-way interaction between Time and Poser Group, Pillai's Trace, was significant, F (3, 46) = 3.00, p = .04. This finding showed that treatments affected EF differently over time, but these did not differ as a function of gender. The data for the Time by Poser Group interaction are displayed in Figure 2. Results revealed that EF for NCs did not change over time, EF for no Tx PD and ARTIC groups decreased over time, and the LSVT group increased across time. Gender and Time emerged as trends, Pillai's Trace, F (1, 46) = 2.92, p = .09, and Pillai's Trace, F (1, 46) = 3.54, p = .07, respectively. Women (M = .99, SE = .08) were rated as having higher EF than men (M = .82, SE = .06), and EF was lower in pre-treatment (M = .90, SE = .05) than post-treatment (M = .91, SE = .05).

Treatment effects in emotional intensity. For EI, a significant 3-way interaction emerged between Time, Poser Group, and Gender, Pillai's Trace, F (3, 46) = 2.93, p = .04, and the 2-way interaction between Time and Poser Group revealed a trend Pillai's Trace, F (3, 46) = 2.46, p = .08, indicating that significant differences between the treatments were found over time, but the pattern of results differed for men and women. The data for the Time by Poser Group by Gender interaction are displayed in Table 9. Results revealed that for NCs, no Tx PD, and ARTIC groups, the men and women had opposite patterns of behavior over time, that is, when EI for one gender

increased, EI for the other gender decreased, except for the LSVT group. In the LSVT group, EI for men and women increased over time. This pattern of results is further reflected in a significant main effect for Gender, Pillai's Trace, F (1, 46) = 5.86, p = .02, with women (M = .93, SE = .08) displaying significantly more EI than men (M = .69, SE = .06). The emotion main effect showed a trend towards significance, F (3, 44) = 2.28, p = .09, with Happy (M = .87, SE = .05), followed by Angry (M = .83, SE = .05), Neutral (M = .79, SE = .05), and finally Sad (M = .77, SE = .05).

Treatment effects in emotional variability. A significant 3-way interaction also emerged for EV for Emotion, Time, and Poser Group, Pillai's Trace, F (3, 46) = 3.27, p = .03, and the 2-way interaction between Time and Poser Group revealed a trend, Pillai's Trace, F (3, 46) = 2.73, p = .06. These results indicate that ARTIC and LSVT affect EV differently over time, and that these differences changed as a function of Gender. The data for the Time by Poser Group by Gender interaction is displayed in Table 1. The pattern of results in EV was identical to that of EI. While men and women in the LSVT group increased over time, men and women in all three other poser groups displayed opposite patterns of behavior; when EV for one gender increased, EV for the other gender decreased. This pattern was reflected in the significant Gender main effect, F (1, 46) = 6.52, p = .01. Women (M = .91, SE = .07) were rated a s significantly more emotionally intense than men (M = .69, SE = .05). A significant main effect for Emotion was also found, F (3, 44) = 4.09, p = .01, with Happy (M = .84, SE = .04), followed by Angry (M = .82, SE = .04), Neutral (M = .77, SE = .05), and finally Sad (M = .75, SE = .05).

Aim 3: Effect of voice treatments on specific aspects of emotional expression. To assess whether the different voice treatments (ARTIC, and LSVT) had differential effects on our

Facial Rating Variables (EF, EI, and EV) across Time (pre-treatment, post-treatment), emotion (angry, happy, neutral, sad), and Gender (men and women), we conducted a mixed-model ANCOVA 4 x 3 x 2 x 4 x 2, with repeated measures for Time, Emotion, Facial Rating Variable, and BDI-II score as a covariate. Results indicated that the different voice treatments did not differentially affect one Expression Type over another. This is reflected in the in the non-significant 4-way interaction for Poser Group, Expression Type, Time, and Gender, Pillai's Trace, F (6, 92) = .72, p = .63, and in the non-significant 3-way ANCOVA for Time, Poser Group, and Expression Type, Pillai's Trace, F (6, 92) = .45, p = .84. The results of Aim 3 are summarized in Table 11.

Aim 4: Relationship between different aspects of emotional expression. Two approaches were used to examine Aim 4; the first employed correlation statistics and the second used ANCOVAs. In the first approach, a series of two-tailed Pearson Partial Product-Moment correlations were conducted to assess the relationship between our three emotional expression variables (i.e., EF, EI, & EV). The partial correlations were conducted covarying for the Beck Depression Inventory scores (natural log transformed), thus removing the effect of depression from the correlations. For the rationale for this approach see Aim 1. The correlations were conducted between variables that would be meaningful when compared. As such, the correlations were conducted separately for each emotion, and separately for each time point. For example, the values for pre-treatment angry EI were correlated with values for pre-treatment angry EV. It was decided that it would not be meaningful to compare values for pre-treatment and post-treatment, and beyond the scope of this aim to examine correlations between the different emotions. This approach yielded 8 correlation matrices. We also examined the relationship between these

variables separately for each poser group to ascertain if there were different patterns of relationships among the variables as a function of voice treatment received (i.e., ARTIC or LSVT). Finally, we examined these correlations separately for men and women due to the significant gender effect in emotional expression documented in the literature as well as in previous analyses conducted in this study. This overall data analytic approach yielded 256 correlation coefficients. These matrices were combined for clarity and are shown in two large matrices, one for men and another for women. See Table 12 and Table 13 for the full correlation matrices. Results showed that there was a high positive relationship among our dependent variables, with correlation coefficients ranging from ($r = .54$, $p < .05$) to ($r = .99$, $p < .05$). Despite these high correlations, it was decided to keep the variables independent as opposed to combining them to create a composite index of facial expression. This decision was informed by conceptual reasons, quantitative analysis, and empirical research. For further detail about the rationale for analyzing the emotional expression variables separately, see the Discussion.

In the second approach to examining Aim 4, we conducted a 3 x 4 x 2 mixed-model ANCOVA (Facial Expression Variable X Poser Group X Gender), with repeated measures for Expression Type, and the BDI-II score as a covariate, to assess whether there were differences between our emotional expression variables across Gender at baseline, and if so, whether these varied as a function of Poser Group. Therefore, we conducted the analyses separately for each Poser Group, in the same fashion as we did for the partial correlations discussed above. Results showed that significant differences were found between the emotional expression variables for NCs, Pillai's Trace, F (2, 11) = 21.76, $p < .001$, but not for any of the PD groups: no Tx PD, F (2, 10) = 1.48, $p = .27$, ARTIC, F (2, 10) = 0.39, $p = .69$, and LSVT, F (2, 9) = 1.43, $p = .29$. For

NCs, EF (M = .98, SE = .09) differed significantly from both EI (M = .85, SE = .09) and EV (M = .89, SE = .08), but EV and EI did not differ from each other. The results of this portion of Aim 4 are summarized in Table 14.

CHAPTER IV.

Discussion

Facial emotional expression is the observer's window into an individual's subjective emotional state. It is the external reflection of our inner landscape; the physical manifestation of our emotions. It is how we connect with each other. The work described in this dissertation examines the ways in which this connection is compromised in individuals with PD and the ways in which voice treatment can affect change in facial emotional expression.

Summary of Findings

Our findings revealed that PD male posers displayed lower facial expression on all the variables we examined. PD men in our sample displayed significantly lower FM, EF, EV, and EI than did PD women posers, and significantly lower FM, EF, and EV compared to NC men. However, this pattern was not observed for PD women. Reduced facial mobility and expression were documented in our PD sample, but only for PD men. When examining the effect of our voice treatments over time, our predictions about the effects of LSVT were confirmed. Findings showed that patients who received LSVT were rated as having higher FM, EF, EV, and EI after treatment compared to before treatment. In fact, of our four poser groups, statistically significant increases in facial expression were only observed in the LSVT group.

When we examined the relationship between our emotional expression variables, results revealed that variables were positively correlated with each other, although significant differences were found between them on the baseline data. The differences between the various aspects of facial emotional expression were found only for NCs and not for any of the PD groups. We also investigated whether ARTIC and LSVT differentially affected the facial

expression variables (i.e., EF, EI, and EV), but no such differential effects were found. This result is consistent with our finding that EF, EI, and EV were similar in the PD population, and thus could not be differentially affected by the voice treatments.

