THE EFFECT OF THE LEE SILVERMAN VOICE TREATMENT (LSVT) ON EMOTIONAL EXPERIENCE, SOCIAL ENGAGEMENT, AND FACIAL MOBILITY IN PARKINSON'S DISEASE

by

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Abstract

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Dissertation Chairperson: Joan C. Borod, Ph.D.

Research has shown that Parkinson's disease (PD) is associated with emotional processing deficits. The impact of PD on communication and social interaction is gaining appreciation. Although successful treatments exist for motor signs in PD, few exist for the non-motor symptoms. This study examined the impact of a voice treatment (Lee Silverman Voice Treatment [LSVT; Ramig, Pawlas, & Countryman, 1995]) on facial mobility, social engagement, and emotional experience in PD. Fifty-three poser participants (39 PD; 14 demographically-matched healthy controls [HCs]) were studied. The PD posers were assigned to three groups: 12 received voice therapy (LSVT), 14 received articulation therapy (ARTIC; Spielman et al., 2012), and 13 received no therapy (Untreated). All posers were video-taped, before and after treatment, while producing emotional (happy, sad, and angry) and neutral monologues from the New York Emotion Battery (Borod, Welkowitz, & Obler, 1992). Monologues were divided into 15-second segments and evaluated by 18 naïve, yet trained and reliable, raters for facial mobility (amount of non-emotional facial movement) and social engagement (how much the rater wanted to interact with the poser). In addition, immediately following each emotional monologue, posers evaluated three aspects of their emotional experience: (1) intensity of their emotional feelings immediately following the monologue, (2) accuracy with which they carried out the monologue task, and (3) intensity of their emotional feelings throughout the monologue. Results revealed a treatment effect for LSVT, such that PD posers in this group demonstrated improvement in facial mobility, intensity of emotional feelings during the monologue, and immediate feelings after the monologues. Additionally, male posers in the LSVT group reported improved accuracy during the angry monologue following treatment. There were also gender differences; ratings for female posers on facial mobility and immediate emotional feelings were higher than those for male posers. There were no significant results for social engagement. The findings for facial mobility and emotional experience have clinical implications. Enhanced emotional experience may help improve mood disorders that are frequently co-morbid with PD. Further, LSVT might be useful in a broader range of psychiatric disorders. Finally, the findings regarding emotional experience provide exciting avenues for future research.

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Introduction

Parkinson’s disease (PD) was first described in 1817 by James Parkinson. Since that time, it has become the second most common neurodegenerative disorder, following Alzheimer’s disease (de Lau & Breteler, 2006). The prevalence of PD is significant: approximately 1.5 million people have idiopathic PD (e.g., Ramig et al., 2001), with the prevalence and incidence of the disease increasing as individuals age (Tanner & Goldman, 1996; Scheife, Schumock, Burstein, Gottwald, & Luer, 2000). Approximately equal numbers of men and women are affected, although there is still some disagreement in the literature (de Lau & Breteler, 2006). However, there is a difference in prevalence among various races, with Caucasians the most likely to be affected and African-Americans and Asian-Americans the least likely to be affected (Zhang & Román, 1993). The burden of PD on the economy and healthcare system is high. PD costs the United States about $26 billion each year and has been shown to be taxing on the healthcare system, as well as on caregivers (Dodel et al., 1998).

The exact etiology of PD is not well understood. While there have been important advances, the genetics behind PD are not completely known. A great deal of attention has been paid to the environmental causes of PD. Some correlational relationships have been determined between PD diagnosis and various environmental factors (e.g., pesticides, diet, and smoking), but no causal relationships have been demonstrated (Levine et al., 2003; Tanner & Goldman, 1996). However, what is becoming clear is that the etiology of PD is very likely multifactorial, with a complex interaction between genetic and environmental factors (Riess et al., 1998; Veldman, Wijn, Knoers, Praamstra, & Horstink, 1998; Williams, Smith, Waring, & Ramsden, 1999). Perhaps environmental factors act on genetically susceptible individuals as they age (Levine et al., 2003).

A great deal more is known about the neuropathology of PD. It is a progressive neurodegenerative disorder, characterized by a loss of dopaminergic neurons in the substantia nigra of the basal ganglia. Approximately 60-80% of these neurons are lost before signs and symptoms first appear (Riess, Jakes, & Kruger, 1998). Both motor and non-motor signs and symptoms occur in PD. The hallmark motor signs are resting tremor, rigidity, and bradykinesia. The range and impact of non-motor symptoms are becoming more appreciated. Non-motor symptoms include depression and cognitive impairment. Of particular note, there is evidence that some of these non-motor symptoms may occur significantly earlier than the more widely recognized motor signs (Horneykiewicz, 1975; Tolosa, Compata, & Gaig, 2007).

Emotion Processing Deficits in Parkinson's Disease

One non-motor symptom of PD that has received considerable attention in the literature is emotion processing. The three areas of emotional processing that have received the most attention by researchers are the expression, perception, and experience of emotion. According to a recent review of the emotion processing literature (Zgaljardic et al., 2003), the expression of emotion is the most detrimentally impacted in PD. Emotional perception is also consistently deficient. Importantly, emotional experience does not seem to be impaired in PD. In addition to these areas of emotional processing, there are several channels through which emotion can be processed, including face, voice (i.e., prosody), and verbal (i.e., lexical). Most research on emotional expression and perception in PD has focused on the face and the voice. This project focuses on whether an intensive voice treatment changes emotional expression and experience in patients with Parkinson's disease. To that end, the research findings on emotional expression and experience will be described further.

Emotional expression.

Face. Since PD has a large motoric component, the emotional deficits in posed and spontaneous emotional facial expression have been proposed to be differentially affected (Heilman, Blonder, Bowers, & Crucian, 2000). It has been suggested that PD patients would have more deficits in spontaneous facial expression, since PD largely affects subcortical structures, which in turn control involuntary (i.e., spontaneous) motor movement. Specifically, it has been suggested that the basal ganglia plays a large role in spontaneous emotional expression (Blair, 2003). On the other hand, posed (i.e., voluntary) facial emotional expression would be spared in PD patients, since this is largely controlled by cortical areas, which are responsible for voluntary movement. The frontal cortex, in particular, appears to be involved with posed facial expression (Blair, 2003).

While spontaneous facial expressions are consistently found to be impaired in PD patients relative to controls, the results for posed facial expressions have been less consistent, with some studies finding impairments in PD (Jacobs, Shuren, Bowers, & Heilman, 1995; Simons, Ellgring, & Pasqualini, 2003) and other studies not finding deficits, relative to healthy control participants (Borod et al., 1990; Madeley, Ellis, & Mindham, 1995; Smith, Smith, & Ellgring, 1996). Furthermore, PD patients display fewer facial movements and significantly slower movements when producing facial emotional expressions in comparison to healthy controls (Bowers et al., 2006). Researchers have also found individuals with PD to have difficulty in voluntarily moving certain muscles groups, such as the eyebrows and eyelids, as well as the mouth area (Griffin & Greene, 1994; Simons et al., 2003).

Voice. Benke, Bosch, and Andree (1998) found that cognitively intact PD participants were worse than control participants when asked to read sentences with an emotional tone of voice. Other researchers have also found deficits in the expression of emotional prosody in PD

(Borod et al., 1990; Caekebeke, Jenkins-Schinkel, van der Linden, Buruma, & Roos, 1991; Darkins, Fromkin, & Benson, 1988), despite normal perception of emotional prosody.

Emotional experience. Considerably fewer studies have examined emotional experience in PD patients (for review, see Borod & Brickman, 2001). By and large, research has shown that individuals with PD are similar to healthy controls in their subjective experience of emotion (e.g., Maddely et al., 1995; Simons, Pasqualini, Reddy, & Wood, 2004; Smith et al., 1996; Vincente et al., 2011). Most studies have utilized film clips to elicit emotions in study participants. For example, Smith and colleagues (1996) found that PD participants rated their feelings of emotion as similar to control participants after watching film clips intended to elicit specific emotions (happiness, sadness, fear, disgust, and anger). Notably, experience may be preserved over the course of disease. Vincente and colleagues (2011) found no difference between early and late stage PD participants in their ratings of emotion experienced during film clips.

Communication Deficits and Their Treatment in Parkinson's Disease

In addition to emotional processing deficits, individuals with PD also exhibit communication deficits. A growing body of literature suggests that the lack of emotional expressivity in PD leads to various problems in social communication and interpersonal relationships (Brozgold et al., 1998; Katsikitis & Pilowsky, 1996; Pentland, Pitcairn, Gray, & Riddle, 1987; Pitcairn, Clemie, Gray, & Pentland, 1990). Individuals with whom the PD patient is interacting often describe the patient in negative terms, such as "anxious, depressed, and suspicious" (Pentland et al., 1987). PD patients are also often characterized as relating poorly to others, not enjoying social interactions, and as being less likeable (Pentland et al., 1987). Naturally, such social perceptions can adversely impact not only the patient's social interactions with strangers, but also interactions with significant individuals in the patient's life (e.g., family, friends, and health professionals).

Researchers have attempted to understand the nonverbal behaviors that contribute to the perception of individuals as being socially engaged during interpersonal interactions. A number of studies have attempted to delineate what exactly are the positive characteristics of social interactions. Coker and Burgoon (1987) found that greater expressivity in voice, face, and gestures contributes to higher ratings of "conversational involvement." When individuals were asked to demonstrate greater physical and vocal "energy" via facial animation, positive facial expressions (i.e., smiling and laughing), vocal loudness, and variety in vocal pitch, they were perceived as more socially engaged in conversations with their partners compared to earlier baseline conversations. Thus, it follows that improving the facial and vocal/speech expressivity of persons with PD could potentially improve their well-documented problem with social communication and interpersonal relationships.

Although not a great deal of research to date has attempted to treat the lack of facial expressivity in PD, more work has explored the treatment of the vocal and speech deficits commonly associated with PD. To date, there are relatively few successful voice/speech therapies available for PD patients (Schulz & Grant, 2000). Even though 89% of PD patients have voice/speech signs, only 3-4% of patients receive treatment for these deficits (Logemann, Fisher, Boshes, & Blonsky, 1978). Furthermore, more attention has been paid to the treatment of the motor signs of PD (i.e., tremor, bradykinesia, rigidity), and these motor treatments (i.e., medication and surgery) have been relatively successful. However, voice and speech deficits do not improve with these same treatments for motor signs (Schulz & Grant, 2000). The Lee Silverman Voice Treatment (LSVT; Ramig, Pawlas, & Countryman, 1995) is one treatment that has demonstrated significant improvement in dysfunctional speech and voice for PD patients. LSVT is an intensive form of therapy consisting of 16 sessions over one month (four sessions per week). Patients engage in loudness training (i.e., repeating simple sounds as loudly and clearly as possible). Therapists provide frequent prompting and reminders to focus on how it feels and sounds for them to speak more loudly. Therapists also provide feedback to patients regarding their performance with respect to speaking more loudly. The feedback helps to promote “sensory awareness” (Fox, Morrison, Ramig, & Sapir, 2002), regarding the amount of effort and volume needed to achieve the appropriate level of loudness and clarity in voice and speech.

PD produces a number of physical signs within the vocal/speech system that contribute to the vocal/speech problems of PD patients. Just as PD produces reduced and disordered motor movements due to reduced activity in motor circuits, it also seems to produce reduced and disordered functioning of the respiratory and laryngeal muscles subserving vocal/speech production (Baker, Ramig, Luschei, & Smith, 1998). Poor vocal fold closure (i.e., glottal incompetence) contributes to voice/speech dysfunction in PD (Perez, Ramig, Smith, & Dromey, 1996). Although it is not known what causes glottal incompetence, the possibilities include vocal fold rigidity or paralysis (Jiang et al., 1999; Plasse & Lieberman, 1981), decreased muscle or connective tissue volume for the vocal fold, and reduced tension in the thyroarytenoid (TA) muscle due to rigidity of the cricothyroid (CT) muscle (Baker et al., 1998). Also contributing to vocal/speech deficits are monotone voice, poor articulation movements, lack of facial movement, and swallowing disorders.

A wide range of studies has demonstrated an improvement in the vocal and speech deficits noted above in individuals with PD after receiving LSVT. There have been reports of improvements in vocal fold movement following LSVT (Garren, Brosovic, Abaza, & Ramig, 2000; Smith, Ramig, Dromey, Perez, & Samandari, 1995). In comparison to healthy age-matched individuals, two patients with PD exhibited increased TA activity after LSVT therapy (Ramig, 2000). Further evidence finds improvement in the activity of both TA and CT muscles following LSVT (Baker et al., 1998), which would contribute to better vocal fold movement. Researchers have also determined that voice sound pressure level increases post-LSVT (Ramig, Countryman, Thompson, & Horii, 1995) and that progress is maintained one and two years later (Ramig, Countryman, O'Brien, Hoehn, & Thompson, 1996; Ramig et al., 2001).

LSVT also positively alters the orofacial system overall. Dromey and colleagues (1995) noted changes in the speech of patients, which was attributed to improvements in orofacial muscle coordination, leading to better articulation (Spielman, Ramig, Story, & Fox, 2000). Researchers also found PD patients to have better swallowing (El Sharkawi et al., 2002) and tongue strength (Ward, Theodoros, & Murdoch, 2000) after LSVT. The loudness and quality of patients' voices were considered to be enhanced as well (Baumgartner, Sapir, & Ramig, 2001; Ramig & Dromey, 1996).

Of significant clinical value is that the improvements in voice/speech described above are perceived by others. Listeners of PD patients after receiving LSVT treatment indicated that the PD patients have louder and clearer voice/speech (Baumgartner et al., 2001; Ramig et al., 1995, 1996). Furthermore, these improvements are fairly long-lasting. Patients have maintained these improvements for as long as 1-2 years post-treatment (Sapir, Ramig, Hoyt, & Countryman, 1999; Sapir, Ramig, Hoyt, O'Brien, & Hoehn, 2002). These long-term changes provide further evidence for a true neural change occurring as a result of LSVT.

In addition to the benefits provided to voice and speech functioning, LSVT has shown potential in also improving the facial expressivity of PD patients. Spielman, Borod, and Ramig

(2003) found increased facial emotional expression in PD patients who had successfully completed LSVT. Furthermore, this increased facial expressivity was not simply due to changes in mood (i.e., feeling happier) or positive reinforcement secondary to the LSVT (Spielman et al., 2003). Rather, LSVT appears to be offering a treatment method that improves motor and functional abilities in such a way that cognitive effort is minimized (Fox, Ebersbach, Ramig, & Sapir, 2012). In other words, this improved expressivity may be due to neural changes in brain areas related to emotional regulation of the face and possibly even the voice (Fox et al., 2002; Porges, 1995).

According to Ramig and her colleagues, there are valid reasons as to why LSVT would successfully treat voice/speech disorders in PD, despite the lack of success from other voice/speech treatments (Ramig, Bonitati, Lemke, & Horii, 1994). Since LSVT only focuses on increasing the loudness of an individual's voice, it is a straightforward technique. This straightforward quality likely contributes to its efficiency because the variable of loudness has widespread effects across multiple components of the vocal system (Ramig et al., 1994). Fox and her colleagues (2002) theorize that LSVT probably affects the laryngeal, respiratory, and orofacial systems used during speech. Furthermore, since loudness control is a fundamental aspect of speech that is highly practiced and familiar to both humans and animals, LSVT probably accesses areas of the brain storing neural patterns for motor aspects of speech, which may be unaffected in PD patients (Fox et al., 2002). Thus, LSVT is ideal for treating vocal problems in individuals with PD.