Facial Expression Differences between PDs and NCs

In Aim 1, we hypothesized that all 3 PD patient groups would exhibit reduced facial expression on the emotional expression variables, as compared to NCs. Our findings revealed that PD male posers displayed significantly lower facial expression than NCs on two of three emotional expression variables: EF and EV. A reduction of facial emotional expression was consistent with our predictions for our PD sample. We also found a statistically significant amount of reduced facial mobility in PD men compared to NC men, consistent with expected motor impairments in this movement disorder. These findings are consistent with the literature, where evidence of reduced facial expression and mobility in PD has been previously observed (Borod et al., 1990; Buck & Duffy, 1980; Katsikitis & Pilowsky, 1988, 1991; Simons et al., 2004; Smith et al., 1996). However, this finding was only evidenced for PD men, whereas PD women did not differ from NC women on FM.

Gender Differences in PD and NC Emotional Expression

Our results also revealed that women PDs, contrary to our predictions, did not exhibit reduced emotional expression compared to male or female NCs. This gender difference occurred despite the fact that PD men and women did not differ with respect to stage of PD and thus disease severity. This finding can be reconciled if we venture that female PD patients overcompensate, whether consciously or unconsciously, for their reduced facial expressivity. This potential overcompensation can be understood if we take into account the negative social

consequences associated with masked facies (Brozgold et al., 1998) and the important role socialization plays in PD women's lives in comparison to PD men's (Solimeo, 2008). There is evidence from the literature to support this possible explanation. Hemmesch, Tickle-Degnen, & Zebrowitz, (1999) showed that older adults expressed less interest in beginning relationships with female PD patients with facial masking, but that this effect did not extend to men. Given that masked facies has negative social consequences, specifically for women (Hemmesch et al., 1999), and that socialization has a differentially important role for women, it is reasonable that women would overcompensate for any masked facial features that may reduce their quality of life.

Another potential explanation for our gender differences in masked facies is that the disease process is different in men and women and thus yields different symptoms. There is evidence that there are differences in motor symptom presentation and cognitive profiles in men and women with PD (Miller & Cronin-Golomb, 2010). Haaxma and colleagues (2007) showed that more women than men presented with tremor as their initial symptom as opposed to rigidity or bradykinesia. This finding in itself could explain our gender difference in masked facies. Tremor is a typically observable in the extremities, not on the musculature of the face. Thus, the fact that men are more likely to present with rigidity and bradykinesia, motor symptoms that are observable on the face, may explain why men in our sample showed pronounced masked facies whereas women did not. The physiological underpinning of gender differences in PD are unknown, although some suggest that estrogen has a protective effect against degeneration of dopaminergic neurons in the substantia nigra, although there are conflicting results regarding this explanation (Miller and Cronin-Golomb, 2010).

With respect to our sample of normally aging adults, our results showed that there were no differences in the intensity, frequency, or variability of emotional facial expression and facial movement between men and women. This result is inconsistent with the expected gender differences for emotional facial expression. The literature on gender differences for emotional facial expression shows that women typically display a larger amount of facial movement and expressivity compared to men (Borod & Madigan, 2000; Wallbott, Harald, Scherer, & Klaus, 1991).

Importance of Facial Expressions for Humans as a Species

Our findings show that facial expressions are significantly compromised in male PD patients compared to healthy age-matched men. The question is: why is this impairment so clinically relevant? Previous studies suggest that facial expressions impact the impressions, attitudes, and perceptions of social functioning that observers make about individuals. For example, Riggo and Friedman (1986) showed that the variability of facial expression in healthy adult women was related to positive impression ratings. In a series of studies, Pitcairn and colleagues (1987, 1988, and 1990) documented that impaired facial expressions are associated with PD patients being perceived as more negative (e.g., more anxious, depressed, and passive). Furthermore, reduced intensity of facial expression has been associated with poor social functioning (Brozgold et al., 1998).

The empirical evidence cited above corroborates what is intuitive about the importance of facial expression for our social functioning. However, facial expressions are important beyond their function of facilitating healthy social interactions in our present society. Charles Darwin argued that modern man has facial expressions because they were so critical for early man. He

theorized that the ability for humans as a species to produce pronounced facial expressions was so important for early man that the skill was incorporated into our species through natural selection. Darwin argued that men who were best able to project a threat to their rivals through facial expressions had an advantage compared to men who were not able to produce such facial expressions. Darwin wrote *"*Nor must we overlook the part which variation and natural selection may have played; for the men which succeeded in making themselves appear the most terrible to their rivals, or to their other enemies, if not of overwhelming power, will on an average have left more offspring to inherit their characteristic qualities, whatever these may be and however first acquired, than have other men" (Darwin, 1872, p. 104). Given that there is an entire book devoted to the subject of facial expression written by a luminary scientist, such as Charles Darwin, coupled with the experimental evidence from modern research studies, it is evident that the ability for individuals to effectively produce facial expressions is very important. Thus, impaired facial expression in PD likely has significant effects on individuals' functioning in society. The well-established evidence of masked facies in the literature (Borod et al., 1990; Buck & Duffy, 1980; Katsikitis & Pilowsky, 1988, 1991; Simons et al., 2004; Smith et al., 1996), and in the present study, and the importance of facial expressions in daily life highlight masked facies as a treatment target in PD that should not be overlooked.

Facial Expression Differences in PD as a Function of Emotion

The second prediction we made about our PD sample was that patients would display reduced frequency of emotion (EF) compared to NCs specifically in the happy monologues and increased frequency of emotion compared to NCs specifically in the sad monologues. These predictions were made based on work documenting reduced frequency of smiles in PDs

compared to NCs (Pitcairn et al., 1990), and increased expressions of sadness in PDs compared to NCs (e.g., Brozgold et al., 1998). Our results are partially consistent with our hypothesis, although only at a trend level. Findings showed that the PD patients produced less frequent emotional expressions across all emotions Our prediction that PD patients would display increased frequency of sad expressions was not supported, however, our results are consistent with those of Pitcairn and colleagues (1990), who also documented reduced frequency of positive emotion (i.e., smiles) in PD. However, a direct comparison of our data to those of Pitcairn and colleagues (1990) was only appropriate for the happy monologues since both studies assessed happy expressions (i.e., happy smiles). Other than smiles, the type of data that were collected in the Pitcairn study was specific to individual muscle movements and not emotional expressions of anger or sadness, and thus cannot be directly compared to our results. However, the authors noted that PD patients showed very few muscle movements other than ungenuine smiles, which is qualitatively consistent with our findings.

Effects of Voice Treatments on Facial Expression

For Aim 2, we hypothesized that EF, EV, and EI would be most improved by the LSVT as compared to the ARTIC treatment and as compared to the no treatment PD and NC groups. Findings supported our hypothesis for all variables examined. Our second hypothesis was that EF, EV, and EI would also improve following ARTIC treatment but to a lesser degree than after LSVT. In fact, facial expression and mobility did not change to any significant degree over time following ARTIC treatment, and for female PD patients, it actually decreased EV and EI compared to baseline levels.

When examining the LSVT group at the individual poser level, the results of our study

are even more convincing. Our results indicate that the treatment effect following LSVT is present for a vast majority of posers and not isolated to one or two responders. Of 12 posers who received LSVT, 9 showed an increase in facial mobility, frequency, intensity, and variability of emotional expression, and a tenth poser showed an increase in three of the four variables we examined. Only one poser did not make any gains in emotional facial expression following LSVT. These findings indicate that LSVT is a robust and effective treatment for masked facies in PD.