Additional support for the use of LSVT in treating voice/speech deficits in PD is found in descriptions of the neuroanatomical areas involved in voice/speech dysfunction. Several studies demonstrate that lesions of the limbic system, thalamus, basal ganglia, or anterior cingulate cortex lead to various vocal abnormalities, such as hypophonia, hypoprosodia, and hypokinetic articulation (e.g., Ho, Bradshaw, Iansek, & Alfredson, 1999; Ho, Iansek, & Bradshaw, 1999; Jürgens & von Cramen, 1982; Meissner, Sapir, Kokmen, & Stein, 1987; Sapir & Aronson, 1985). It is noteworthy that these same areas of the brain are known to be involved in vocal intensity and emotion (Aitken, 1981; Davis, Zhang, Winkworth, & Bandler, 1996; Jürgens & von Cramen, 1982; Jürgens & Zwirner, 1996; Larsen, 1985, 1988; MacLean & Newman, 1988; Muller-Preuss & Jürgens, 1976; West & Larsen, 1995). A very similar neural grouping (the limbic system, thalamus, basal ganglia, periaquedautal gray, and their interconnections) has been proposed as a primitive emotional neural system that is accessed during LSVT (Cummings, Benson, Houlihan, & Gosenfeld, 1983; Fox et al., 2002). Finally, researchers have suggested that this widespread multi-system effect of LSVT may be accomplished by improving the functioning of the fronto-striatal circuits, which are negatively affected by PD (Davis et al., 1996; Devinsky et al., 1995; Larsen, 1985).

Another possible explanation for the effectiveness of LSVT is related to the sensory processing deficits commonly found in PD. It has been well-established that there are a wide range of sensory-related abnormalities included in the nonmotor symptoms of PD (Fox et al., 2002). For example, patients have reported feeling numbness, tingling, pain, and cold or burning sensations (Koller, 1984). Additionally, both neural and behavioral evidence have been gathered in support of these sensory deficits. Boecker and colleagues (1999) reported a decrease in PET activity in response to sensory stimuli in the frontal and parietal lobes and in the basal ganglia of PD participants as compared to healthy controls. Other studies have described PD patients as having problems with orofacial perception (Schneider, Diamond, & Markham, 1987) and with using proprioceptive information when making movements (Jobst, Melnick, Byl, Dowling, & Aminoff, 1997).

The source of the sensory dysfunction described above is theorized to be the basal ganglia, which may be a site for the filtering of sensory information related specifically to movement (Schneider, 1987). As the basal ganglia deteriorate during the course of PD, patients experience more difficulties with appropriately utilizing sensory information as a source of feedback to make correct motor movements (Markham, 1987). Since it has been shown that the voice/speech deficits in PD disappear with external cueing (Ho, Bradshaw, et al., 1999; Ho, Iansek, et al., 1999), a treatment focusing on improving sensory processing has been recommended (Jobst et al., 1997). LSVT is well-suited to address PD patients' sensory deficits and their inability to use sensory feedback, as it emphasizes the patient's awareness of the volume of their voice. Patients also receive a great deal of external cueing from the therapist, as well as practice both during treatment sessions and at home. The ultimate goal of LSVT is to have patients realize that their "normal" voice is too soft and for them to maintain their louder voice on their own (i.e., without any external cueing from a professional or spouse). It is acknowledged that it may be inevitable that patients receiving voice treatment, such as LSVT, revert back to their pre-treatment voice, as it may be too much effort for them to maintain the increased amplitude of their voice (Fox et al., 2002). Although the effects of LSVT appear to last for up to two years post-treatment, there may be a natural deterioration of the treatment effect over time.

Neuroimaging studies are providing evidence for the hypotheses above regarding the neural mechanisms behind LSVT. Liotti and his colleagues (2003) obtained PET scans of PD and healthy matched control participants both before and after LSVT treatment. The imaging was conducted while participants were reading or producing sounds both with and without the experimenter cueing for a louder voice. Before treatment, PD patients exhibited increased activity in their premotor cortex during speech, indicating speech as an effortful and voluntary process. This pattern of brain activity was not seen in the control participants. However, after receiving LSVT, PD patients had increased activity in the basal ganglia, anterior cingulate cortex, anterior insular cortex, and dorsal lateral prefrontal cortex during speech. This pattern of brain activity is indicative of automatic behaviors and is more in line with normalized brain activity. The recruitment of the anterior cingulate cortex and anterior insular cortex (i.e., limbic areas), as well as the dorsal lateral prefrontal cortex, seen in PD patients following LSVT may represent a compensatory neural strategy (Liotti et al., 2003). Additionally, it may also contribute to the improved emotional expressivity seen in individuals with PD after successful LSVT treatment.

Neural Substrates of Parkinson's Disease and Emotion

Pathophysiology of Parkinson's disease. The pathophysiology underlying PD is complex. By the time that clinical signs and symptoms (primarily motor) start to emerge, an entire series of changes has already occurred in the nervous system. Braak and his colleagues (2006) describe a staging model of PD, which outlines the pathological changes throughout the course of the disease. The primary molecular change in PD is the appearance of Lewy bodies (LBs) and Lewy neurites (LNs), which are composed of aggregates of abnormally folded proteins called alpha-synucleins. According to Braak et al., alpha-synucleins first appear in anterior olfactory structures (i.e., the olfactory bulb and olfactory stalk) and in the dorsal motor nucleus of the vagus nerve in Stage 1. As the LBs and LNs continue to increase in density in these areas during Stage 2, additional pathological lesions develop in brainstem nuclei, including the lower raphae nuclei, magnocellular portions of the reticular formation, and the locus coeruleus, which is a significant noradrenergic area of the brain.

Damage to these brainstem areas continues through Stage 3, along with the appearance of LNs and LBs in the amygdala (specifically the central nucleus), magnocellular nuclei of the basal forebrain (e.g., basal nucleus of Meynert), and the pars compacta of the substantia nigra. In Stage 4, the cerebral cortex is first compromised via the anteromedial temporal mesocortex, which is a critical waystation between the neocortical sensory association areas and the limbic system and the prefrontal cortex (Braak, Rüb, Gai, & Del Tredici, 2003). The amygdala is severely affected at this point in the disease. Also, Ammon's horn of the hippocampus is affected so uniquely in PD from Stages 4-6 that postmortem diagnosis can be made on only this characteristic (Braak et al., 2006). Sometimes in Stage 3 and often by Stage 4, clinical diagnosis can be made based on the appearance of the hallmark motor signs.

By the end stages of PD, the Lewy body inclusions reach the neocortex and continue to become more numerous in the mesocortex (Braak et al., 2006). In Stage 5, the LNs and LBs enter the temporal neocortex, as well as higher-order sensory association and premotor cortical areas. Finally, the lesions are found in primary sensory and motor areas (Braak et al., 2006). The Lewy body-containing neurons are mostly in layers V and VI of the neocortex; however, these cortical regions contain numerous projections to the basal forebrain and the basolateral amygdala (Braak et al., 2003).

Recent evidence demonstrates that the types of neurons affected in PD are not random (Braak et al., 2003, 2006; Dauer & Przedborski, 2003). While the normal unfolded type of alpha-synuclein appears in many healthy neurons, not all neurons containing this compound will develop into the pathological form (Braak et al., 2003, 2006). In particular, there appear to be specific morphological characteristics of the neurons that are susceptible to the formation of LBs and LNs: projection neurons with especially long, thin, and unmyelinated or poorly myelinated axons are the most vulnerable to Lewy body pathology (Braak et al., 2003, 2006). It has been proposed that myelin protects the neuron's energy expenditure, thus producing less oxidative stress on the neuron (Braak et al., 2003, 2006). Furthermore, the oligodendroglial cells composing myelin may have a neuroprotective function, which helps to prevent the abnormal folding of alpha-synucleins (Braak et al., 2003, 2006). Although a number of other diseases also have alpha-synuclein pathology, the work by Braak and his colleagues suggest that the characteristic neuronal pathology and the unique neuroanatomical distribution of Lewy body inclusions allows one to differentiate PD from other synucleinopathies. There are also differences in the patterns of pathology in comparison to normal aging. For example, in PD, the loss of neurons in the substantia nigra pars compacta (SNpc) typically starts in ventrolateral and caudal areas, whereas in normal aging the dorsomedial areas are usually affected first (Dauer & Przedborski, 2003).

Traditionally, most of the focus on the pathology of PD has concentrated on the loss of dopaminergic neurons in the SNpc, which projects to the dorsolateral putamen. However, not all areas of the basal ganglia area equally affected by PD (Dauer & Przedborski, 2003). The dopaminergic neurons of the ventral tegmental area (VTA), which are located near the SNpc and mainly project to the caudate, are not affected by PD. Thus, the caudate does not have low levels of dopamine (Dauer & Przedborski, 2003). Furthermore, dopaminergic systems are not the only neurotransmitter systems affected in PD. As shown in Braak's stages described above, other affected systems include noradrenergic (due to locus coeruleus damage), serotonergic (raphae nuclei), and cholinergic (basal nucleus of Meynert) neurotransmitter systems. Unfortunately, the dysfunction of the non-dopaminergic systems in PD is not as well understood in comparison to the dopaminergic system (Dauer & Przedborski, 2003).

The neuroanatomy of emotion. While the neural mechanisms underlying emotion have traditionally implicated the limbic system (MacLean, 1949), more recent neuroimaging studies have indicated that this is far from the only brain area involved in emotion and emotional processing. Wager and his colleagues (2008) describe the following areas as being important to various aspects of emotion: the brainstem (periaqueductal gray and ventral tegemental area); thalamus and hypothalamus; amygdala; basal forebrain; hippocampus; basal ganglia (nucleus accumbens [which is often considered to be part of the limbic system or limbic striatum as well], ventral striatum, and ventral globus pallidus); orbitofrontal cortex; prefrontal cortex; anterior cingulate cortex; anterior insula; and anterior temporal cortex. In fact, evidence suggests that there is no specific brain area or structure that is consistently activated across all emotional tasks (Phan, Wager, Taylor, & Liberzon, 2004). Clearly, emotion is a complex phenomenon, involving numerous neuroanatomical areas and circuitry throughout the brain.

Just as the various theories of emotion take differing approaches to conceptualizing emotion, there are different approaches to the conceptualization of the neuroanatomy underlying emotion. One approach taken by Davidson and his colleagues has been to explore the neural basis of emotional valence (i.e., positive versus negative emotion) in terms of how it is lateralized in the brain. Using psychophysiological methodology, Davidson's program of research has demonstrated that right anterior brain regions are associated with negative emotions and left anterior regions with positive emotions (Davidson, 1992). These anterior regions are commonly referred to as the prefrontal cortex (PFC; Davidson & Irwin, 1999). Other areas involved in the processing of emotional valence include the ventral striatum (composed of the caudate, putamen, and nucleus accumbens of the basal ganglia) for positive emotion (e.g., Sutton et al., 1997) and the amygdala for negative emotion (Davidson & Irwin, 1999). Interestingly, a meta-analysis by Murphy and her colleagues found that the emotions of happiness and sadness did not consistently activate a particular neuroanatomical area across neuroimaging studies (Murphy, Nimmo-Smith, & Lawrence, 2003), perhaps providing further evidence for a generalized location for positive and negative emotions.

Another approach in the neuroimaging literature for emotion has been to differentiate among the brain areas underlying specific emotions and the areas related to processing emotion in general. In their review of the literature, Murphy and her colleagues (2003) uncovered two areas for the general processing of emotion: the medial prefrontal cortex and the anterior cingulate cortex (ACC). The medial PFC has been described as having an important role in emotion regulation and cognitive processes related to emotion (Phan et al., 2004). The ACC is also involved in regulating cognitive and emotional processing, recalling emotional memories, and using attentional resources to process emotional stimuli (Lane, Fink, Chau, & Dolan, 1997; Phan et al., 2004).

A number of structures have been identified in the processing of specific types of emotion. Probably the most consistent in reviews of the literature has been the activation of the amygdala during fear (Davidson & Irwin, 1999; Murphy et al., 2003; Phan et al., 2004). More precisely, the amygdala is involved primarily in the expression and recognition of fear (Davidson & Irwin, 1999). Its activation has also been related to negative emotions in general, as well as more non-specific stimuli characteristics, such as the salience or the degree of threat found in the emotional stimulus (Davidson & Irwin, 1999; Phan et al., 2004). Finally, there is evidence that the amygdala can operate at the unconscious level. For example, studies have shown amygdala activity after the presentation of emotional stimuli, even if the participants did not perceive a fearful stimulus or report feeling fear subjectively (Morris, Ohman, & Dolan, 1998; Whalen et al., 1998). The lateral orbitofrontal cortex has been shown to be activated with anger (Murphy et al., 2003), and the insula and globus pallidus of the basal ganglia are important in the processing of disgust (Murphy et al., 2003; Phan et al., 2004). The neuroanatomical correlates of disgust fit with the findings of Davidson and Irwin (1999), who reported the insula as being related to the processing of autonomic and visceral types of emotion, of which disgust is a perfect example. Phan et al. (2004) also reported internal emotions and aversive or threatening stimuli as activating the insula.

Why emotional processing deficits might occur in Parkinson's disease. A number of brain areas affected in PD are also involved in emotion, leading to a possible explanation for at least some of the emotional processing deficits present in PD. The brainstem is one area that has been found to be related to both PD (Braak et al., 2006) and emotional processing (Wager et al., 2008); but, different areas of the brainstem appear to be involved in each. In emotion, the periaqueductal gray and the ventral tegmental area are the critical locations (Wager et al., 2008), whereas the raphae nuclei, reticular formation, and locus coeruleus are the location of pathology in Stage 2 of PD (Braak et al., 2006). As described above, Stage 3 of PD results in damage to the amygdala, basal forebrain, and the substantia nigra pars compacta of the basal ganglia (Braak et al., 2006). Also, once the final stage of PD is reached, pathology affects the primary sensory and motor areas, which in turn project to the basal forebrain and parts of the amygdala (Braak et al., 2006). Wager and colleagues (2008) have implicated both the basal forebrain and the basal ganglia in emotion. However, in terms of the basal ganglia, they state that the nucleus accumbens, ventral striatum, and globus pallidus are involved in emotion, whereas Braak et al.

(2006) only implicates the substantia nigra in PD. Although it is the temporal cortex that is affected in Stages 4 and 5 of PD (Braak et al., 2006), there is overlap with emotional functioning, as the anterior temporal cortex has been shown to be active during emotion (Wager et al., 2008). The prefrontal cortex is involved in general emotion functioning, such as emotion regulation (Phan et al., 2004; Wager et al., 2006), as well as positive emotion in the right hemisphere and negative emotion in the left hemisphere (Davidson & Irwin, 1999). PD indirectly impacts the prefrontal cortex via pathology in the anteromedial temporal mesocortex, which connects the sensory association cortex and the prefrontal cortex and limbic areas (Braak et al., 2006). A number of the areas typically considered to be part of the limbic system are damaged during the progression of PD. Finally, there is a high incidence of depression and anxiety in individuals with PD (Aarsland et al., 1999), with increasing evidence that depression occurs usually 3-6 years before the diagnosis of PD (Shiba, et al., 2000). Moreover, it is likely that depression in PD is not simply a psychological reaction to the disease but has a biological basis itself (Tolosa, Compata, & Gaig, 2007).

The Relationship Between Facial and Vocal Emotional Expression: Why LSVT Improves Facial Emotional Expression and Communication Deficits in PD

There are several pieces of behavioral and physiological evidence pointing to a direct relationship between facial and vocal emotional expression. Correlations between these two expressive modes have been found in both brain-damaged and healthy adult populations. There is general support for a correlation between facial and prosodic emotional expression (e.g., Borod, Koff, Lorch, & Nicholas, 1985; Malatesta, Davis, & Culver, 1984; Mehrabian & Wiener, 1967). Borod et al. (1990) found a correlation between facial and prosodic emotional expressions in both healthy adults and selected neuropsychiatric populations (i.e., right brain-damage, Parkinson's disease, schizophrenic, and unipolar depression). Other studies have corroborated this finding in brain-damaged populations (e.g., Borod et al., 1985; Ross & Mesulam, 1979). In addition to emotional expression, researchers have also found correlations between the modes of face and voice for emotional perception as well. Again, this applies to both healthy (e.g., Borod et al., 1990, 1998, 2000; Massaro & Egan 1996) and brain-damaged populations (e.g., Borod et al., 1990; Zgaljardic, Borod, & Sliwinski, 2002). However, it is also important to note that there are studies that have not found an association between facial and vocal emotional expression. For example, Bowers and colleagues propose that face and voice are controlled by separate neural systems (Bowers, Bauer, & Heilman, 1993).