No Differential Effects of Voice Treatments on Facial Expression Variables

In Aim 3 we asked whether ARTIC or LSVT had differential effects on our emotional expression variables (EF, EI, EV). The different aspects of facial expression described in this work had never been examined in the same study using the same population, to our knowledge, and thus this analysis was exploratory in nature. Results showed that the two voice treatments in this study, ARTIC and LSVT, did not differentially affect one type of facial expression over the others. Thus, sensitivity to treatment did not emerge as a significant finding in this study. This result could be expected given our findings from Aim 4 which showed that there were no significant differences among the emotional expression variables in the PD group. Since different types of facial expression were not distinct in the population that received treatment, it would follow that different voice treatments would not have different effects on one over another.

Relationships Between Emotional Expression Variables: Correlational Statistics

The focus of Aim 4 was to examine the relationships among the emotional expression variables developed for this project. Since these three aspects of emotional expression, (i.e., frequency, intensity, and variability), to our knowledge, have never been examined in a PD

population, or examined within the same study in the same sample of healthy adults, it was of interest to examine the relationship among them. Our results revealed that the three variables were highly correlated. One interpretation of these findings is that the variables are measuring the same underlying construct. This construct could be considered a global measure of facial expression. Although this approach is statistically sound, we decided to keep the positively correlated variables independent for three reasons that draw upon conceptual reasons, quantitative analysis, and empirical research.

The first reason is conceptual in nature; we maintain that variability of emotional expression across time is conceptually distinct from frequency and intensity of emotional expression. Drawing from the clinical literature, emotional variability could be likened to emotional lability in patient populations. When a patient presents in a clinical setting with emotional lability, the symptom may point to a differential diagnosis of personality or mood disorder, perhaps Borderline Personality Disorder or Manic-Depressive Disorder (American Psychiatric Association, 2000). However, when a patient presents with reduced frequency of facial expression, this could point to PD or Major Depressive or other mood disorder. Finally, very exaggerated expressive emotional intensity could point to Histrionic Personality Disorder (American Psychiatric Association, 2000). Of course, no clinical diagnosis is ever based on one observed symptom, but this illustration is provided to highlight the inherent differences among frequency, intensity, and variability of emotional expression and how deviations from normal ranges in each variable may be indicative of distinct clinical phenomena.

The second reason we decided to examine frequency, intensity and variability as separate variables was that we found statistically significant differences among them for the healthy

control posers despite the high correlation among them. Based on the conceptual reasons discussed above, we investigated if there were significant differences in the overall means for each variable at baseline. Results were consistent with our predictions and showed that EF differed significantly from EV and EI. Aggregated means for FM showed the highest absolute value in most of the Poser Groups, followed by EF, EI, and EV.

The third and final reason we kept the three variables separate in all analyses is because we wanted to compare our data with results from the literature. Many studies that examine facial expression in PD measure only one or two of these variables. Most commonly, only one variable is reported. To be able to compare our findings to findings from other studies in a meaningful way, we wanted to examine our data using similar methodology.

Relationships Between Emotional Expression Variables: General Linear Model Statistics

The second analytic approach taken in Aim 4 revealed that keeping EF, EV, and EI as independent variables allowed us to find meaningful differences among them, despite the high correlations among them. In this approach, we were able to document statistically significant differences between frequency of emotional expression and the other two aspects of emotional expression, EV and EI, in NCs. No differences were found between EV and EI. However, none of these differences were present for any of the PD patient groups. This result is meaningful because it broadens our knowledge in two areas. First, these results further characterize the expression deficit in PD. It has been known for many years that PD patients present with masked facies, simply defined as reduced facial mobility and expression. Our finding suggests that masked facies could in fact represent a qualitative difference between PDs and NCs, not simply a quantitative difference as has long been assumed. Masked facies could be a departure from the

varied aspects of facial emotional expression seen in normal population, such as intensity of expression, not simply a reduction in frequency. Second, this finding lends support to our assumption that the three emotional expression variables are separate and can be regarded as conceptually unique, at least in normal populations. Thus, this finding lends some validity for the use of this scale in normal populations.

Depression in PD

When we examined Beck Depression Inventory-II scores for the full score range, we found significant differences between the PD and NC groups. None of the PD patients in our study had severe symptoms of depression, as that was an exclusionary criterion. The vast majority of PD patients with any depressive symptoms were within the mild range of severity (n = 39), and only two patients had BDI-II scores reflecting a moderate level of depression. Despite the fact that PD posers were randomly assigned into treatment groups, this was not possible with PD and NC posers by virtue of their identity. It has been widely documented, in work from our lab and many others, that the incidence of depression is high in PD. It is estimated at approximately 40% (Raskin et al, 1990), with some variability reported in the literature (Zgaljardic et al., 2003). Since the incidence of depression in this patient population is so high, it could be expected that we would find significant differences between the patients and NCs. In fact, the difference in depression scores between our PDs and NCs suggests that the current sample is representative of the PD population. The implication of this difference in depression levels has some bearing on the results that emerged from the present study. It has been documented that unipolar depression is associated with reduced facial expression (Jaeger et al.,

1986). Therefore, it was important to statistically remove the potential effect of depression from our results.

Ratings of Emotions as a Function of Gender and Poser Group

In additional to exploring our aims and hypotheses, the data set collected provides a rich source of information about emotional processing, and our results include interesting findings that extend beyond the scope of our initial aims. We used these data to provide supplementary information about our sample and to compare relative magnitudes of ratings to previous work from our own lab and from the larger literature. Our dataset provides information about ratings of facial expressions produced while NC and PD posers recounted salient and vivid emotional experiences. We collected rating data on three emotional monologues: anger, happiness, and sadness. Posers were also asked to recount an emotionally neutral experience. This condition was included to provide a non-emotional control against which the emotional monologues could be compared. Including different emotions in our study allowed us to compare relative values of ratings for anger, happiness, and sadness via the main effect of Emotion. It also allowed for exploration of differences among the intensity, variability, and frequency of different emotions via the Facial Expression Variable by Emotion interaction.

The main effect of Emotion revealed that for NCs, ratings of the sad monologues received the lowest ratings compared to the other two emotional monologues on all variables explored. With respect to the emotional monologues, the angry monologues always received the highest ratings, followed by happy, and finally sad. This pattern of results is consistent with that of Bowers and colleagues (Bowers, Miller, Bosch, Gokcay, Pedraza, Springer, & Okum, 2006), despite the fact that individuals in this study were asked to deliberately pose the different

emotional facial expressions whereas posers in our study produced facial expressions spontaneously. This study included six emotions compared to our three, but they found the same relative order for the emotions, with the highest movements for angry, followed by happy, and the lowest amount of movement for sad.

Etiology of Masked Facies and Mechanisms of Action for LSVT: Motor, Emotional, or Both?

The present study was designed to explore differences in emotional facial expression between NCs and PDs. It is widely accepted that PD patients have impairments in facial expression (Borod et al., 1990; Buck & Duffy, 1980; Katsikitis & Pilowsky, 1988, 1991; Simons et al., 2004; Smith et al., 1996). However, there is no consensus in the literature regarding the etiology of these impairments. Are they due to motor or emotional processing deficits? It is undeniable that PD is a movement disorder that affects mobility of gross and fine motor skills and likely also affects the musculature of the face. Emotional facial expression in all individuals recruits both motor systems to execute facial movements as well as higher-order (i.e., cortical) and more primitive (i.e., basal ganglia and amygdala) emotional systems to inform those movements with emotional meaning. It could therefore be argued that both masked facies and the positive treatment effects found following LSVT are purely a function of motor processing. In fact, our own findings suggest that ratings of facial mobility, presumably devoid of emotional content, are decreased for PDs relative to NCs and that they are affected by LSVT. However, results from a neuroimaging study of LSVT (Liotti et al., 2003) revealed that neural activations in PD patients shifted from bilateral recruitment pre-treatment to largely right hemisphere and right insula activation post-treatment. Since the right hemisphere and insula are essential for

emotional processing and are not particularly specific to motor functioning, we can speculate that the effect of LSVT on facial expression occurs due to emotional processing improvements. Our own results demonstrate that the effects of LSVT are not exclusively due to emotional processing improvements, but result from either the improvement of motor functioning, or more likely, to the combination of motor and emotional functioning.