In addition to the behavioral evidence described above, there is also strong physiological evidence for a relationship between facial and vocal emotional expression. First, facial and vocal expression share a number of neural substrates, including the anterior cingulate cortex (ACC), the periaqueductal gray, the basal ganglia, and the thalamus (Jürgens & Zwirner, 1996; Phan et al., 2004; Ploog, 1992; Wager et al., 2006). Facial expression largely involves the basal ganglia, reticular formation, and the ACC. The basal ganglia, which is an evolutionarily old brain structure, has long been implicated in movement. It likely evolved from a structure mainly responsible for drive-related behavior to a structure involved in more complex motor-, cognitive-, and emotion-related behaviors (Rinn, 1984). Spontaneous facial emotional expressions probably originated as one of a number of automatic physiological responses from these phylogenetically old neural circuits and were retained over time to be used for appropriate social communication (Rinn, 1984). These circuits then became the basal ganglia, with multiple cortical and subcortical connections, allowing for complex influences and modifications of motor, cognitive, and emotional behaviors (Rinn, 1984). The reticular formation directly modulates the movement of the facial muscles responsible for spontaneous expressions and is involved with behavioral arousal as well (Rinn, 1984). The ACC has been implicated in movement related to emotion (Devinsky, Morrell, & Vogt, 1995) and appears to integrate the basal ganglia and the reticular formation for spontaneous emotional facial expressions.

Vocal expression is mainly regulated by the periaqueductal gray (PAG). The PAG produces species-specific vocalizations (Jürgens & Zwirner, 1996; Ploog, 1992) and appears to be specially used for emotional vocalizations (Larson, 1985). For example, although vocal fold vibration will continue when the PAG is chemically blocked during neocortical stimulation, it does not vibrate when other known emotional vocalization areas, such as the ACC, are stimulated (Jürgens & Zwirner, 1996). The control of vocal fold movement occurs via two pathways (Jürgens & Zwirner, 1996). One is a neocortical route, which concerns voluntary vocalizations and consists of direct connections between the facial motor cortex to the laryngeal motoneurons of the brainstem. The other route is via the limbic system and is more related to spontaneous expression. It projects from the ACC to the PAG and other limbic structures, and then, as in the neocortical route, to the laryngeal motoneurons of the brainstem. Thus, the PAG appears to be critical for the movement of the vocal fold when it is specifically related to emotional vocalization. The ACC has been described as the beginning point of the emotional vocal pathway (Ploog, 1992).

Because the neuroanatomy underlying vocalizations and facial expression is shared, it follows that engaging in LSVT will directly affect facial expression. Indeed, individuals with PD who receive LSVT demonstrate increased facial movement at the end of treatment (Spielman et al., 2003). Additionally, there is evidence that increasing facial and vocal expressiveness leads to the perception of more involvement in social interactions (Coker & Burgoon, 1987). Thus, LSVT could potentially improve interpersonal communication deficits, as well as facial expression impairments commonly associated with PD.

Overview of Current Study

The overall aim of this project was to examine the effects of LSVT on facial mobility, social engagement, and emotional experience in individuals with Parkinson's disease. Preliminary data suggest that LSVT improves facial movement and social engagement (Spielman et al., 2003). In the current study, three groups of PD patients were included: participants receiving LSVT, participants receiving a control voice treatment (ARTIC), and a control group of PD participants receiving no treatment. There was also a group of demographically-matched healthy control participants, who did not receive treatment during the course of the study. Participant gender was included as an independent variable, since men and women are known to process emotion differently.

Five dependent variables were examined in this project: facial mobility, social engagement, and three components of emotional experience. Because preliminary research has demonstrated that LSVT affects facial movement (Spielman et al., 2003), facial mobility was utilized as a measure of pure facial muscle movement. Social engagement was intended to be a measure of interpersonal interaction (i.e., how much would one like to interact with the individual). Additionally, emotional experience was examined, given the lack of research in this area regarding this variable with respect to Parkinson's disease. Emotional experience was to be explored more carefully and systematically by examining three different aspects: Immediate Feelings, Intensity, and Accuracy. More detailed descriptions of these components are provided in the Methods section.

To our knowledge, emotional experience has not been previously evaluated in this manner. Therefore, analysis of the three components of this variable was primarily exploratory in nature, and there were not specific hypotheses for each component of experience. There were, however, predictions for emotional experience as a whole, which are outlined below.

Aims and Hypotheses

The following aims and hypotheses were proposed.

Aim 1. Examine the differences at baseline (i.e., pre-treatment) between the Parkinson's disease (PD) groups and the healthy control (HC) group on facial mobility (FM), social engagement (SE), and emotional experience (EE).

Hypothesis 1. The participants in the PD groups will have lower ratings of FM and SE at baseline in comparison to the HC group participants.

Hypothesis 2. The participants in the PD groups will have similar ratings of EE at baseline in comparison to the HC group participants.

Aim 2. Examine FM, SE, and EE from pre-treatment to post-treatment time periods across all groups.

***Hypothesis 3*.** The LSVT group will improve significantly in FM and SE ratings from the pre- to post-treatment time periods. The ARTIC group will also improve significantly in FM and SE ratings from the pre- to post-treatment time periods, but less so than the LSVT group. The PD Untreated and HC groups will not significantly change over time. EE ratings will not change across group or time period.

Aim 3. Examine the influence of participant gender at each time period (pre- and post-treatment) for SE and EE. There is considerable support for gender differences in emotional processing (e.g., Borod & Madigan, 2000). That is, in general, women have been found to be better than men on tasks involving emotional processing.

***Hypothesis 4*.** Male participants will have lower ratings of SE and EE in comparison to the female participants.

Aim 4. Examine emotional valence (positive vs. negative emotion) across time for SE and EE.

***Hypothesis 5*.** Because PD patients tend to look more negative in their emotional expressions compared to healthy individuals, it is expected that the negative monologues for PD participants will exhibit a greater improvement over time than will either the positive or neutral monologues for SE. It is hypothesized that EE will remain the same.

Aim 5. Examine the potential mechanism behind LSVT by comparing the interaction effects of EE with FM and SE.

***Hypothesis 6*.** If LSVT is indeed affecting emotion systems, then EE will demonstrate the greatest change over time, with moderate change in SE and no change in FM over time.

***Hypothesis 7*.** If LSVT is affecting motor systems rather than emotion systems, then the greatest change in ratings over time will occur for FM, with moderate changes in SE and no change in EE over time.

Aim 6. Examine the association of EE (i.e., posers' experience during the monologue) with FM and SE.

***Hypothesis 8*.** If PD has a true emotional processing deficit component (i.e., emotional deficits are in addition to the motor deficits), then there will be a greater positive correlation between EE and SE than between EE and FM.

***Hypothesis 9*.** If PD is purely a motoric disorder (i.e., emotional deficits are result of the motor deficits and not a separate symptom), then there will be a moderate positive correlation between EE and SE and no correlation between EE and FM.

Method

Participants

There were two groups of participants in this study: posers and raters. Posers were the individuals from whom the experimental emotion data were collected (i.e., the three PD and one HC participant groups). The raters were individuals who had been recruited and trained to evaluate the experimental data on the five dependent variables (i.e., FM, SE, and the three EE variables).

Posers. There were 57 posers in total: 43 patients diagnosed with idiopathic Parkinson's disease and 14 healthy, age-matched control participants. The posers were divided into four participant groups, consisting of one experimental group (14 individuals with PD receiving LSVT) and three control groups (14 individuals with PD receiving an articulation treatment [ARTIC], 15 individuals with PD receiving no therapy [Untreated], and 14 healthy individuals receiving no treatment [HC]). The purpose of the ARTIC group was to control for the effects of changes related to receiving a treatment over time, whereas the Untreated group was included to control for changes over time due to the effects of having PD as well as being in treatment.

Finally, the HC group was included to account for changes in participants due to the passage of time.

All posers were screened on an extensive number of medical, psychiatric, and cognitive variables. Inclusion criteria requirements are: age 45-85 years; diagnosis of idiopathic PD by a neurologist; no or mild dementia, based on Mini Mental State Examination (MMSE) score of 27 or higher (Folstein, Folstein, & McHugh, 1975); no, mild, or moderate depression, based on Beck Depression Inventory (BDI-II) score of less than 29 (Beck, Steer, & Brown, 1996); and diagnosis of mild, moderate, or severe speech/voice disorder by a speech-language pathologist. Exclusion criteria include characteristics that might interfere with voice/speech treatment or confound the effects of voice/speech treatment during the course of the study. Thus, any disorders or conditions that affect the orofacial area (e.g., head, face, jaw, or mouth) or vocal system (e.g., neck, throat, tongue, or lungs) were incorporated. In addition, any characteristics that may affect mood or emotion were also included in order to ensure that measures of emotion were not potentially confounded by the presence of mood disorders. The complete list of exclusion criteria were as follows: diagnosis by a neurologist of neurological signs and symptoms, other than those due to PD; history of neurological surgery, other than as for treatment for PD; head or neck cancer; gastrointestinal disease, as diagnosed by an otolarynologist; history of gastrointestinal surgery; severe temporomandibular joint (TMJ) disorder; diagnosis by speech-language pathologist of speech-language disorder not due to PD; diagnosis by otolaryngologist of laryngeal pathology; laryngeal surgery; intensive speech treatment in the past two years; smoking in the last four years; substance abuse; severe depression, as assessed by the BDI-II; and moderate to severe dementia, as assessed by the MMSE.

Poser participants were recruited in the Boulder and Denver, Colorado areas via physician referrals, support groups, aging centers, and through advertisements posted in newspapers, newsletters, websites, email listservs, and educational and research facilities. Participants were compensated $10 for completing the screening procedures and $40 for completing the experimental tasks.

Raters. The raters consist of 18 students from the Queens College community, who were recruited via campus flyers and classroom announcements. Due to known gender differences in emotion perception (e.g., Brody & Hall, 1993), equal numbers of male and females raters were recruited. Inclusion criteria were: age 20-30; right-handed; Caucasian; and native English speakers or learned English by age seven. Because research indicates "in-group" biases in emotion perception (e.g., Elfenbein & Ambady, 2002), only Caucasian individuals were recruited as raters, since the ethnicity of the posers is largely Caucasian. All raters were screened for medical, neurological, psychiatric, and drug use history. Exclusion criteria are: history of neurological disorder or head injury; history of psychiatric disorder; learning disability; substance abuse; and uncorrected visual problems. All raters were compensated $9 per hour for their participation in the study. Participants were recruited and consented according to IRB-approved procedures, and they had completed all IRB-required certifications for working with human subject data.

Materials and Procedures

Collecting monologues from posers. All posers were evaluated at two time periods: pre-treatment and immediately post-treatment. All participants were tested and receive the voice treatments at the University of Colorado. Each poser was tested individually. At each time period, posers were administered the emotional (happy, sad, and angry) and non-emotional (going to the supermarket) monologue tasks from the New York Emotion Battery (NYEB; Borod, Welkowitz, & Obler, 1992). For the emotional monologues, participants were asked to recall a time that they felt the target emotion (happy, sad, and angry) with great intensity. They were then requested to try and re-experience the emotional event as vividly as possible, providing as much detail as they can during their monologue. The instructions for the non-emotional monologue are similar. Participants were asked to remember and reconstruct the last time that they went to the supermarket in as much detail as possible. After providing these instructions, the experimenter read aloud examples of emotional and non-emotional monologues. The participants were provided with some time to recall the event that they will speak about. If necessary, there were standard probes that the experimenter might provide to help the participant choose or remember an appropriate event. Participants were to speak about their personal emotional or non-emotional experiences for at least 90 seconds per monologue. The experimenter provided cues to the participant if their monologues are too short or are getting too long.

Experimental sessions were videotaped in order to analyze facial expressions. While producing the monologues, participants were located in a sound-proof booth and seated in a dental examination chair equipped with a headrest. Posers were video-recorded using a Canon XL1S mini-DV camera. The participant was seated approximately nine feet away from the camera, which was zoomed in to capture as much of the head, face, and neck as possible. The experimenter was seated next to the video camera, in order for the participant to look directly into the lens and to provide a picture of their entire face. Deidentified poser participant data were sent to Queens College on DVDs for analysis.

Collecting emotional experimental data from the posers. In order to more thoroughly and systematically evaluate the subjective emotional experience of the poser participants during and after completing the monologues, EE was divided into three component parts. After generating each monologue, poser participants were asked to rate how they felt while speaking about their emotional and non-emotional experiences. Using a seven-point Likert scale (1 is minimum and 7 is maximum), posers indicate: (a) the intensity of their emotional feelings immediately after completing each monologue, (b) the accuracy with which they carried out the recollected experience during the monologue, and (c) the intensity of their feelings throughout the monologue.

Rater evaluation of monologues. At Queens College, the videotaped data were evaluated by trained raters on five dependent variables: facial mobility (how much the face makes non-emotional movements); emotional frequency (the amount of emotional expression); emotional intensity (the strength of the emotion); emotional variability (the amount of different emotional expressions); and social engagement (the likeability of the poser). Each variable was rated on a seven-point Likert scale, with 1 as minimum and 7 as maximum. Due to the broad scope of the larger project, the study presented here focused on the facial mobility and social engagement variables. Facial mobility was intended to determine the degree of facial movement overall. Social engagement was designed to evaluate a specific aspect of interpersonal interactions, namely how interested a person is in interacting with the poser.

Each monologue was divided into 15-second segments for rating purposes. Using video editing software (Corel Video Studio ProX3), any irrelevant parts of the monologue (e.g., experimenter providing instructions to participant) were removed from the beginning and end of each monologue. Monologues were then divided into 15-second segments, starting from the end of the monologue and working backward. Often, a monologue would not divide evenly into 15 second segments (i.e., there would be a segment at the end of this process that would be less than 15 seconds long). In these cases, the “short” segment was discarded. Previous research in our lab has shown that the most emotionally intense sections of these monologues are at the middle and end of the monologue (Kazandjian, Borod, & Brickman, 2007). Therefore, all discarded portions of the monologue that were too short to analyze (i.e., from the very beginning of the monologue) were likely the least emotionally intense portions of the monologue.

Due to the extensive amount of data to be evaluated, the posers’ monologues were rated by three separate cohorts of raters. Each cohort consisted of six raters, who were naïve to the hypotheses of the study. All raters were trained on the definitions of the dependent variables and the rating scale, and they had opportunities to practice rating monologue segments. Every attempt was made to have all six raters in attendance at each training session. When schedules conflict, as many raters as possible were trained together as a group, and the raters with scheduling conflicts were trained together to the criteria of the larger group.

Raters were trained based on the procedures outlined in Canino, Borod, Madigan, Tabert, and Schmidt (1999). Each training session lasted approximately two hours and consisted of four phases: Defining the variable, Exemplar presentation, Conferencing, and Inter-rater reliability. During the first phase, the dependent variable to be rated is introduced and defined. The experimenter provided a detailed description of the facial characteristics that are involved in relation to the dependent variable and provided demonstrations as appropriate. The experimenter also showed the rating scale and provided the raters with a short description of each point on the scale. The Appendix contains excerpts (variable definition and rating scale) from the training materials for facial mobility and social engagement.

During the exemplar presentation, the experimenter showed two video clips (one male, one female) as examples for each point on the scale. Since the rating scale was a seven-point Likert scale, rater participants saw 14 total exemplars. The exemplars were presented one at a time, with an explanation as to why that particular clip was a good example of that point on the rating scale. Raters were allowed to view the clip as often as necessary and were provided opportunities to ask questions about the reasoning behind the ratings. The exemplars were presented on a desktop computer using the Superlab program (version 4.5 Pro), and the video image was shown on a flat panel LCD monitor.