Limitations of the Present Study

One limitation of the present study, and possibly all studies examining facial expression in PD, is the role of depression. Given the significant effect that depression has on facial expression, the high incidence of depression in PD, and the significant differences in depressive symptomatology in our sample, we statistically removed the effects of depression by covarying each poser's score on a rating scale of depressive symptomatology, the Beck Depression Inventory. This allowed us to isolate the effects of the two disease processes that are comorbid in this population: Parkinson's and Major Depressive Disorder. However, this is a statistical technique and these factors are not dissociable when observing an individual in a social situation. Therefore, while we may argue that differences between groups are due to one factor (i.e., PD), it may be difficult to dissociate the relative effects of PD and depression in certain individuals.

Another limitation of the present study is that the Likert rating scale used to rate facial expression and facial mobility yielded a small range of scores. Raters attributed lower ratings to the faces of both PDs and NCs more often than they did higher ratings. Great care was taken during training sessions to prevent this problem; as the raters were instructed to and trained to use the entire range of numbers on the scale. Despite these precautions and the fact that thousands of data points were collected, many of our variables were not normally distributed and

were positively skewed. We were able to adjust for this statistically by transforming the data using a natural log transformation; however, the range of mean scores remained small. A small range of scores means that there is less variability within the data which makes it more difficult to detect statistically significant effects. It would be important to address this issue in future studies using these rating scales.

An additional limitation of the present study is the fact that PD patients' medication status was not explored as a variable. It is possible that medication type, changes, and side-effect profiles could have affected our results. Finally, our study is the first to examine emotional frequency, intensity, and variability following LSVT in PD. Replication of our findings with a larger sample size will substantiate the positive treatment effects of LSVT on facial emotional expression.

Future Directions

One interesting avenue of further exploration would be to recruit PD patients with a specific prominent motor symptom, that is, rigidity, tremor, or bradykinesia. It is possible that differences in facial expression between our patient and normal groups would decrease if the prominent motor symptom in our patient group were tremor as opposed to rigidity or bradykinesia. This investigation would further characterize the deficit in PD and possibly limit it to only a subset of PD patients. Another potential avenue of investigation would be to examine the effects of the different PD medications on EF, EV, and EI. It is possible that the voice treatments would have differential effects on facial expression as a function of the medication a patient is taking. Furthermore, it would be important to gather data on medication compliance for

each patient, as there is evidence in the literature of cognitive and affective changes that occur when patients are on medication or off medication (Michely, Barbe, Hoffstaedter, Timmermann, Eickhoff, Fink, & Grefkes, 2012).

Several additional avenues for future research are offered below. Future research should assess treatment changes not only as a group result, but also evaluate individual responses within the group. Evaluating individual poser treatment responses would permit us to further establish LSVT as efficacious and generalizable treatment for facial expression deficits in PD. It is also suggested that EF, EI, and EV be explored as one composite variable to potentially improve power in future research. It also may be fruitful to explore the relationship between changes in voice treatments and changes in facial expression in future research.

Conclusions

Findings from the present study confirmed many of our hypotheses, but also uncovered interesting and unexpected results. Our results document the expected facial expression impairments in PD, but also show that these impairments are gender-dependent in our sample. PD men were rated as having significantly lower facial mobility, emotional frequency, emotional intensity, and emotional variability than NC men, whereas PD women did not differ from NC women on any of these parameters. We also documented clear and consistent improvements in both men and women on all aspects of facial expression examined following LSVT. All other treatment groups, including the control treatment group, showed no improvement, and even a decrease of expression over time in certain conditions. Furthermore, we were able to show that the three measures of facial expression, specifically designed for this study, can be differentiated

in a normal population but not in our PD sample. Thus, our emotional expression variables appear separate in healthy adults, and it may be fruitful to use these variables in normal populations in future research.

The findings of the present study have several important clinical implications. Results from our data set indicate that masked facies is not an inevitable symptom of PD, as has long been documented. Factors, such as gender, may interact with disease process to determine the presence or absence of this clinical phenomenon. Furthermore, our treatment findings demonstrate that masked facies is a condition that can be remediated. No other study, to our knowledge, has documented improvements in the emotional aspects of masked facies following any type of treatment.

Our results extend the literature by illustrating that LSVT produces significant improvements in facial expression in individuals with Parkinson's disease. Our findings further showed that the positive treatment effect resulting from LSVT was not isolated to a few responders, but extended to all but two posers, representing an 83% response rate from our sample of 14 poser participants. The fact that a very high proportion of posers in our group who received LSVT displayed significant improvements in several aspects of facial emotional expression suggests that LSVT is a robust treatment which is effective for both men and women at various stages of illness severity, and may thus have high clinical utility. The broader clinical implications of our findings are that treatment of one system of emotional expression (voice) can affect changes in a different but connected system, (i.e., facial emotional expressivity and non-emotional movement). These findings lend further support to the connection between human voice and facial expression and the ability of one to cause changes in the other.

Table 1
Demographic Characteristics for Poser-Participants

Variable	Parkinson's Disease Groups ($N = 41$)						Healthy Controls ($N = 15$)	
	LSVT ($n = 13$)		ARTIC ($n = 14$)		PD untreated ($n = 14$)			
	Male ($n = 9$) *M (SD)*	Female ($n = 4$) *M (SD)*	Male ($n = 9$) *M (SD)*	Female ($n = 5$) *M (SD)*	Male ($n = 9$) *M (SD)*	Female ($n = 5$) *M (SD)*	Male ($n = 8$) *M (SD)*	Female ($n = 7$) *M (SD)*
Age (years)	66.89 (7.20)	66.25 (4.30)	67.11 (9.02)	7.80 (12.90)	67.44 (8.40)	58.00 (9.30)	69.13 (6.45)	61.70 (8.10)
Education (years)	16.56 (2.40)	15.67 (4.70)	17.38 (3.70)	15.40 (5.30)	17.00 (2.60)	14.40 (2.60)	18.25 (2.40)	15.67 (2.00)
Stage	2.11 (0.49)	2.63 (0.48)	2.33 (0.50)	2.30 (0.97)	2.00 (0.66)	2.00 (0.00)	---	---
Time since diagnosis	3.41 (2.60)	11.13 (13.50)	3.89 (3.60)	4.19 (2.60)	7.28 (4.40)	1.70 (0.98)	---	---
BDI-II score	7.67 (4.60)	18.00 (2.20)	7.56 (6.10)	6.60 (2.30)	4.75 (3.00)	9.20 (5.40)	3.38 (4.30)	2.57 (2.90)
MMSE score	29.22 (1.30)	28.25 (2.10)	28.56 (1.30)	28.40 (1.10)	28.89 (0.78)	29.20 (0.83)	29.00 (0.76)	3.00 (0.00)

Note. LSVT = Lee Silverman Voice Treatment; ARTIC = Articulation Voice Treatment; PD = Parkinson's Disease; BDI-II = Beck Depression Inventory; MMSE = Mini Mental State Exam.