For the conferencing stage, the raters viewed 12 video segments and provided ratings for each segment independently. Video clips were randomized according to gender, participant group (i.e., Parkinson's disease or healthy control), emotion (i.e., one of the emotions from the NYEB), and rating (i.e., 1-7 on Likert scale). Raters were shown each of the 12 segments one at a time. They were able to view the clip up to three times. After viewing the clip, they wrote down their rating on a record form. When everyone had completed their rating, each rater stated their rating aloud to the group. If all ratings were within two points of each other, then the next video segment was shown. If there were greater than a two-point discrepancy among the raters, then the experimenter facilitated a discussion about how the raters decided upon their rating. Each rater was encouraged to explain why they chose their rating, and the clip was viewed again as needed during the discussion. The experimenter ensured that all raters were utilizing the scale and variable definitions appropriately. After the discussion was completed, raters were asked to view the same clip again and to provide a second rating, that is, they could either keep their original rating or make a different one based on the discussion.

The final phase of training was inter-rater reliability. For this stage, the raters viewed 40 video segments and made their ratings independently without sharing or discussing their ratings with each other. As in the conferencing stage, video segments were randomized for gender, participant group, emotion, and rating. Raters were able to watch the video clip up to three times. After all segments had been rated, the raters were given a short break, during which time the experimenter entered the ratings into a data analysis program (SPSS) and conducted a one-way random intra-class correlation (ICC). If the ICC value were equal to or greater than 0.80, then the raters had successfully completed the training. All raters met criteria for successful training. Even if the training criterion were met, the experimenter reviewed the ratings of all six raters. Any segments that were discrepant by more than two points were reviewed again with the group, using the conferencing procedures.

Raters were trained on one dependent variable at a time. After they were successfully trained, they were scheduled to complete their ratings of the experimental data on that variable for approximately the next two to three weeks. The raters completed the ratings of the experimental data at their convenience, but for no more than three hours at a time in order to prevent fatigue. The ratings took place in a quiet lab room equipped with two desktop computers on separate desks. There could be one or two raters working at any time. All raters were supervised by trained research assistants, who were present to answer any questions or resolve any technical problems that emerged (e.g., an error message). Raters were asked to take frequent breaks as needed. They were also instructed to not listen to music or otherwise distract themselves while completing the ratings.

Ratings were made using a video presentation program created in Microsoft Access 2007. This program presented each video clip with the rating scale underneath it. After the video was over, the rater could view it again (up to three times) or provided their rating by clicking on the appropriate point of the scale. Monologue segments were randomized by gender, participant group (i.e., LSVT, ARTIC, Parkinson's disease untreated, or healthy control), emotion (happy, sad, angry, neutral), and time period (pre or post). The video images were presented on the same flat panel LCD monitor as during the training sessions.

Results

Participant Demographics

Posers. Demographic characteristics of the poser participants are presented in Table 1. Poser groups (LSVT, ARTIC, Untreated, and HC) were matched as closely as possible according to age, gender, ethnicity, education, and Hoehn and Yahr (Hoehn & Yahr, 1967) disease stage. Information regarding time since patient diagnosis for PD, pre-treatment MMSE score, and pre-treatment BDI-II score were also available. Age, education, disease stage, time since diagnosis, MMSE score, and BDI score were separately analyzed using a one-way ANOVA. The four poser participant groups did not significantly differ from each on age ($F[3, 49] = .379$, $p = .769$); education ($F[3, 46] = .693$, $p = .561$); disease stage ($F[2, 36] = 1.063$, $p = .356$); time since diagnosis ($F[2, 36] = .557$, $p = .578$); or MMSE score ($F[3, 49] = 1.627$, $p = .195$). Participants did differ on the BDI score (F $[3, 49] = 4.917$, $p = .005$), such that the LSVT participants (M = 10.50, SD = 6.46) endorsed more depressive symptoms than did the HC participants (M = 3.21, SD = 3.68). The other group comparisons were not significant. Of note, the mean BDI score for the LSVT participants was only in the mild range of depression.

Gender could not be analyzed using the Chi square statistic or Fisher's exact z test, due to the small cell sizes. Visual inspection of the data showed that each of the four participant groups had nine men; however, there were fewer women in each participant group (three in the LSVT group, five in the ARTIC group, four in the Untreated group, and five in the HC group). Ethnicity also was not able to be analyzed statistically due to small cell sizes. All participants in our sample were Caucasian, except for two Hispanic individuals in the LSVT group.

Raters. Demographic information for the rater participants can be found in Table 2. Equal numbers of male and female Caucasian raters were recruited for the three cohorts of the study. Because the majority of the poser participants were Caucasian, the raters consisted exclusively of Caucasian individuals. The research literature shows that individuals have more difficulty perceiving and interpreting the emotional expressions of members from cultural groups outside their own (Elfenbein & Ambady, 2002). Participants within each cohort were matched for age and education.

Inter-Rater Reliability for Training and Experimental Data

For Facial Mobility (FM) and Social Engagement (SE), intra-class correlations (ICC) were conducted to evaluate the inter-rater reliability among our raters. Four sets of reliability analyses were conducted: two sets during rater training (conferencing and inter-rater reliability stages of training), one set for the ratings of the experimental data for each cohort, and one set for each variable for the entire dataset (i.e., across all three cohorts). Results for the training reliability analyses are displayed in Table 3. The raters had high agreement for their ratings of FM and SE in all three cohorts. Table 4 displays the reliability analyses for the experimental data. Raters had high agreement with each other within the majority of cohorts. The inter-rater reliability in Cohort 2 on SE was somewhat low, but still in the acceptable range.

Tests of Normality, Homogeneity of Variance, and Data Transformations

In order to determine whether parametric or non-parametric analyses were appropriate, all collected experimental data were examined using the Shapiro-Wilk tests of normality

(Shapiro & Wilk, 1965) and Levene's test of the homogeneity of variance (Levene, 1960). Shapiro-Wilk tests were performed on all dependent variables (Facial Mobility [FM], Social Engagement [SE], Emotion Experience – immediate feelings [EE-F], Emotion Experience – overall intensity [EE-I], and Emotion Experience – accuracy [EE-A]), with comparisons across time (pre- and post-treatment) and monologue type (happy, sad, angry, and/or neutral) for all four participant groups (Articulation therapy [ARTIC], LSVT therapy [LSVT], PD untreated [Untreated], and healthy controls [HC]) and for all PD groups together (ARTIC, LSVT, and Untreated). SE was normally distributed, but all other dependent variables were not normally distributed. Table 5 displays the Shapiro-Wilk statistics for all five dependent variables before transformation procedures were performed. Attempts were made to normalize FM and all three EE variables using square root and logarithmic (natural log and log10) transformations. Although natural log transformations successfully normalized the distributions for FM, all attempts to normalize the distributions for EE-F, EE-I, and EE-A were unsuccessful. Therefore, whenever possible, non-parametric procedures were used to analyze the emotional experience variables. The Shapiro-Wilk statistical values following natural log transformation for all five dependent variables are shown in Table 6.

In addition, tests for homogeneity of variance were conducted on SE to ensure that assumptions for homogenous variances were upheld for analyses of variance (ANOVAs). The Levene statistical test was conducted across time (2), monologue type (4), and participant group (5) as described above. Out of the eight comparisons conducted for each of the 5 participant groups, there was only one significant comparison for SE, which is well below chance expectations. Thus, parametric statistical procedures were used to analyze the SE data.

Aim 1: Differences at Baseline (Pre-Treatment) for PDs and HCs on All Dependent Variables

Hypothesis 1. It was predicted that the PD participants would have lower ratings of FM and SE at baseline in comparison to the HC participants. To examine FM and SE, a mixed-design repeated-measures ANOVA (Group [2: PD & HC] x Monologue Type [4: Happy, Sad, Angry, and Neutral]) was conducted for FM and SE on the pre-treatment data, separately, for each dependent variable. Significant values ($p \leq .05$) and trends ($.05 < p \leq .10$) are reported for all analyses in this section and for all following sections in the Results. For FM (see Figure 1), there was a significant main effect of Group ($F[1, 51] = 5.719$, $p = .021$), such that PD participants were rated lower on FM ($M = .84$) than were HC participants ($M = 1.07$). There was also a significant main effect of Emotion for FM ($F[3, 53] = 2.826$, $p = .041$). Pairwise comparisons indicated a trend ($p = 0.65$) for angry monologues ($M = 1.01$) being rated as more mobile than neutral monologues ($M = .93$). There were no significant main effects or interactions for the SE ratings.

Hypothesis 2. PD and HC groups were predicted to have similar ratings of EE (i.e., for the three emotional experience ratings) at baseline testing. Due to non-normal distributions, even after transformations, the Kruskal-Wallis test was conducted. Separate one-way ANOVAs on Group (2: PD & HC) were conducted for each of the three experiential ratings, separately, for each of the three monologue types at the pre-treatment testing time (i.e., baseline). There were no significant main effects of or trends for Group for the sad or angry monologues for any of the three EE variables, providing support for this hypothesis. However, there were main effects of Group for the happy monologues for all three EE variables (see Figure 2). For immediate feelings following the monologues (EE-F), the PD participants (mean rank = 23.08) indicated significantly less intense feelings than did the HC participants (mean rank = 32.38) after the happy monologues (χ^2[1] = 4.247, *p* = .039). Further, the PD participants (mean rank = 22.61) endorsed significantly less overall emotional intensity (EE-I) during the happy monologues compared to the HC participants (mean rank = 33.73; χ^2[1] = 5.985, *p* = .014). Finally, there was a trend for PD participants (mean rank = 23.27) reporting that they were less accurate (EE-A) during their happy monologues in comparison to the HC participants (mean rank = 31.85; χ^2[1] = 3.541, *p* = .060).

Aim 2: Effects of Time (Pre- and Post-Treatment) on Ratings of FM, SE, and the Three EE Variables for All Four Participant Groups

For Aims 2-4, a mixed-design repeated-measures 4-way ANOVA (Group [4] x Gender [2] x Time [2] x Emotion [4]) was performed for FM and SE. In order to determine the effects of time for the three EE variables, parametric procedures (i.e., the 4-way ANOVA described directly above) were also used as within-subject variables cannot be evaluated using the Kruskal-Wallis test. Of note, analyses of variance have been shown to be relatively robust to violations of non-normality (Lix & Keselman, 1998; Ramsey, 1994).

Hypothesis 3. It was predicted that the LSVT group would significantly change over time in FM and SE compared to the other three participant groups. Further, ARTIC was predicted to also change over time, but less significantly than the LSVT group. The PD Untreated and HC groups were predicted not to change significantly over time. This hypothesis was examined through the Group x Time interaction (see Figure 3), which showed a trend for FM (*F*[3, 45] = 2.470, *p* = .074) but not significant for SE. Post-hoc pairwise comparisons for FM indicated that the LSVT group participants were rated as significantly more facially mobile post-treatment (*M* = .96) than pre-treatment (*M* = .87; *p* = .036). Also, there was a trend for

Untreated participants to have fewer facial movements post-treatment (*M* = .95) compared to pre-treatment (*M* = 1.01; *p* = .10). Thus, with regard to FM, LSVT did change significantly over time on facial movement. As predicted, the HC group did not significantly change over time on FM. However, contrary to expectations, facial movement in the Untreated participants tended to decrease over time. The ARTIC group did not change over time, contrary to expectations. Of note, there were no other significant interactions involving Group or Time for FM or SE.

Although we did not hypothesize the posers to exhibit significant changes in emotional experience over time, there were significant findings or trends for Group x Time interactions for immediate emotional feelings (EE-F) and overall emotional intensity (EE-I) following monologue production. These interactions are displayed in Figure 4 for EE-F and in Figure 5 for EE-I. With regard to EE-F, there was a trend for the Group x Time interaction ($F[3, 42] = 2.327$, $p = .088$), such that the LSVT participants endorsed significantly greater immediate emotional feelings post-treatment ($M = 5.59$) than pre-treatment ($M = 4.83$; $p = .008$). Furthermore, a trend for EE-F for Group x Gender x Time ($F[3, 42] = 2.384$, $p = .083$) revealed a significant increase in immediate emotional intensity for women in the LSVT group from pre- ($M = 4.56$) to post-treatment time ($M = 6.11$; $p = .002$).

A significant Group x Time interaction was also found for EE-I ($F[3, 40] = 6.530$, $p = .001$). Again, the LSVT group participants described greater feelings of emotion ($p = .005$) throughout the monologue at post-treatment ($M = 5.78$) than at pre-treatment ($M = 5.07$). Further, the HC poser participants had less ($p = .016$) overall emotional intensity at post-treatment ($M = 5.40$) than at pre-treatment ($M = 5.94$).

Aim 3: Gender Effects Over Time for SE and EE Across All Four Participant Groups.

Hypothesis 4. Women were expected to have higher ratings of SE and EE in comparison to men at each testing time. Although there were no significant main effects of or trends for Gender for SE, EE-A, or EE-I, there was a significant main effect of Gender for EE-F (F[1, 42] = 5.478, p = .024), with female participants (M = 5.59) endorsing more intense emotional feelings immediately after the monologue compared to male participants (M = 4.99), irrespective of participant group (see Figure 6). In addition, there was a significant Gender x Time interaction for EE-F (F[1, 42] = 4.093, p = .049). At post-treatment, women had significantly (p = .004) greater feelings of emotion directly after the monologues (M = 5.80) than did the men (M = 4.95), despite having relatively similar levels of emotional feelings at pre-treatment (female M = 5.38, male M = 5.04). Furthermore, post-hoc pairwise comparisons showed that females exhibited significantly greater changes over time in their self-ratings of EE-F, with higher ratings at post-treatment (M = 5.80) than at pre-treatment (M = 5.38) time periods (p = .053). Lastly, a trend towards a significant three-way interaction (Gender x Time x Emotion) emerged for SE (F3, 135] = 2.246, p = .086), with pairwise comparisons showing a trend (p = .104) for women to receive higher ratings on angry monologues at pre-treatment in comparison to men.

Unexpectedly, there were significant differences between men and women with regard to facial mobility. Figure 6 displays the significant main effect of Gender for FM (F[1, 45] = 14.667, p = .000), such that female participants (M = 1.13) were rated as having a significantly greater degree of facial movement than were male participants (M = .81). A trend towards a significant Gender x Time interaction (F[1, 45] = 2.753, p = .104) was also present. Women were rated as significantly (p = .000) higher than men on FM at both pre-treatment (female M = 1.14, male M = .80) and post-treatment (female M = 1.12, male M = .83; p = .001).

Aim 4: Changes in Ratings of Emotion Over Time for SE and EE Across All Four Participant Groups.

Hypothesis 5. It was predicted that PD participants would exhibit a greater improvement over time for negative emotional monologues in comparison to positive emotional or neutral monologues for SE, but EE would remain the same. There were no significant interactions for Group x Emotion x Time for these dependent variables. However, there was a trend for the Group x Gender x Time x Emotion interaction for all groups for EE-A during the monologues ($F[6, 80] = 1.891$, $p = .092$). When post-hoc comparisons were conducted, the only finding present was a trend ($p = .080$) for higher self-ratings of emotional accuracy for the men in the LSVT group following the anger monologues at post-treatment ($M = 5.33$) compared to pre-treatment ($M = 5.11$). This interaction is displayed in Figure 7.

Aim 5: Comparing the Interaction Effects of EE with FM and SE as a Possible Mechanism of LSVT.

Hypotheses 6 and 7. It was proposed that if LSVT were affecting emotion systems, then EE would have the greatest change from pre- to post-treatment, SE would have a moderate change, and FM would not change. On the other hand, if LSVT were primarily targeting motor rather than emotion systems, then FM would have the greatest change over time, SE would have a moderate change, and EE would not change. We proposed to evaluate this by examining the strength of the F-values for FM, SE, and EE by visual inspection. The F-values for the Group x Time interaction were chosen, as this interaction examines changes in ratings between the four participant groups from pre- to post-treatment time periods. The F-values for each of the dependent variables for this interaction were as follows: EE-F = 2.327, EE-A = 1.480, EE-I = 6.530, SE = .220, and FM = 2.470. The largest F-value corresponded to EE-I (overall intensity of emotional experience following the monologues), which supports the hypothesis that LSVT may be affecting emotion systems directly. However, the F-values for FM and SE did not follow the hypothesized pattern. SE had the lowest F-value instead of the moderate value hypothesized, and FM had a moderate F-value rather than the lowest value. The values for the other two emotional experience variables (EE-A and EE-F) were both in the moderate range, which also does not clearly support the hypotheses. Thus, neither hypothesis 7 or 8 was fully supported.