Table 2

Demographic Characteristics for Rater-Participants

Cohort	Age *M (SD)*	Education in Years *M (SD)*
Cohort 1 (*n* = 6)	25.3 (6.15)	15.5 (0.55)
Cohort 2 (*n* = 6)	23.8 (2.03)	16.3 (0.75)
Cohort 3 (*n* = 6)	23.8 (6.59)	14.5 (1.50)

Table 3

Normality Assessment using Wilk's Shapiro Test: Raw Data

Variable	Pre				Post			
Treatment Group	Angry	Happy	Neutral	Sad	Angry	Happy	Neutral	Sad
Facial Mobility								
NC	.041*	.183	.012*	.622	.682	.945	.063ŧ	.483
No Tx PD	.471	.688	.915	.552	.994	.912	.439	.162
ARTIC	.261	.315	.236	.004*	.962	.045*	.618	.543
LSVT	.120	.018*	.013*	.015*	.032*	.161	.039*	.097ŧ
Emotional Frequency								
NC	.005*	.164	.008*	.350	.632	.375	.016*	.198
No Tx PD	.062ŧ	.038*	.067ŧ	.185	.504	.119	.122	.044*
ARTIC	.372	.093ŧ	.751	.003*	.358	.010*	.669	.079ŧ
LSVT	.010*	.004*	.018*	.029*	.038*	.281	.218	.372
Emotional Intensity								
NC	.008*	.041*	.017*	.091	.057	.042*	.000*	.009*
No Tx PD	.428	.054ŧ	.708	.125	.071ŧ	.794	.209	.200
ARTIC	.030*	.133	.137	.001*	.296	.192	.269	.152
LSVT	.010*	.019*	.331	.046*	.748	.972	.654	.330
Emotional Variability								
NC	.071ŧ	.051ŧ	.035*	.295	.276	.689	.016*	.082ŧ
No Tx PD	.275	.100	.320	.301	.534	.183	.367	.841
ARTIC	.165	.119	.435	.025*	.420	.184	.202	.917
LSVT	.007*	.012*	.033*	.010*	.047*	.017*	.276	.054ŧ

Note. NC = Normal Control. Tx = Treatment; PD = Parkinson's Disease; LSVT = Lee Silverman Voice Treatment; ARTIC = Articulation Voice Treatment.
* $p < .05$; ŧ $p < .10$ *(trend)*

Table 4

Normality Assessment using Wilk's Shapiro Test: Natural Log Transformed Data

Variable	Pre				Post			
TxGroup	Angry	Happy	Neutral	Sad	Angry	Happy	Neutral	Sad
Facial Mobility								
NC	.130	.620	.056	.866	.999	.957	.768	.957
No Tx PD	.580	.814	.746	.559	.530	.240	.802	.240
ARTIC	.180	.617	.083ŧ	.076ŧ	.265	.998	.902	.998
LSVT	.573	.098ŧ	.048*	.037*	.550	.515	.355	.515
Emotional Frequency								
NC	.069ŧ	.625	.073ŧ	.776	.520	.726	.703	.915
No Tx PD	.403	.447	.548	.405	.971	.828	.718	.311
ARTIC	.527	.683	.384	.218	.921	.327	.381	.311
LSVT	.054ŧ	.040*	.052ŧ	.068ŧ	.315	.525	.689	.631
Emotional Intensity								
NC	.281	.729	.154	.956	.462	.442	.032*	.692
No Tx PD	.410	.322	.574	.743	.029*	.339	.658	.634
ARTIC	.438	.363	.158	.069ŧ	.492	.754	.566	.979
LSVT	.201	.113	.630	.119	.867	.812	.884	.508
Emotional Variability								
NC	.264	.261	.135	.850	.734	.992	.431	.919
No tx PD	.519	.828	.541	.551	.242	.509	.909	.821
ARTIC	.604	.508	.371	.283	.518	.677	.530	.986
LSVT	.062ŧ	.037*	.093ŧ	.025*	.160	.054ŧ	.504	.099ŧ

Note. NC = Normal Control. Tx = Treatment; PD = Parkinson's Disease; LSVT = Lee Silverman Voice Treatment; ARTIC = Articulation Voice Treatment.
* $p < .05$; ŧ $p < .10$ (trend)

Table 5

Intra-class Correlations for Training Sessions: Conferencing and Inter-rater Reliability

	Training Variable			
	FM	EV	EF	EI
	Cohort 1			
Conferencing	.962	.902	.918	.976
Inter-rater Reliability	.921	.848	.910	.937
	Cohort 2			
Conferencing	.932	.895	.943	.923
Inter-rater Reliability	.860	.893	.883	.924
	Cohort 3			
Conferencing	.948	.903	.946	.953
Inter-rater Reliability	.908	.883	.875	.910

Note. FM = Facial Mobility; EV = Emotional Variability; EF = Emotional Frequency; EI = Emotional Intensity. The ICCs for conferencing are based on the original rating evaluation given by each rater, not the re-rating value that *may* have changed after the discussion part of conferencing.

Table 6

Intra-class Correlations for Experimental Rating Data

	Training Variable			
	FM	EV	EF	EI
	Cohort 1			
ICC	.812	.694	.836	.849
	Cohort 2			
ICC	.896	.767	.847	.731
	Cohort 3			
ICC	.867	.867	.757	.885

Note. FM = Facial Mobility; EV = Emotional Variability; EF = Emotional Frequency; EI = Emotional Intensity. ICC = Intraclass Correlation Coefficient.

Table 7

Aim 1: Poser Group by Gender by Emotion ANCOVA (2 x 2 x 4), Significance of Effects

Effect	Facial Rating Variable			
	FM	EF	EI	EV
Group	.132	.203	.568	.204
Emotion	.116	.411	.257	.040*
Gender	.048*	.226	.058	.054
Emotion X Group	.331	.709	.669	.118
Emotion X Gender	.294	.676	.710	.572
Group X Gender	.009*	.091	.072	.062
Emotion X Group X Gender	.845	.302	.413	.420

Note. FM = Facial Mobility; EV = Emotional Variability; EF = Emotional Frequency; EI = Emotional Intensity. Natural log transformed BDI-II score was used as a covariate in this analysis.
* $p < .05$

Table 8

Aim 2: Poser Group by Gender by Emotion by Time ANCOVA (4 x 2 x 4 x 2), Significance of Effects

Effect	Facial Rating Variable			
	FM	EF	EI	EV
Time	.078	.066	.167	.204
Poser Group	.335	.363	.501	.298
Emotion	.001*	.269	.092†	.010*
Gender	.001*	.094	.020*	.014*
Time X Poser Group	.031*	.040*	.075†	.055†
Time X Emotion	.469	.068	.103	.158
Time X Gender	.397	.653	.429	.170
Emotion X Gender	.219	.803	.466	.267
Emotion X Poser Group	.781	.979	.503	.720
Time X Poser Group X Gender	.126	.164	.043*	.030*
Time X Poser Group X Emotion	.084	.041*	.108	.228
Time X Emotion X Gender	.591	.195	.035*	.396
Emotion X Poser Group X Gender	.861	.830	.844	.722
Time X Poser Group X Emotion X Gender	.515	.086	.054†	.330

Note. FM = Facial Mobility; EV = Emotional Variability; EF = Emotional Frequency; EI = Emotional Intensity. Natural log transformed BDI-II score was used as a covariate in this analysis.
* $p < .05$; † $p < .10$ (trend)

Table 9

Aim 2: Means for Poser Group by Gender by Time Interaction for Emotional Intensity

	NCs		No Tx PD		ARTIC		LSVT	
Gender	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Men	.891	.827	.728	.767	.550	.587	.536	.653
Women	.903	.973	1.113	.959	.967	.901	.755	.887

Note. Tx = Treatment; PD = Parkinson's Disease; LSVT = Lee Silverman Voice Treatment; ARTIC = Articulation Voice Treatment. Means are presented using the natural log transformed data.

Table 10

Aim 2: Means for Poser Group by Gender x Time Interaction for Emotional Variability

	NCs		No Tx PD		ARTIC		LSVT	
Gender	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Men	.915	.880	.685	.721	.70	.590	.524	.607
Women	.921	.958	1.050	.936	.868	.811	.813	.898

Note. Tx = Treatment; PD = Parkinson's Disease; LSVT = Lee Silverman Voice Treatment; ARTIC = Articulation Voice Treatment. Means are presented using the natural log transformed data.