Aim 6: The Association of FM with EE and SE with EE.

Hypotheses 8 and 9. It was proposed that if PD included a true emotional processing deficit (over and above the motor deficit that affects emotional expression), then there would be a greater positive correlation between SE and EE than between FM and EE. On the other hand, if PD does not include an emotional processing deficit (and emotional processing deficits are a direct result of motor signs), then there would be a moderate positive correlation between SE and EE and no correlation between FM and EE. Because EE was not normally distributed,

Spearman's rank-order correlations were conducted for all emotions and all PD participants at pre-treatment. Thus, 36 correlations were conducted each for the FM-EE comparison (FM Emotions [4] x EE Emotions [3] x EE variables [3]) and the SE-EE (SE Emotions [4] x EE Emotions [3] x EE variables [3]) comparison. Spearman rho correlation values for the FM-EE analysis are in Table 7, and the values for the SE-EE analysis are displayed in Table 8. The FM and EE comparison resulted in five significant correlations (all in the positive direction), whereas the SE and EE comparison resulted in no significant correlations. These findings do not clearly support either hypothesis. Although there were more significant correlations present than would be expected by chance for the FM-EE comparison, the correlations do not follow the hypothesized pattern. Thus, we did not find strong evidence pointing to the emotional processing deficits in PD seen in this study as a purely emotional processing deficit or a purely motoric deficit.

Discussion

The aim of this project was to investigate the effects of a voice treatment (LSVT) on facial expression, interpersonal interactions, and emotional experience in individuals with Parkinson's disease. Overall, LSVT did influence facial mobility and certain aspects of emotional experience; however, these findings were gender- and emotion-specific. LSVT appeared to have minimal effect on social engagement for the poser participants.

Emotional Expression and Experience Prior to Treatment (Aim 1)

First, baseline ratings (i.e., pre-treatment time period) were examined to determine whether PD and HC participants differed with respect to FM, SE, or EE. It was hypothesized that PD and HC participants would be similar for EE, but that PD participants would have lower ratings for FM and SE than HC participants. Results provided partial support for the hypothesis. PD participants did, indeed, have less facial mobility than the HC participants, but there was no difference for social engagement. PD participants also reported less emotional experience (across all three variables) than HC participants, but only for the Happy monologues; there was no group difference for the Sad and Angry monologues. In other words, PD participants were feeling less strongly for positive emotions than the HC participants. These findings for EE are consistent with the PD literature describing individuals with PD as expressing and experiencing fewer positive emotions than healthy controls (e.g., Brozgold et al., 1998; Pentland et al., 1987; Pitcairn et al., 1990).

Effects of Time and Treatment (Aim 2)

It was hypothesized that the greatest change in FM and SE ratings over time would occur for the LSVT group. The ARTIC participants would have the next highest change in FM and SE ratings, whereas the Untreated and HC groups would not be significantly different from pre- to post-treatment. Ratings for EE were not expected to vary across groups, as it was hypothesized that individuals with PD would have intact emotional experience.

The hypothesis was partially supported for FM, as ratings for the LSVT participants improved over time. On the other hand, FM ratings for the ARTIC and HC groups did not change from pre- to post- treatment. FM ratings for participants in the Untreated group decreased over time. It is possible that without any therapeutic contact from health professionals, the PD patients in the Untreated group deteriorated over time, whereas the patients in the ARTIC group remained stable because they benefitted from regular interactions with health professionals. The hypothesis for SE was not supported by the results as SE ratings did not change from pre- to post-treatment for any participant group.

Contrary to expectations, EE-F and EE-I changed over time for the LSVT and HC groups. Self-ratings of immediate emotional feelings (EE-F) following the monologue increased over time for women in the LSVT group, but not for men. The gender difference for EE-F (where women experienced more intense feelings than did men) could be due to women being "more emotional" than men (e.g., Brody & Hall, 1993).

Overall emotional intensity (EE-I) also increased for LSVT participants from pre- to post-treatment, whereas ratings for the HC participants decreased across time periods. The HC group may have experienced less emotional intensity over time due to habituation or practice effects. Even though the pre- and post-treatment testing times were one month apart, HC participants may have experienced less emotion at the second testing time because they had become acclimated to the task and, therefore, no longer experiencing emotion as intensely as during the first testing session. One possible explanation for the LSVT group result can be found in the James-Lange theory of emotion and the closely related facial feedback hypothesis. The facial feedback hypothesis (e.g., Buck, 1980) postulates that emotional feelings are directly linked to facial emotional expression. The James-Lange theory of emotion describes the relationship between physiology and the subjective experience of emotion more broadly: feelings of emotion result from changes in the autonomic system (e.g., muscle tension, sweating, and heart rate). In other words, emotions are experienced because of physical changes in the body, including facial muscle movement. In the current study, participants may have been experiencing more intense emotional feelings because their facial muscles were moving more. Recent studies have demonstrated that emotional experience can be modulated by facial expression (Davis, Senghas, & Ochsner, 2009). In other words, a person who smiles will subsequently endorse and experience greater feelings of happiness, and a person who frowns is more likely to report feelings of sadness afterwards. Davis and colleagues demonstrated that not only can feelings of emotion increase with greater facial expressivity, but emotional experience can *decrease* when facial expressions are *inhibited*. Perhaps the LSVT participants of this study experienced a similar phenomenon, given that they had higher ratings of facial movement.

Effects of Gender on Social Engagement and Emotional Experience (Aim 3)

Given the documented presence of differences across gender for emotional processing, we expected both social engagement and emotional experience to be higher in women than in men. This was not supported for SE. However, women had higher ratings than men overall for immediate emotional feelings (EE-F; see Figure 3). There were no significant differences between men and women for emotional accuracy (EE-A) or overall intensity (EE-I).

Unexpectedly, there were significant differences between men and women for FM ratings. Overall, women were rated higher in facial mobility than men.

The pattern of results in terms of gender continues to be in line with the large body of literature on gender differences in emotion, with women outperforming men on measures of emotional expression and perception (for reviews, see Borod & Madigan, 2000; Brody & Hall, 1993). What is unique about the current data set is that women are displaying increased facial movement compared to men. Facial mobility (FM) was designed to measure *non-emotional* facial movement and raters were specifically instructed to not incorporate emotional facial movement into their ratings. Potentially, raters may be biased toward perceiving women as more facially expressive as males. There is some evidence suggesting that women are expected to be more expressive in their social interactions than men (Briton & Hall 1995). Thus, women may simply be more facially expressive than males, due to social norms and expectations.

Effects of Time on Emotional Valence (Aim 4)

The negative emotional monologues (i.e., angry and sad) by PD participants were predicted to exhibit the most substantial improvement from pre- to post-treatment compared to the positive (i.e., happy) and neutral monologues for the SE and EE variables. There were no significant findings for SE, EE-F, or EE-I. However, male participants of the LSVT group had higher self-ratings of emotional accuracy (EE-A) following their angry monologues at post-treatment that at pre-treatment. It is important to note that it is possible that this result occurred by chance due to the large number of statistical analyses that were conducted.

Potential Mechanism of LSVT (Aim 5)

In this project, we sought to determine whether successful treatment via LSVT occurs by affecting emotion systems or motor systems. It was proposed that a primarily motoric mechanism would be indicated by the largest change over time occurring in FM, whereas SE would have moderate changes over time, and EE would not change over time. On the other hand, if EE presented with the greatest change over time, SE with moderate change, and FM with no change over time, then LSVT may more likely be affecting emotion systems. An examination of F-values for these dependent variables did not provide results that clearly fit with either hypothesis. Overall intensity of emotional experience (EE-I) presented with the relatively largest change over time, which provides support for the possibility that LSVT is directly affecting emotion systems. However, social engagement (SE) presented with the least degree of change, and FM presented with moderate changes from pre- to post-treatment, which does not fit either hypothesis. Although the hypothesis for LSVT tapping into emotion systems for its mechanism of change has some support, neither hypothesis is unequivocally supported. Further research would benefit from pursuing other statistical techniques (e.g., examining effect sizes) with regard to this research question.

Are the Emotional Processing Deficits of PD a Side Effect of Motor Dysfunction? (Aim 6)

For the final aim, we sought to provide evidence for whether the emotional processing deficits that are present in PD are a direct result of a true emotion dysfunction, or whether they are a consequence of the motor signs of PD (i.e., reduced facial expressivity secondary to masked facies and/or bradykinesia). Correlations were conducted to determine the relationship of EE, separately, to FM and to SE. It was proposed that a true emotion dysfunction would be indicated if SE and EE had a greater positive correlation than did FM and EE. Alternatively, a purely motor deficit would be suggested if EE and SE were moderately correlated while EE and FM were not correlated. Analyses found more positive correlations between FM and EE than between SE and EE, which does not clearly support either hypothesis proposed. Based on our results, it cannot be determined at this time whether the emotion deficits in PD are due to a true emotion processing deficit or are a result of motor dysfunction. Further analyses with this particular dataset could utilize disease stage in order to examine motor signs more thoroughly. Future research in this area would benefit from a more direct consideration of motor signs, for example, by obtaining scores on the Unified Parkinson's Disease Rating Scale (UPDRS; Fahn et al., 1987) for PD participants.

Evaluating Groups Differences for Depression

Because a significant difference was found between the LSVT participants and the other three participant groups on a screening measure of depression (BDI-II), we conducted further analyses to determine whether analysis of covariance (ANCOVA) might be useful in understanding the influence of depression on the five dependent variables that were studied here. For example, it is possible that the significant difference between the LSVT and HC groups on depressive symptoms could be masking any differences that might have been present among the participant groups on SE. There is a considerable literature describing the co-morbidity of depression with PD, with prevalence rates at approximately 45% (e.g., Lemke at al., 2004; Zgaljardic et al., 2003). Also, depression is known to affect facial emotional expression for both positive and negative emotions (Jaeger, Borod, & Peselow, 1986). In order to determine whether an ANCOVA would be appropriate statistically, poser participant BDI scores for all PD and HC participants were correlated with of the five dependent variables (FM, SE, EE-F, EE-I, and EE-A). Spearman's rank-order correlations were conducted for SE and the three EE variables (due to non-normal distributions), whereas Pearson's product-moment correlations were conducted for FM (due to normal distributions after natural log transformation). There were no significant correlations present between BDI and the five dependent variables. Consequently, ANOVAs were not conducted. Since BDI scores were not significantly correlated with the five dependent variables, it is unlikely that depression is affecting these measures.

Limitations & Future Directions

One significant limitation of our dataset is that the Cohort 2 rater agreement (i.e., inter-rater reliability) was relatively low for the experimental data for SE (ICC = .696), which may indicate unreliable data. In other words, low inter-rater reliability usually indicates that the raters did not clearly understand how to make their ratings or were using differing strategies to make their ratings. The low inter-rater reliability on SE for Cohort 2 was unexpected, as this group of raters had high inter-rater agreement during training. Although the other two cohorts of this project had high inter-rater reliability (Cohort 1 ICC = .841 and Cohort 3 ICC = .815), lower inter-rater reliability for one of the three rater cohorts may explain the lack of significant findings for SE on this project. SE could be re-examined by removing Cohort 2, and all the SE data could be re-analyzed with only Cohorts 1 and 3. It is also important to consider why there were no significant findings for SE. For example, perhaps our definition of social engagement was too broad and not specific enough. In future research, it might be worthwhile to re-conceptualize SE into multiple components, as was done with emotional experience. Another limitation is that our PD poser participants had relatively mild forms of the disease. The average Hoehn and Yahr stage for the PD participant groups was Stage 2 (out of five possible stages). Patients at this stage have just begun to have bilateral motor signs and are considered to have “minimal disability.” Thus, perhaps the PD patients in this study did not demonstrate significant changes over time in response to treatment (particularly with regard to social engagement) because their deficits were not severe enough. It would be ideal to include patients with more severe deficits in the study sample. In addition, future research in this area would benefit from controlling for the severity and number of motor signs in the PD participant groups. Unfortunately, there may be some disadvantages to having participants with more severe deficits. It was informally noted by the experimenters that the video clips creating the most disagreement among the raters during training were often for posers who had the most noticeable motor signs (e.g., head movement or tremor). It is possible that including patients at more severe stages of the disease could actually create lower inter-rater reliability in the experimental data.

The final limitation of the project is related to the age of the raters and posers. It is possible that utilizing raters who were younger than our posers created a cohort effect. For example, Neiss and colleagues recently demonstrated that younger participants rated positive pictures as less arousing compared to older participants (Neiss, Leigland, Carlson, & Janowsy, 2009). Younger adults could potentially rate older posers differently than posers who are closer to them in age. However, previous research in our lab utilizing similar procedures (e.g., Borod et al., 2004) has not found any cohort age effects during ratings of facial emotional expression. Ideally, we would have recruited raters who are closer in age to the poser participants. However, the length of time required from the raters (several hours per week for 2-3 months) for the rating sessions led us to believe that recruiting students from the Queens College community (where the study took place) was more feasible, was more convenient for the raters, and made rater retention more likely. In the future, it would be interesting to recruit older raters and to ask them to rate the current data. It is plausible that older adult raters may provide different ratings than younger raters on variables that are related to social interactions, such as social engagement. Of note, such variables were not examined in the study by Borod et al. (2004).

Conclusions

Our study was able to provide some support for current theories with regard to emotional deficits in Parkinson's disease and gender differences in emotional processing. At baseline, PD participants exhibited decreased facial mobility and fewer feelings of positive emotion (i.e., happiness) compared to their healthy control counterparts. We also found women to be more facially expressive and to experience more emotion than men. Unfortunately, we were not able to provide additional information about social engagement, the mechanisms of LSVT, or the underlying cause of emotional deficits in PD. However, we were able to demonstrate that LSVT improves facial movement and facilitates more intense emotional experience. This finding has clinical implications for PD patients. LSVT appears to be enhancing the subjective experience of emotion in individuals with PD. This raises the possibility of utilizing LSVT in a psychotherapeutic capacity (e.g., to aid in alleviating mood disorder symptoms), in addition to its original intended use as a voice treatment. The potential psychotherapeutic properties of LSVT also suggest that it could potentially be useful in other neuropsychiatric populations, such as schizophrenia or major depression. Finally, an important contribution of our study involves emotional experience. Previous research has conceptualized experience as a unitary variable, and results have thus far been mixed as to whether experience is impaired in PD. The current study, in its attempts to evaluate experience more thoroughly, demonstrated changes in experience in PD participants following LSVT, thereby providing potential avenues of research for the future.

Table 1
Demographic characteristics of poser participants

	Parkinson's Disease Groups (n = 39)			Healthy Control Group (n = 14)
	LSVT (n = 12)	**ARTIC (n = 14)**	**Untreated (n = 13)**	**HC (n = 14)**
Age (years)	66.7 (6.6)	68.4 (10.2)	65.5 (8.8)	66.5 (8.3)
Gender (% men)	75%	64%	69%	64%
Education (yrs.)	15.9 (2.6)	16.6 (4.3)	16.4 (2.7)	17.7 (2.8)
Stage of PD	2.2 (.5)	2.3 (.7)	2.0 (.5)	--
Years since dx.	6.0 (8.3)	3.8 (3.2)	5.5 (4.5)	--
MMSE score	28.8 (1.6)	28.5 (1.2)	29.0 (.8)	29.4 (.8)
BDI-II score	10.5 (6.5)	7.2 (5.0)	5.9 (4.3)	3.2 (3.7)

Note: Values are listed as mean (standard deviation) and percentages.