Table 11

Aim 3: Poser Group by Gender by Time by Facial Rating Variable x Emotion ANCOVA (4x 2 x 2 x 3 x 4), Significance of Effects

Effect	*p*
Time	.676
Poser Group	.402
Expression Type	.967
Emotion	.086†
Gender	.028*
Time X Poser Group	.186
Time X Expression Type	.167
Time X Emotion	.118
Time X Gender	.316
Expression Type X Poser Group	.281
Expression Type X Emotion	.370
Expression Type X Gender	.243
Emotion X Gender	.781
Emotion X Poser Group	.934
Emotion X Expression Type	.370
Time X Poser Group X Gender	.309
Time X Poser Group X Emotion	.203
Time X Emotion X Gender	.056†
Time X Expression Type X Gender	.394
Time X Expression Type X Emotion	.068†
Expression Type X Emotion X Poser Group	.127
Expression Type X Emotion X Gender	.051†
Emotion X Poser Group X Gender	.891
Expression Type X Emotion X Poser Group X Gender	.039*
Time X Poser Group X Emotion X Gender	.203
Time X Expression Type X Poser Group X Gender	.183
Time X Expression Type X Poser Group X Emotion	.017*
Time X Expression Type X Gender Emotion	.051†
Time X Poser Group X Expression Type X Emotion X Gender	.204

Note. Natural log transformed BDI-II score was used as a covariate in this analysis.
* $p < .05$; † $p < .10$ (trend)

Table 12

Aim 4: Partial Correlation for Emotional Expression Variables as a Function of Treatment Group and Emotion Type: Men

Monologue	Treatment Group	Pre			Post		
		EF*EI	EF*EV	EI*EV	EF*EI	EF*EV	EI*EV
Happy	LSVT	.82*	.89*	.94**	.65	.83*	.82*
	ARTIC	.81*	.75*	.93**	.85*	.74*	.91*
	No Tx PD	.74*	.88*	.88*	.83*	.85*	.97**
	NC	.97**	.88*	.94*	.86*	.54	.72
Angry	LSVT	.95**	.93**	.97**	.92**	.97**	.90*
	ARTIC	.74*	.77*	.94**	.82*	.79*	.83*
	No Tx PD	.85*	.95**	.94**	.90	.99*	.93
	NC	.94*	.93*	.98**	.81*	.56	.78*
Sad	LSVT	.99**	.92**	.87*	.90*	.81*	.77*
	ARTIC	.71*	.66	.95*	.73*	.67	.92**
	No Tx PD	.77*	.67	.93**	.92**	.90*	.81*
	NC	.93*	.91*	.85*	.88*	.80*	.70
Neutral	LSVT	.82*	.80*	.88*	.82*	.80*	.88*
	ARTIC	.89*	.75*	.95**	.89*	.75*	.95**
	No Tx PD	.77*	.82*	.82*	.76*	.82*	.82*
	NC	.76*	.87*	.93*	.76*	.87*	.93*

Note. EV = Emotional Variability; EF = Emotional Frequency; EI = Emotional Intensity; LSVT = Lee Silverman Voice Treatment; ARTIC = Articulation Voice Treatment; PD = Parkinson's Disease; NC = Normal Controls. BDI-IIscore was controlled for in these partial correlations
* $p < .05$; ** $p < .001$

Table 13

Aim 4: Partial Correlations for Emotional Expression Variables as a Function of Treatment Group and Emotion Type: Women

Monologue	Treatment Group	Pre			Post		
		EF*EI	EF*EV	EI*EV	EF*EI	EF*EV	EI*EV
Happy	LSVT	.99	.99	.99*	---	---	---
	ARTIC	.93*	.96*	.99*	.94	.95*	.99**
	No Tx PD	.86	.94	.88	.94	.99*	.93
	NC	.97**	.99**	.98**	.88*	.99**	.93*
Angry	LSVT	.99	.99*	.98	---	---	---
	ARTIC	.99*	.99**	.99*	.92	.90	.99**
	No Tx PD	.89	.39	- .06	.95**	.97**	.98**
	NC	.96*	.99**	.96*	.98**	.99**	.98**
Sad	LSVT	.99	.99	.96	---	---	---
	ARTIC	.99*	.99*	.99*	.90	.97*	.98*
	No Tx PD	.61	.96*	.69	.76	.98*	.84
	NC	.99**	.99**	.99**	.96*	.99**	.98**
Neutral	LSVT	.98	.98	.93	.98	.98	.93
	ARTIC	.93	.95*	.99*	.93	.95*	.99*
	No Tx PD	.92	.96*	.98*	.92	.96*	.98*
	NC	.99**	.96*	.97**	.99**	.96*	.97**

Note. EV = Emotional Variability; EF = Emotional Frequency; EI = Emotional Intensity; LSVT = Lee Silverman Voice Treatment; ARTIC = Articulation Voice Treatment; PD = Parkinson's Disease; NC = Normal Controls. BDI-IIscore was controlled for in these partial correlations. Empty cells represent groups with less than the minimum number of participants necessary to calculate correlation.
* $p < .05$; ** $p < .001$

Table 14

Aim 4: Gender by Emotional Expression Type by Emotion ANCOVA (2 x 3 x 4), Significance of Effects

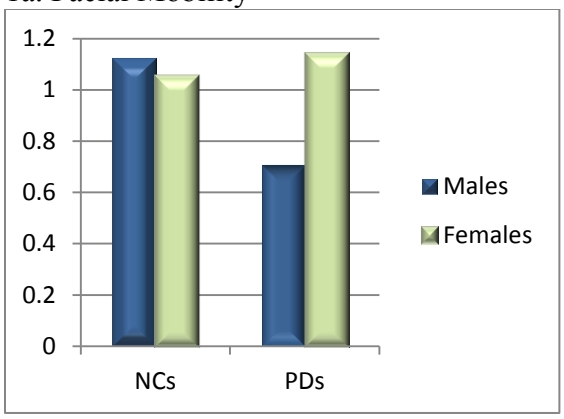
Effect	Treatment Group			
	NC	No Tx PD	ARTIC	LSVT
Expression Type	.000*	.274	.688	.289
Gender	.962	.041*	.156	.218
Emotion	.857	.082	.631	.741
Emotion X Gender	.769	.060	.882	.990
Expression Type X Gender	.232	.639	.131	.521
Expression Type X Emotion	.276	.802	.040*	.737
Expression Type X Emotion X Gender	.426	.548	.302	.598

Note. LSVT = Lee Silverman Voice Treatment; ARTIC = Articulation Voice Treatment; PD = Parkinson's Disease; NC = Normal Controls. Natural log transformed BDI-IIscore was used as a covariate in this analysis
* = $p < .05$
ŧ = $.05 < p < .10$ *(trend)*

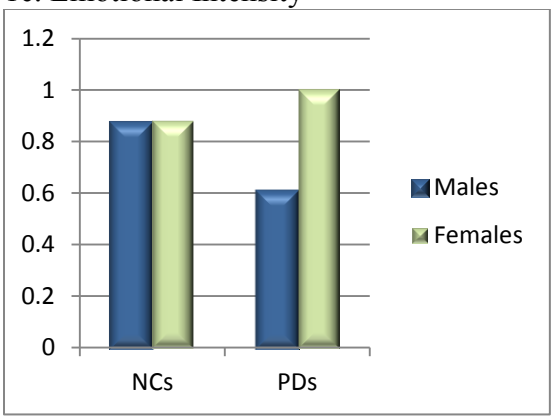
Figure 1

Aim 1: Group by Gender interaction for FM, EF, EI, & EV

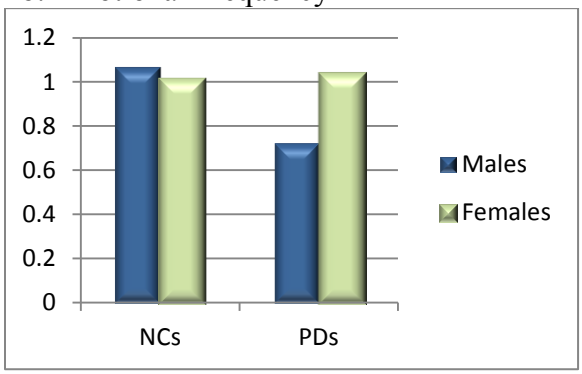
1a. Facial Mobility



1c. Emotional Intensity



1b. Emotional Frequency



1d. Emotional Variability

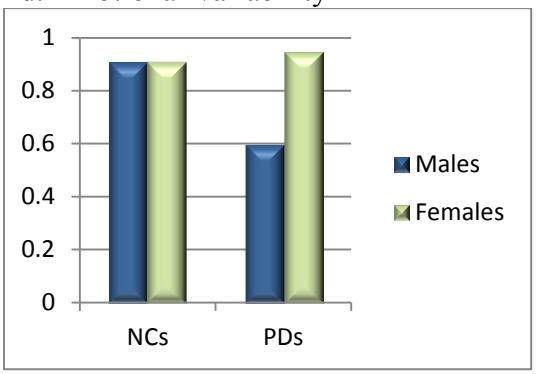
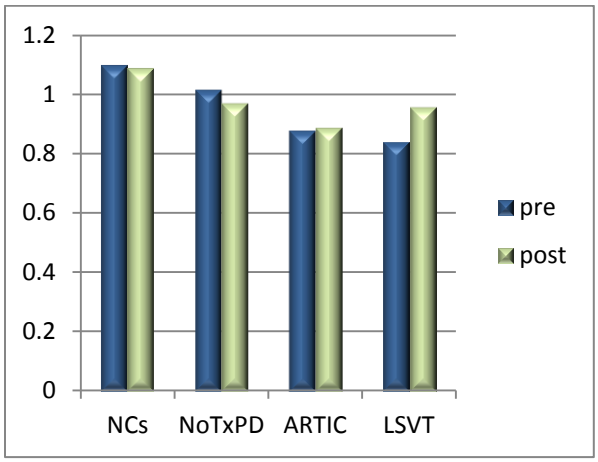


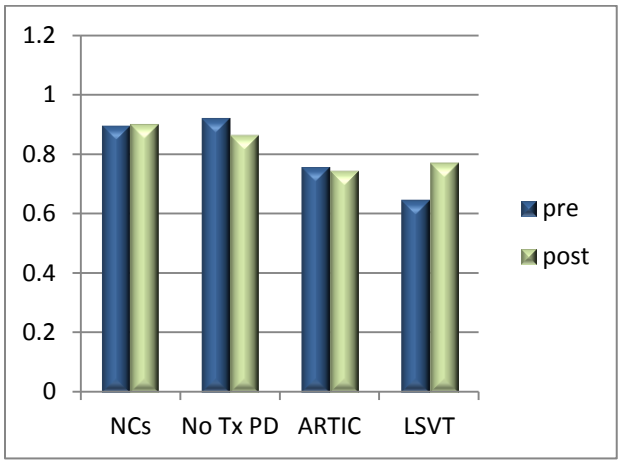
Figure 2

Aim 2: Poser Group by Time interaction for FM, EF, EI, & EV

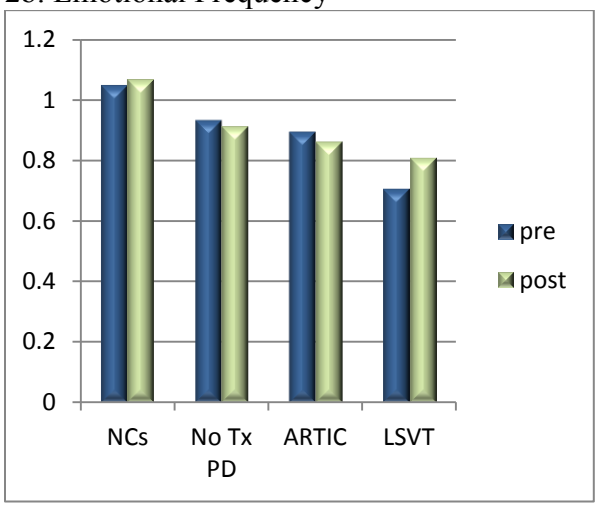
2a. Facial Mobility



2c. Emotional Intensity



2b. Emotional Frequency



2d. Emotional Variability

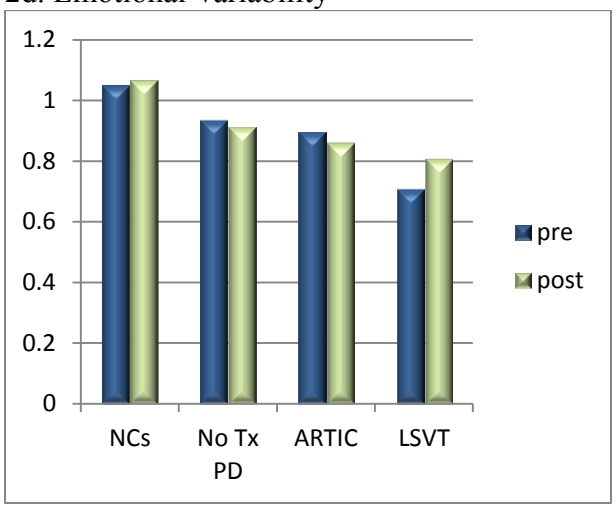
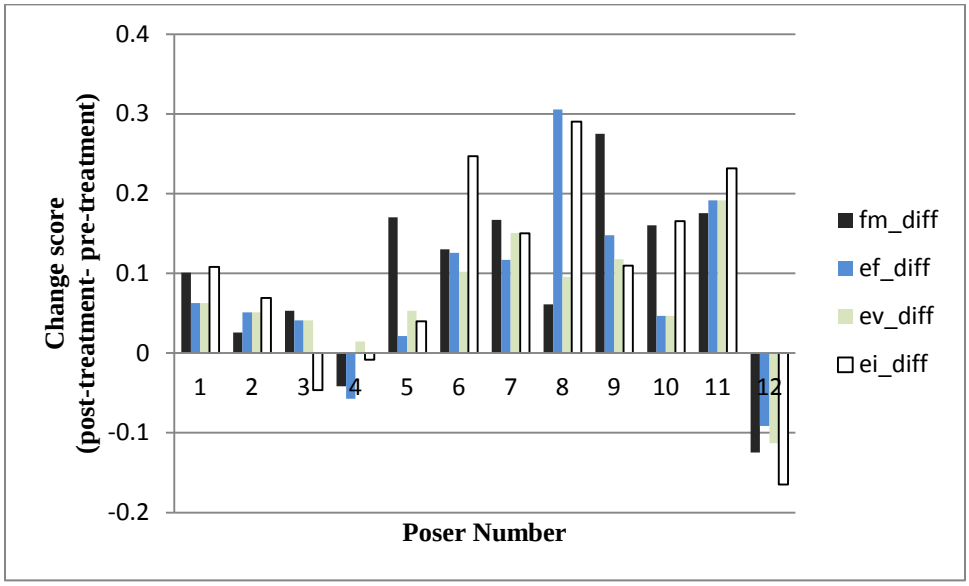


Figure 3

Aim 2: LSVT treatment responses in individual posers



APPENDIX A

Lee Silverman Voice Treatment

The following description of the specific exercises used in LSVT is derived from work published by Ramig and colleagues (Fox et al., 2006). There are 5 unique exercises that patients perform in each 60 minute therapy session led by a speech-language pathologist: simple phonation, pitch range, sentence, hierarchy, and calibration.

For the first exercise, a simple phonation task, patients are asked to say "ah" loudly for as long as they can. Patients are asked to repeat this exercise 12-15 times for a total duration time of 10 minutes. The rationale for this exercise is to improve reduced vocal loudness by stimulating increased vocal fold adduction, loudness, and duration of phonation.

For the second exercise, a pitch range task, patients are asked to say "ah", sustained for 5 seconds, in the highest and lowest pitches they can produce. This task is also repeated 12-15 times per session, and patients are continually encouraged to increase the range of highest and lowest pitches. The rationale behind this exercise is to improve intonation by improving the range of motion in the cricothyroid muscle, a muscle whose rigidity has been associated with a monotone pitch (Fox et al., 2006).

For the third exercise, a sentence task, patients are asked to produce 10 phrases/sentences that they use in everyday life in a loud voice. These are repeated 10 times each for a total of 100 repetitions. This exercise is incorporated into the treatment to facilitate carryover of a loud voice from the training sessions to communication during daily life.