Table 2
Demographic characteristics of rater participants

	Gender % men	**Age** Mean (SD)	**Education** Mean (SD)
Cohort 1 (n = 6)	50%	25.3 (6.15)	15.5 (.55)
Cohort 2 (n = 6)	50%	23.8 (2.03)	16.3 (.75)
Cohort 3 (n = 6)	50%	23.8 (6.59)	14.5 (1.50)
All Raters (n = 18)	50%	24.6 (5.38)	15.5 (1.26)

Table 3
Intra-class correlations for rater training sessions: Conferencing and inter-rater reliability

	Training Variable	
	FM	**SE**
Cohort 1		
Conferencing	.962	.984
Inter-rater reliability	.921	.926
Cohort 2		
Conferencing	.932	.948
Inter-rater reliability	.860	.863
Cohort 3		
Conferencing	.948	.901
Inter-rater reliability	.908	.946

Table 4
Intra-class correlations for experimental rating sessions

	Experimental Variable	
	FM	**SE**
Cohort 1	.812	.841
Cohort 2	.896	.696
Cohort 3	.867	.815

Table 5
Shapiro-Wilk tests of normality for all five dependent variables: Raw data

Facial Mobility

	Pre-Treatment				Post-Treatment			
	Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
ARTIC	.925	.921	.762**	.918	.978	.909	.903	.951
LSVT	.866*	.834**	.823**	.832**	.844**	.910	.885*	.842**
Untx.	.904	.939	.936	.969	.986	.931	.932	.967
HC	.891*	.914	.907	.950	.965	.863**	.955	.963

Note: Values in table are the Shapiro-Wilk statistic value.
*.05 ≤ $p \leq .10$
** $p \leq .05$

Social Engagement

	Pre-Treatment				Post-Treatment			
	Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
ARTIC	.954	.919	.957	.931	.949	.937	.898	.974
LSVT	.898	.950	.934	.940	.945	.931	.944	.903
Untx.	.963	.950	.964	.961	.963	.966	.927	.962
HC	.967	.898	.962	.941	.965	.919	.957	.953

Note: Values in table are the Shapiro-Wilk statistic value.
*.05 ≤ $p \leq .10$
** $p \leq .05$

Table 5 (continued)
Shapiro-Wilk tests of normality for all five dependent variables: Raw data

Emotional Experience – Immediate Feelings

	Pre-Treatment				Post-Treatment			
	Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
ARTIC	.897	.917	.879*	--	.851**	.912	.890*	--
LSVT	.905	.781**	.857**	--	.920	.807**	.781**	--
Untx.	.884	.863*	.799**	--	.887	.486**	.909	--
HC	.907	.807**	.780**	--	.920	.688**	.845**	--

Note: Values in table are the Shapiro-Wilk statistic value.
*.05 ≤ p ≤ .10
** p ≤ .05

Emotional Experience – Overall Intensity

	Pre-Treatment				Post-Treatment			
	Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
ARTIC	.954	.900	.801**	--	.915	.941	.740**	--
LSVT	.920	.920	.830**	--	.848**	.824**	.831**	--
Untx.	.831**	.889	.803**	--	.863*	.906	.878*	--
HC	.900	.777**	.773**	--	.846**	.862**	.830**	--

Note: Values in table are the Shapiro-Wilk statistic value.
*.05 ≤ p ≤ .10
** p ≤ .05

Table 5 (continued)
Shapiro-Wilk tests of normality for all five dependent variables: Raw data

Emotional Experience -- Accuracy

	Pre-Treatment				**Post-Treatment**			
	Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
ARTIC	.899	.839**	.862**	--	.901	.812*	.893	--
LSVT	.798**	.906	.878*	--	.917	.935	.882*	--
Untx.	.774**	.855**	.916	--	.904	.795**	.871*	--
HC	.920	.811**	.823**	--	.892	.905	.934	--

Note: Values in table are the Shapiro-Wilk statistic value.
*$.05 \leq p \leq .10$
** $p \leq .05$

Table 6
Shapiro-Wilk test of normality for all five dependent variables: Natural log transformed data

Facial Mobility

	Pre-Treatment				**Post-Treatment**			
	Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
ARTIC	.908	.891*	.868*	.920	.960	.954	.959	.959
LSVT	.939	.902	.872*	.878*	.875*	.953	.951	.913
Untx.	.921	.950	.948	.973	.985	.953	.945	.981
HC	.924	.952	.941	.956	.947	.935	.964	.987

Note: Values in table are the Shapiro-Wilk statistic value.
*$.05 \leq p \leq .10$
** $p \leq .05$

Social Engagement

	Pre-Treatment				**Post-Treatment**			
	Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
ARTIC	.963	.877*	.963	.916	.910	.886*	.918	.962
LSVT	.964	.958	.958	.955	.952	.953	.956	.884*
Untx.	.895	.942	.935	.937	.952	.901	.919	.959
HC	.957	.948	.967	.966	.964	.978	.958	.995

Note: Values in table are the Shapiro-Wilk statistic value.
*$.05 \leq p \leq .10$
** $p \leq .05$

Table 6 (continued)
Shapiro-Wilk test of normality for all five dependent variables: Natural log transformed data

Emotional Experience – Immediate Feelings

	Pre-Treatment				Post-Treatment			
	Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
ARTIC	.796**	.726**	.793**	--	.817**	.906	.736**	--
LSVT	.875*	.877*	.826**	--	.866*	.807**	.781**	--
Untx.	.709**	.848**	.794**	--	.695**	.486**	.888	--
HC	.765**	.809**	.694**	--	.815**	.655**	.834**	--

Note: Values in table are the Shapiro-Wilk statistic value.
*$.05 \leq p \leq .10$
** $p \leq .05$

Emotional Experience – Overall Intensity

	Pre-Treatment				Post-Treatment			
	Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
ARTIC	.862**	.760**	.862**	--	.774**	.876*	.582**	--
LSVT	.890	.884*	.890	--	.781**	.823**	.808**	--
Untx.	.826**	.890	.826**	--	.853**	.896	.860*	--
HC	.892	.772**	.892	--	.836**	.870*	.816**	--

Note: Values in table are the Shapiro-Wilk statistic value.
*$.05 \leq p \leq .10$
** $p \leq .05$

Table 6 (continued)
Shapiro-Wilk test of normality for all five dependent variables: Natural log transformed data

Emotional Experience – Accuracy

	Pre-Treatment				**Post-Treatment**			
	Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
ARTIC	.892*	.726**	.829**	--	.866**	.803**	.859**	--
LSVT	.730**	.877*	.831**	--	.908	.917	.810**	--
Untx.	.772**	.848**	.889	--	.900	.817**	.866*	--
HC	.884*	.809**	.811**	--	.878*	.884*	.934	--

Note: Values in table are the Shapiro-Wilk statistic value.
*.05 ≤ p ≤ .10
** p ≤ .05

Table 7

Aim 6: Spearman's rho correlations between FM and EE for PD participants

			FM							
			Pre				**Post**			
			Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
EE-F	**Pre**	**Angry**	.197	.165	.169	.192	.036	.011	.077	.108
		Happy	.099	.061	.021	-.012	.039	-.060	.002	.062
		Sad	.351**	.466**	.337**	.314*	.325**	.292*	.318*	.364**
	Post	**Angry**	.462**	.417*	.438**	.476**	.341**	.323**	.401**	.362**
		Happy	.332**	.226	.103	.196	.228	.280*	.188	.224
		Sad	.175	.204	.155	.194	.036	.103	.186	.220
EE-I	**Pre**	**Angry**	.164	.182	.104	.201	036	.011	.077	.108
		Happy	-.029	-.001	-.104	-.047	-.075	-.106	-.109	.538**
		Sad	.341**	.402**	.261	.269	.267	.224	.290*	.322*
	Post	**Angry**	.158	.295*	.162	.331**	.275	.239	.281*	.394**
		Happy	.226	.188	.104	.226	.178	.182	.231	.176
		Sad	.090	.087	.077	.143	-.024	-.012	.124	.136
EE-A	**Pre**	**Angry**	-.006	.079	-.049	.137	.078	.071	.024	.036
		Happy	-.165	.049	-.072	.026	.097	-.031	.130	.161
		Sad	.143	.273*	.138	.158	.225	.210	.267	.254
	Post	**Angry**	-.008	.004	-.127	.079	.070	.041	.059	.072
		Happy	.088	.201	.033	.225	.274*	.206	.238	.238
		Sad	.184	.227	.273*	.290*	.262	.245	.372**	.293*

Note: Values in table are the Spearman's rho statistic value.
*$.05 \leq p \leq .10$
** $p \leq .05$

Table 8

Aim 6: Spearman's rho correlations between SE and EE for PD participants

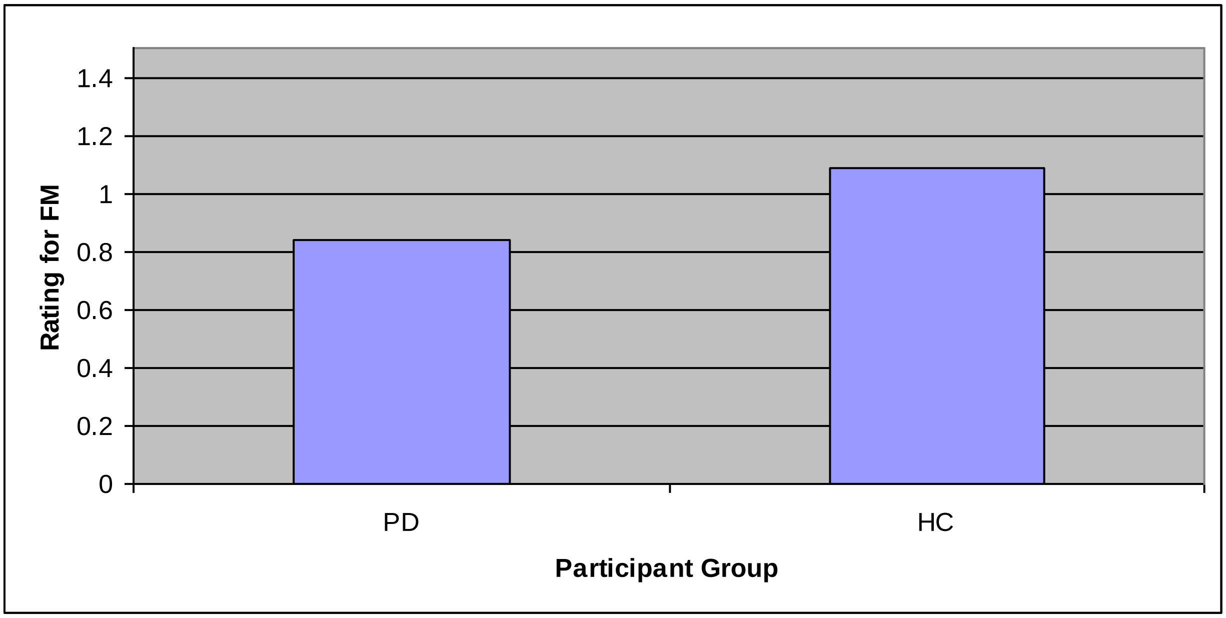
			SE							
			Pre				**Post**			
			Angry	**Happy**	**Sad**	**Neutral**	**Angry**	**Happy**	**Sad**	**Neutral**
EE-F	**Pre**	**Angry**	.187	.173	.055	.126	.116	.120	-.047	.104
		Happy	.179	.166	.270	.174	.132	.175	.148	.235
		Sad	.317*	.301	.202	.286*	.290*	.356**	-.024	.243
	Post	**Angry**	.183	.305*	.115	.153	.152	.115	.025	.096
		Happy	.228	.079	.050	.147	.016	.066	-.052	.110
		Sad	.112	.088	.035	.151	-.129	-.121	-.124	.032
EE-I	**Pre**	**Angry**	.191	.209	.013	.147	.123	.214	-.008	.197
		Happy	.090	.152	-.036	.093	.005	.113	-.088	.113
		Sad	.261	.305*	.172	.270	.251	.323**	-.097	.225
	Post	**Angry**	-.054	.116	-.162	.000	.089	.128	-.106	.117
		Happy	.136	.047	-.073	.059	.005	.076	-.048	-.009
		Sad	.080	.135	.082	.142	-.039	-.011	.032	.129
EE-A	**Pre**	**Angry**	.055	.286*	.034	.213	.241	.352**	.021	.281*
		Happy	-.094	.187	.124	.166	.140	.226	.071	.230
		Sad	-.004	.185	.086	.153	.208	.267	-.064	.046
	Post	**Angry**	-.113	.131	-.017	.060	.099	.152	-.096	.115
		Happy	.118	.207	.016	.161	.289*	.350**	.148	.197
		Sad	.005	.114	.143	.127	.041	.035	.074	.099

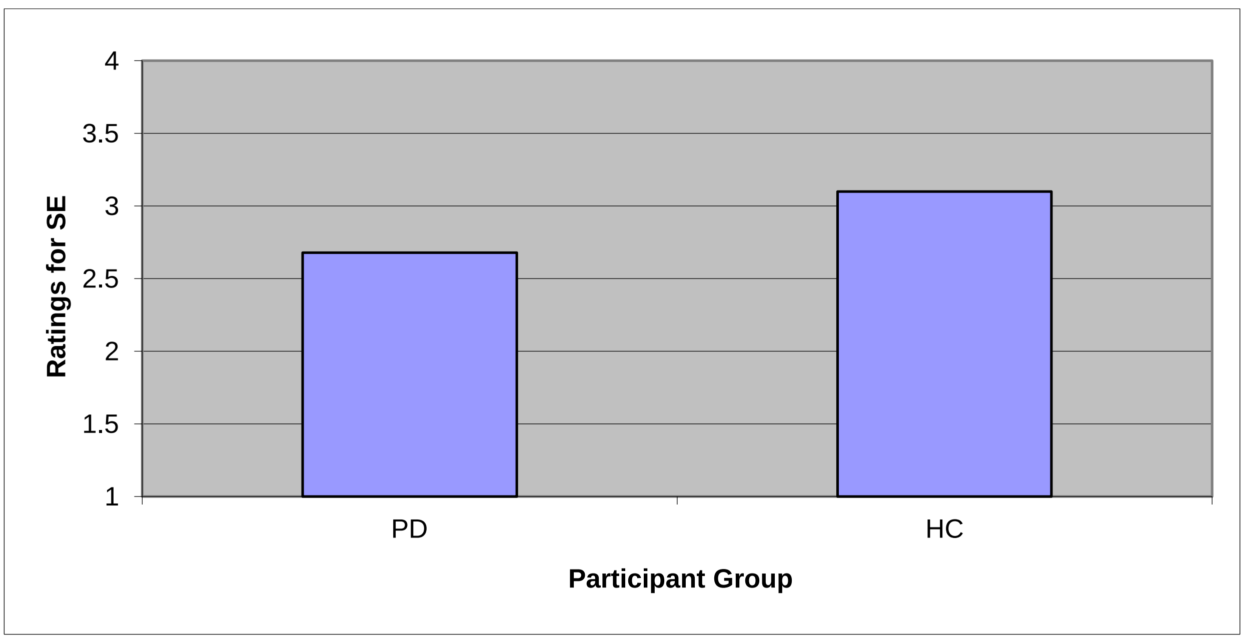
Note: Values in table are the Spearman's rho statistic value.