The fourth, hierarchy exercise, involves hierarchical speech loudness drills, whereby loud voice is practiced in single words or phrases during the first week, in sentences during the second

week, in reading paragraphs during the third week, and finally in conversation during the final week. This exercise is also designed to facilitate carryover of loud voice from the simple "ah" exercises to conversational speech, and draws from the principle of complexity discussed below.

The fifth, and final calibration exercise, is more qualitative and involves the calibration of vocal loudness. This is a continuous process during treatment in which calibration exercises are used to help patients self-monitor their loudness and ensure that their voice is within normal limits.

The specific exercises detailed above are based on the five principles fundamental to LSVT: intensity, complexity, salience, functional use, and timing of intervention. Each of these principles will be described in detail below. The principle of *intensity* is derived from evidence that there is a dose-dependent benefit of motor skills training on motor functioning (Fisher et al., 2008). In the Fisher et al. study, PD patients were randomly assigned to one of three exercise groups of varying intensity: a high-intensity group that involved weight-supported treadmill training, a low-intensity group that involved traditional physical therapy, and a no exercise, zero-intensity group that provided participants with education about PD and quality of life issues. Results showed that the participants in the high intensity group exhibited increases in gait speed, step and stride length, and other measures of motor functioning, whereas the low and zero intensity groups did not show consistent improvements. There is further evidence that neural plasticity does not occur through the mere acquisition of a new motor skill, but only through the continued practice of that motor skill (Kleim, Hogg, VanderBerg, Cooper, Bruneau, & Remple, 2004). Intensive treatment is provided in LSVT by a high frequency of sessions (one hour individual sessions 4 days/week for 4 weeks), a high number of repetitions within sessions

(minimum of 15 repetitions of each task per session), and increasingly higher demands for effort, accuracy, and consistency of vocal loudness throughout the duration of treatment. This intensive approach to speech and voice treatment, while common in behavioral rehabilitation, has not been a part of previous approaches focused on articulation or speech rate, and is unique to LSVT (Fox et al., 2006). Previous behavioral approaches to speech/language therapy in PD have focused on articulation, respiration, prosody (Yorkston, 1996), and reducing speech rate (Hammen & Yorkston, 1996), though all have been limited in terms of long-term success (Ramig, Fox, & Sapir, 2004).

The principle of *complexity* refers to the necessity for complex movements to promote neural plasticity (Fox et al., 2006). The movements of patients with PD become less automatic, and as a result, performing two or more motor tasks concurrently, or performing complex tasks becomes more difficult for patients as the disease progresses. To promote complexity of movements, automaticity is trained. The goal of this treatment approach is to incorporate vocal loudness in increasingly more complex tasks. This principle is incorporated into the treatment in the following way; at first, patients are asked to say words or short phrases using a loud voice while seated and inactive; then, more complexity is introduced by asking patients to speak in a loud voice during conversation, while also being engaged in a complex motor task such as walking to the cafeteria and making a purchase. Complexity is further introduced by varying contexts and adding cognitive load (Farley, Fox, Ramig, & McFarland, 2008).

The principle of *salience* refers to the importance of speech tasks being meaningful and rewarding for the patient. This principle is incorporated into the treatment by having patients practice using their loud voice with their friends and family, who in turn reinforce the behavior

by commenting on how much better the patients sound and how much easier it is for them to be heard. When patients' social partners remark about how great they sound, the act of speaking in a loud voice creates a positive and emotionally salient interaction. Thus, an association is created between the act of speaking loudly and a reward, thus increasing patients' probability of speaking loudly in the future. Therapists also incorporate extensive positive feedback into the therapy (Farley et al., 2008). This not only increases patient motivation, but is crucial in promoting neural plasticity (Fox et al., 2006). Engaging in rewarding or emotionally salient tasks activates parts of the basal ganglia, a crucial component of the brain's reward system. "Rewards are associated with phasic modulation of dopamine levels critical to induction of striatal plasticity and learning/relearning in PD" (Fox et al., 2006, p. 290). Fox and colleagues (2006) argue that it is important to focus on saliency of speech exercises because individuals with PD may also experience depressed mood, decreased motivation, and a feeling of helplessness that may lead to a reduced desire to begin therapy and greater attrition rates.

The next principle is *functional use*, or what Fox and colleagues (2006) term "use it or lose it," as well as "use it and improve it." This refers to the notion that while improvements seen after speech exercises focus on vocal loudness, the benefits quickly degrade if exercises are terminated (Fox et al., 2006). This is supported by research in animal models of PD that has linked motor inactivity to accelerated degeneration (Tillerson, Cohen, Caudle, Zigmond, Schallert, & Miller, 2002). Therefore, continuous practice is necessary to maintain treatment gains. The neural substrates accounting for this process may be the vulnerability of spared but compromised dopamine neurons to periods of inactivity, where inactivity may accelerate deficits. This principle is addressed in LSVT by a focus on incorporating the target behavior, that is, using

a loud voice, into everyday communication. Therefore, continued practice becomes part of the individual's life.

The final guiding principle of LSVT is the *timing of interventions*, which refers to the importance of timing in the initiation of treatment. Evidence from animal studies indicates that when exercise is introduced early enough, there is potential to substantially slow and even halt disease progression (Tillerson, Caudle, Reveron, & Miller, 2003). While animal models of PD are acute and do not perfectly simulate the progressive degeneration of dopamine neurons in humans, these data are very promising. They are so promising, in fact, that basic science researchers discuss the potential merits of physical training treatment programs to slowing the progression of the disease in humans (Tillerson et al., 2003). While the prospect of slowing the progression of PD is very exciting, initiating treatment early enough to maximize effectiveness may translate into recruiting patients *before* the symptoms are apparent and cause functional disability (Farley et al., 2008). The goal of LSVT is therefore to train patients with early PD. However, recruiting patients who are not experiencing problems is often a challenge.

APPENDIX B

The dorsolateral prefrontal circuit originates in Broadmann's areas 9 and 10 and projects to the dorsolateral caudate nucleus (Selemon & Goldman-Rakic, 1985). From there, projections within the direct pathway terminate directly in the dorsomedial globus pallidus (GP) and in rostral portions of the substantia nigra (SN; Parent, Bouchard, & Smith, 1984), whereas projections from the indirect pathway take a longer route before terminating on these same regions (Mega & Cummings, 1994). The fibers from the GP and SN, respectively, then project to the ventral anterior (Kim, Nakano, Jayaraman, & Carpenter, 1976) and mediodorsal thalamus (Ilinsky, Jouandet, & Goldman-Rakic, 1985). The circuit is then closed by these thalamic projections terminating back onto the origin of the circuits, that is, the dorsolateral prefrontal cortex (Giguere & Goldman-Rakic, 1988). Dysfunction within this circuit is typically associated with executive functions, including perseveration, shifting behavioral sets, planning, and reasoning (Tekin & Cummings, 2002). However, as part of the open connections of this circuit, that is, connections that are not part of the closed loop of the circuit, there are minor afferents to the limbic system, perhaps the anatomical mechanism by which this primarily cognitive circuit influences emotional processing. The dorsolateral prefrontal cortex's role in emotional processing may also involve the representation of goal states towards which affective states are directed (Davidson & Irwin, 1999).

The lateral orbitofrontal circuit originates in Broadmann's areas 10 and 11 and projects to the ventromedial caudate nucleus (Selemon & Goldman-Rakic, 1985). From there, projections within the direct pathway terminate directly in the mediodorsal globus pallidus and the rostromedial substantia nigra, whereas projections from the indirect pathway take a longer route

before terminating on these same regions (Mega & Cummings, 1994). These projections synapse onto the ventral anterior and mediodorsal thalamus (Ilinsky et al., 1985). The circuit is then closed by thalamic projections back onto the lateral orbitofrontal cortex (Ilinsky et al., 1985). Reduced metabolic activity in this circuit, specifically in the orbitofrontal cortex and caudate nucleus, has been observed in Parkinson's patients who have been diagnosed with depression (Mayberg et al., 1990). More generally, dysfunction in this circuit is associated with personality changes that include emotional lability and behavioral disinhibition. Patients may respond inappropriately to social cues and lack sensitivity in interpersonal interactions.

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