*$.05 \leq p \leq .10$

** $p \leq .05$

Figure 1
Aim 1: Ratings for facial mobility (FM) and social engagement (SE) at baseline for Parkinson's disease (PD) and healthy control (HC) participants

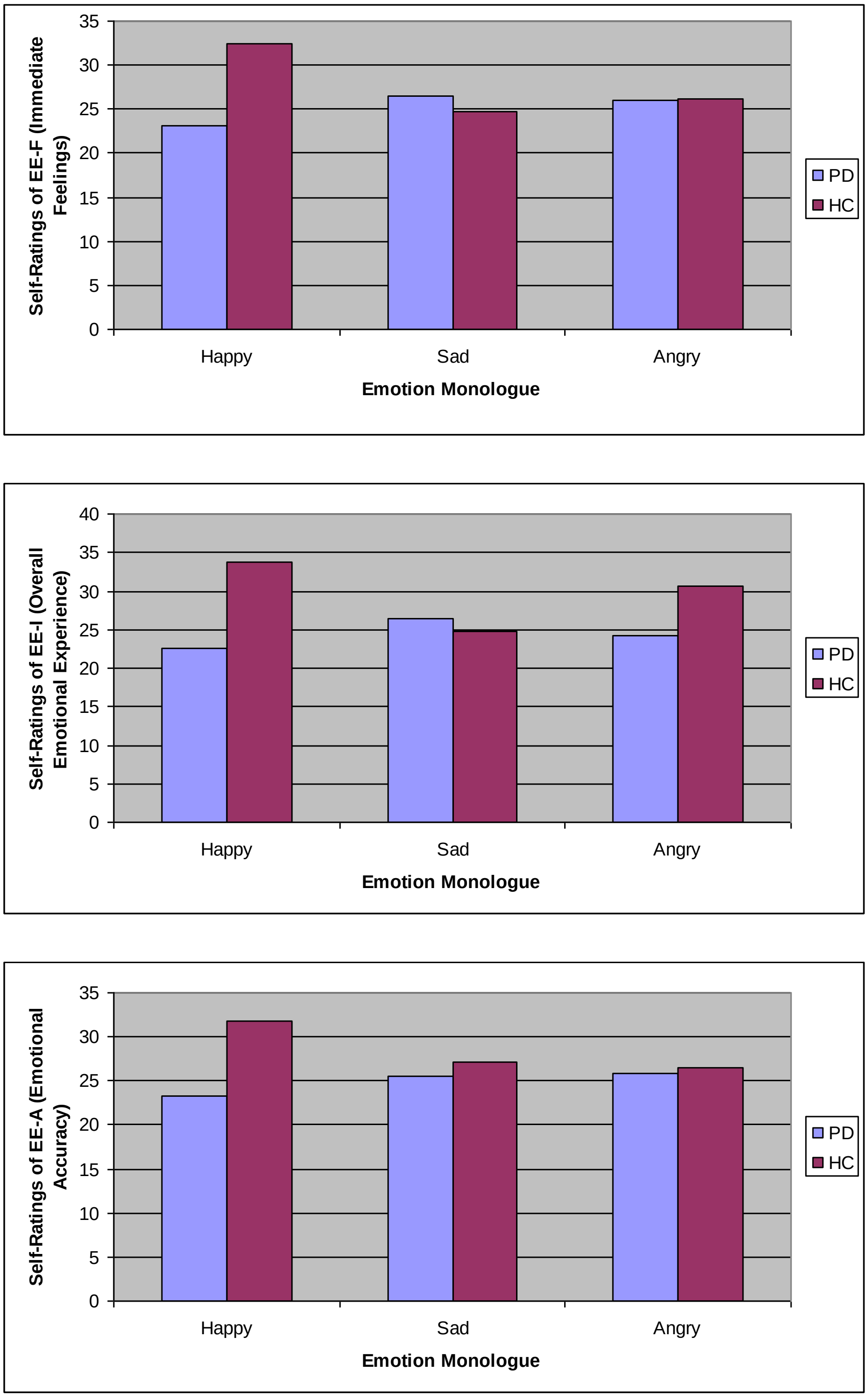




Note: The group differences between the PD and HC participants is significant for FM (p = .021).

Figure 2

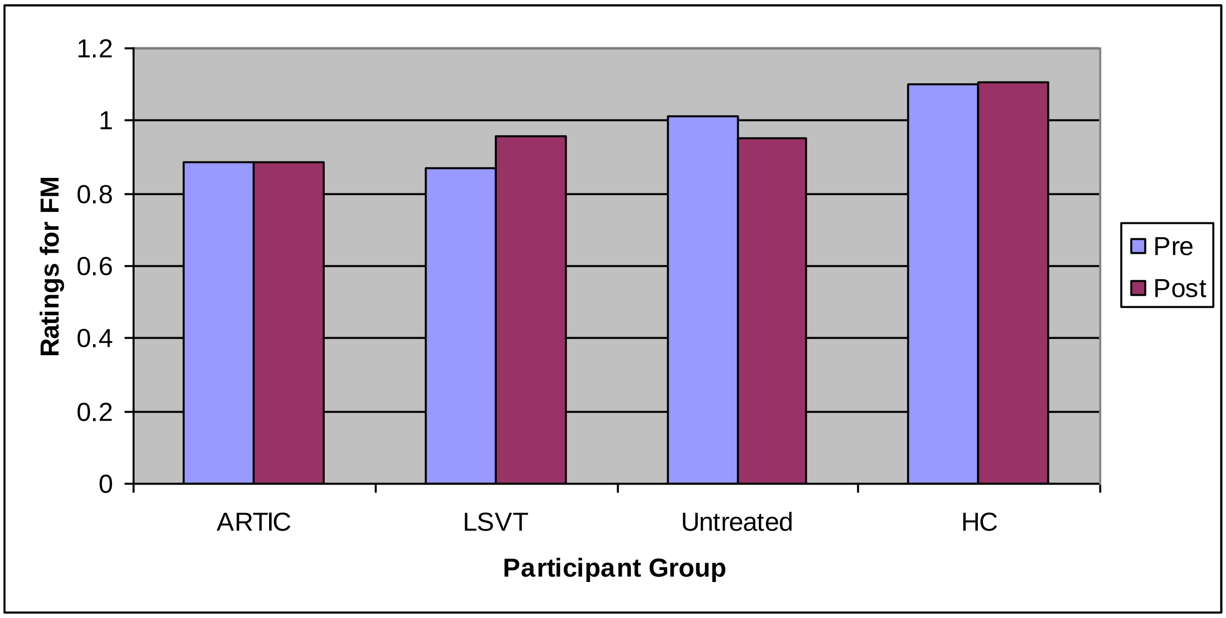
Aim 1: Ratings of immediate emotional experience (EE-F), overall emotional experience (EE-I), and accuracy (EE-A) at baseline for Parkinson's participants and healthy controls (HC)



Note: Values on the y-axis are mean ranks.

Figure 3

Aim 2: Group x Time interaction for facial mobility (FM)



Note: Values on the y-axis are natural log values.

Figure 4

Aim 2: Group x Gender x Time interaction for immediate emotional feelings (EE-F)

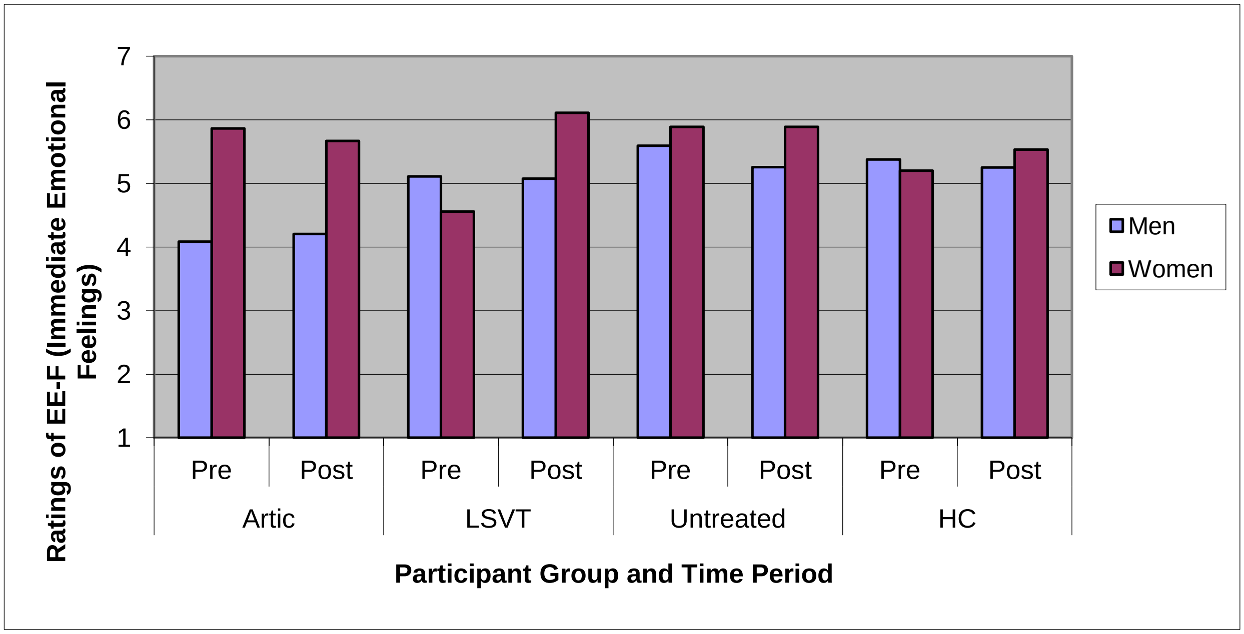
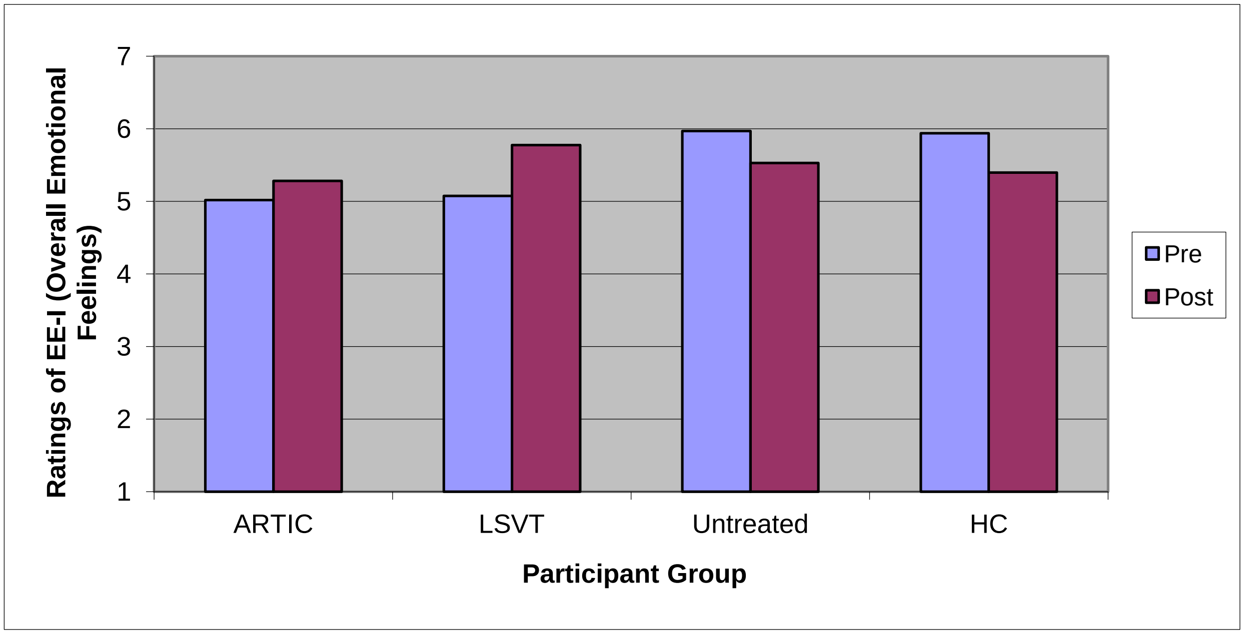


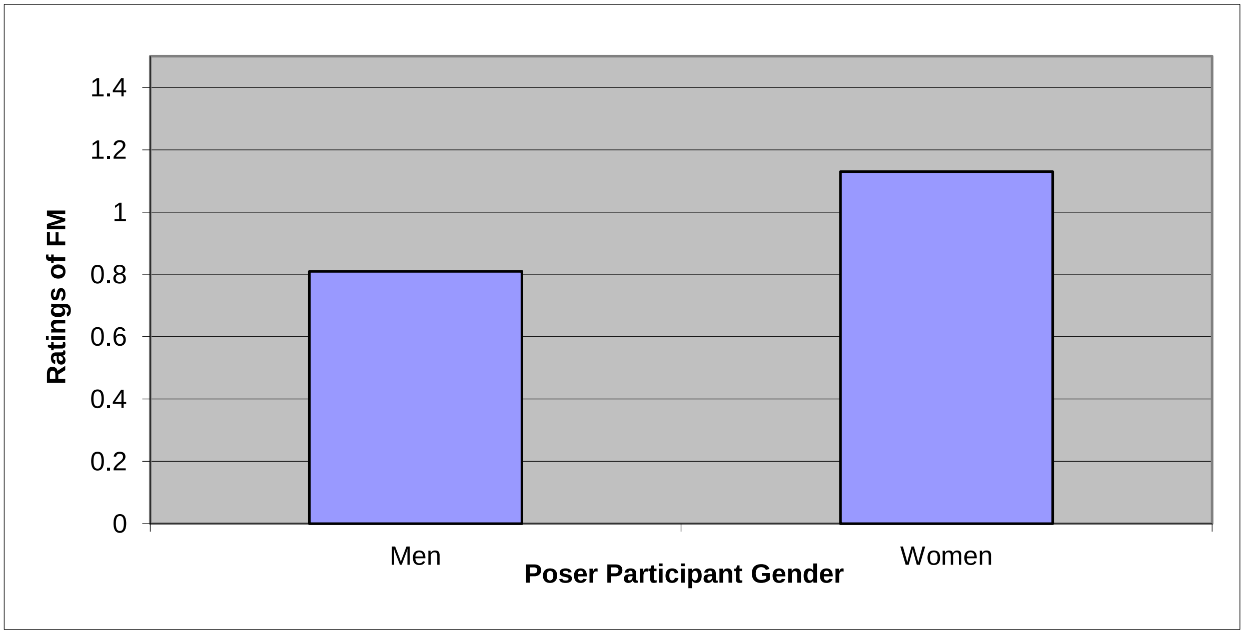
Figure 5
Aim 2: Group x Time interaction for overall emotional feelings (EE-I)

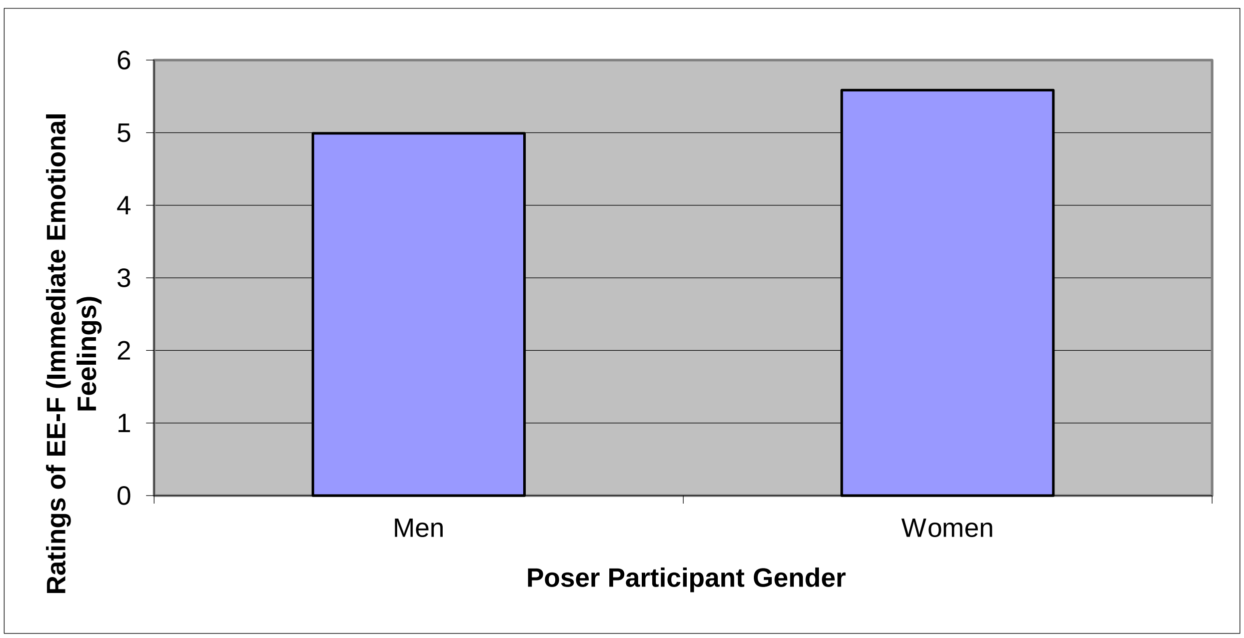


Note: The differences from pre- to post-treatment was significant for the LSVT (p = .005) and the HC (p = .016) groups only.

Figure 6

Aim 3: Gender effects for facial mobility (FM) and immediate emotional feelings (EE-F)

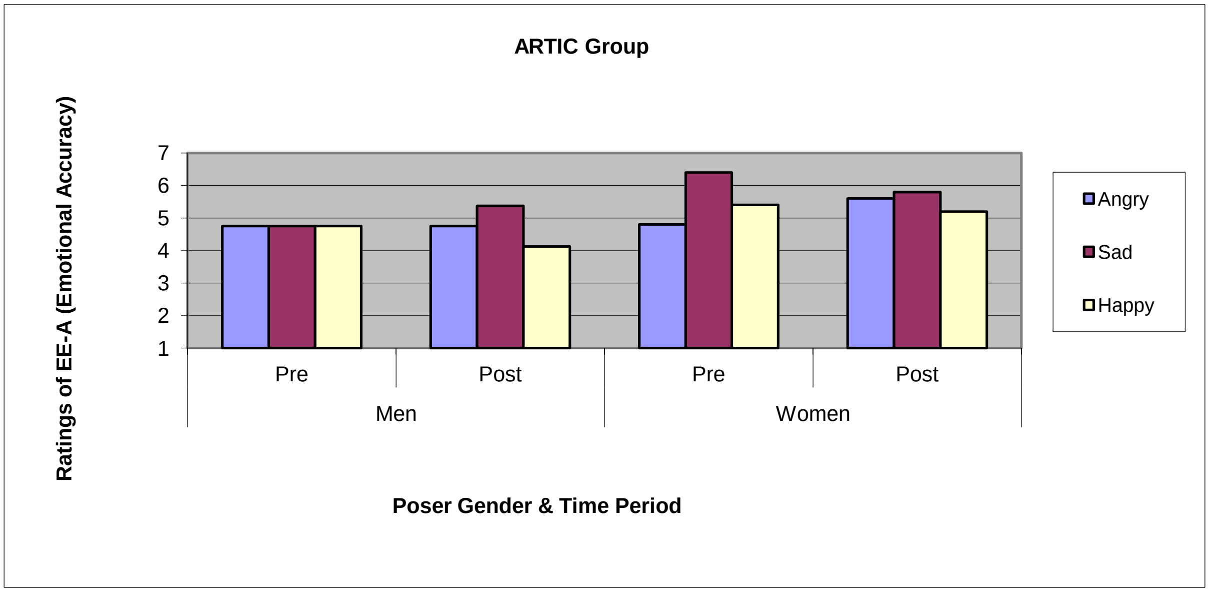




Note: Values on the y-axis for FM are natural log values.

Figure 7

Aim 4: Emotional valence effect via the Group x Gender x Time x Emotion interaction



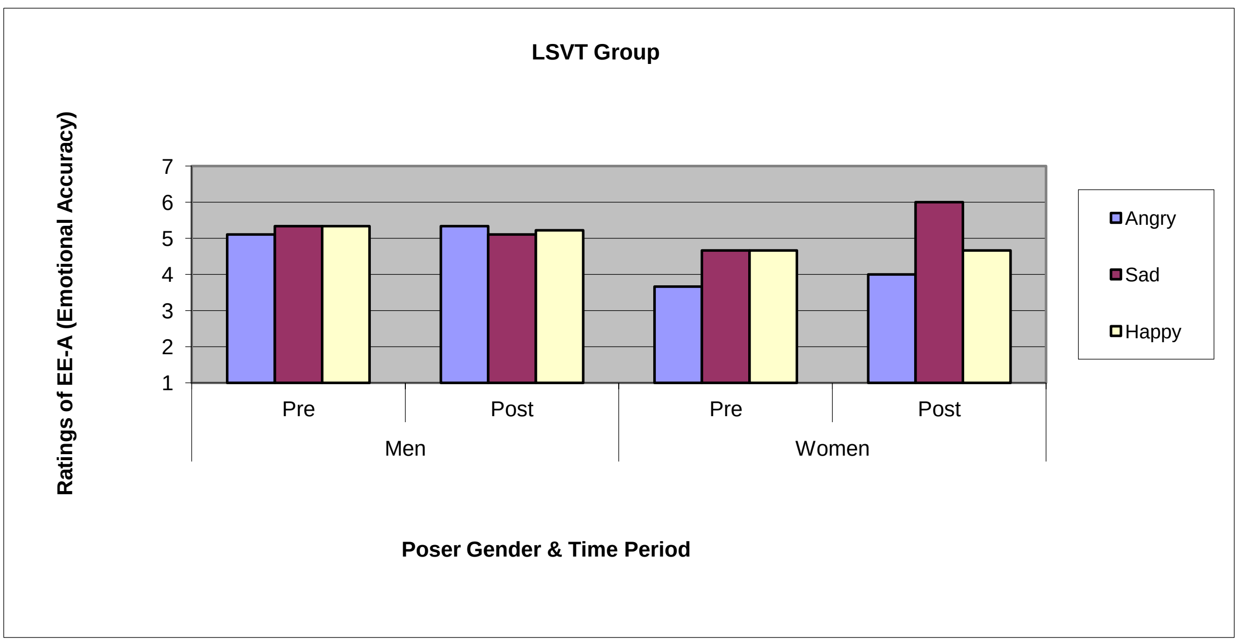
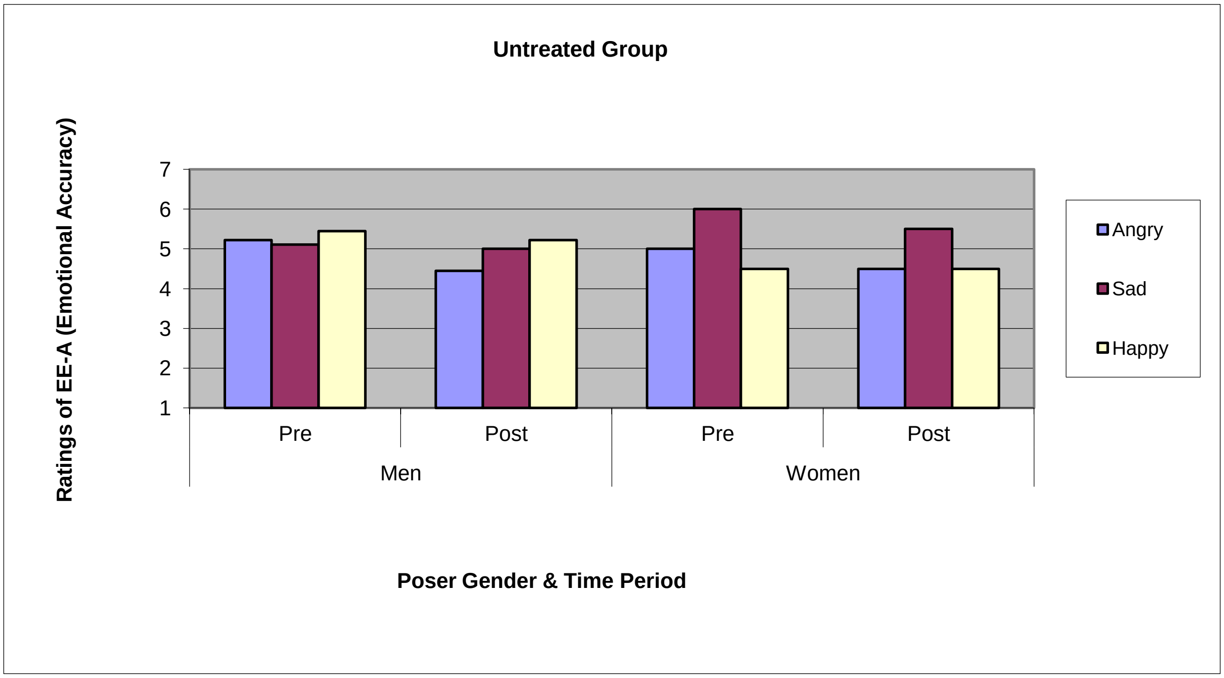
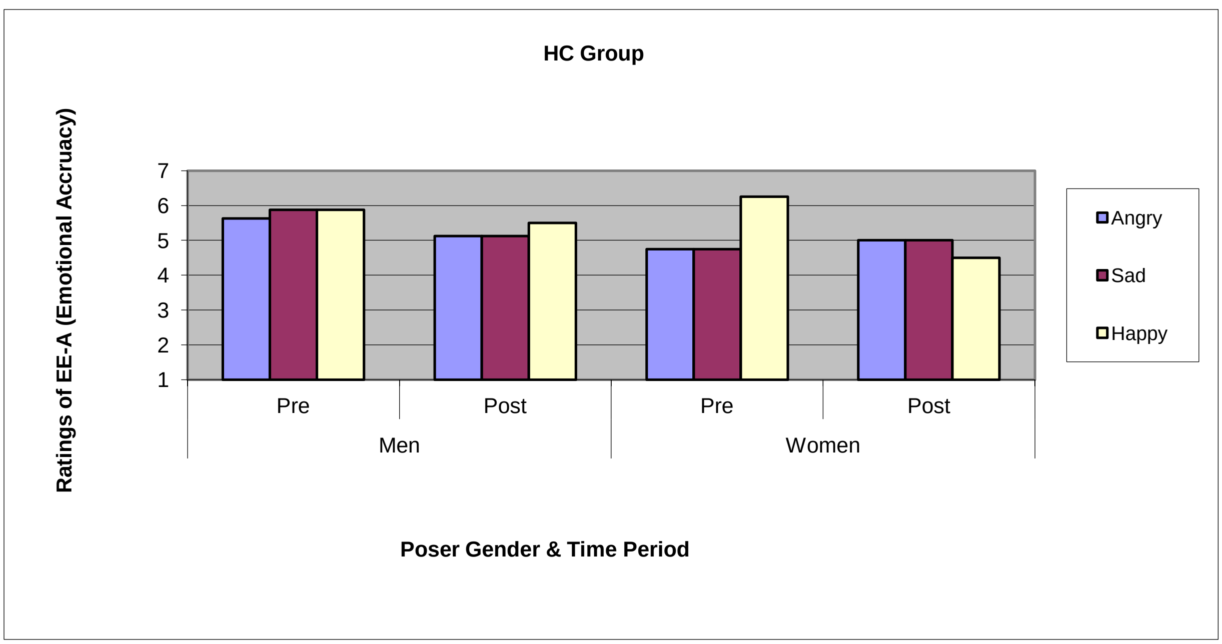


Figure 7 (continued)

Aim 4: Emotional valence effect via the Group x Gender x Time x Emotion interaction





Appendix

Excerpts of Training Materials for Facial Mobility and Social Engagement

Introduction and Definition Stage for Facial Mobility:

Researcher: *Today you will be asked to rate individuals' faces for their degree of facial mobility. We will go through some brief training in order for you to become familiar with the rating process. This process is likely to take about 1-2 hours. If you need to take a short break before then, please ask and I will stop at the next most natural break in our work.*

I'll begin the training phase of our session today by describing what I mean by facial mobility (hereafter: FM) and showing you the scale you will be using.

FM refers to how much movement the face produces. Mobility is defined here as the amount and extent of muscular movement of the face. Faces demonstrate different degrees of FM with varying degrees of muscular involvement. For example, most people would think that this face (Researcher uses own face to model an extremely mobile face) *conveys more FM than this face* (Researcher uses own face to model a minimally mobile face).

When rating FM, look for muscle movement around the forehead, eyebrows, eyes, nose, cheeks, mouth, and chin. Think about the following three things when examining the segments for mobility:

- *How dynamic the face is*
- *How frequently the face moves*
- *What is the range of movement*

There are a number of parts of the face that can demonstrate FM that you can look for when making these ratings. [Researcher should model and point to the different facial features as they are discussed.]

a. Forehead: wrinkled; vertical lines can be seen between the eyes
b. Eyebrows: raised, lowered & drawn together
c. Eyes (i.e. upper and lower eyelids): open or closed to varying degrees
d. Eyes (i.e. eye-balls): shifting of gaze
e. Nose (i.e. sides & bridge): wrinkled
f. Cheeks: flattened, stretched, and raised and rounded
g. Naso-labial fold: different degrees of depth.
h. Mouth: open or closed to varying degrees.
i. Corners of the mouth: drawn back and up
j. Jaw: lowered or moved from side to side
k. Lips: non-contracted or at rest, contracted or pressed.

All of these movements created by the facial muscles can vary to show different degrees of FM. These movements can be observed on both sides or one side of the face. While you will use your own first impression of how mobile the face looks, you can use some of these muscles and features as a way of helping you make your decision.

Try to disregard head movements and concentrate on the movements of facial muscles. Movements which appear to be random (such as excessive blinking, head shaking, and body tremor) should not be included in mobility ratings.

You should also not take into account the person's individual facial features. Since you are being asked to judge the extent of FM, you should not, for example, include a person's age wrinkles as an indication of mobility since those are their natural features. These natural features could occur even in a resting face.

Don't worry; there are no absolute right or wrong ratings. In the initial part of today's session, what we will do, as a group, is try to come to a consensus rating for many faces. In this way, you will learn from each other. The goal is to be able to reach agreement on the rating of as many faces as possible. This is how we will figure out what is "right". Later, you will be rating faces on your own.

ANY QUESTIONS?

The scale you will be using to rate the FM of the faces is a 7-point scale. For this scale, "1" is the lowest degree of mobility. "7" is the highest, and "4" represents the mid-point.

RESEARCHER DIRECTS RATERS TO SCALE.

Let's review each scale point, and what it means.

<u>Facial Mobility Scale:</u>

1 – "Minimally mobile." In other words, the face conveys almost no or minimal mobility.

2 – "Slightly mobile." The face conveys some, but only slight mobility.

3 – "Somewhat mobile." The face conveys more than a slight degree of mobility, but just less than the middle point on the scale.

4 – "Moderately mobile." This is the mid-point on the scale which indicates moderate mobility.

5 – "Quite mobile." The face conveys just more than moderate mobility.

6 – "Very mobile." The face is very mobile, but not quite at the top of the scale.

7 – "Extremely mobile." This is the highest degree of mobility that a face can convey.

Introduction and Definition Stage for Social Engagement:

Researcher: *Today you will be asked to rate individuals' faces for their degree of social engagement. We will go through some brief training in order for you to become familiar with the rating process. This process is likely to take about 1-2 hours. If you need to take a short break before then, please ask and I will stop at the next most natural break in our work.*

I'll begin the training phase of our session today by describing what I mean by social engagement (hereafter: SE) and showing you the scale you will be using.

SE in this study refers to how well one person draws another into an interaction. It is a measure of your desire to either involve yourself or not in conversation with the person (approach or withdraw from the interaction). The more you wish to be involved with this person, the more "engaging" the person appears to you.

While our individual reasons for being attracted to someone tend to be subjective, there are a number of behaviors, which may make a person more engaging than another during a conversation. Usually, the more "attentive" or "involved" someone appears, the more we wish to become involved with them. Such attentiveness can be communicated through facial expression and eye contact. Specific questions to keep in mind while making the SE rating include the following:

- *Does the speaker appear to be attentive and involved?*
- *Does the speaker appear to be connected?*
- *Does the speaker appear to be confident?*
- *Does the speaker appear to be someone a person with whom one would like to interact?*
- *Does the speaker maintain appropriate eye contact, without staring?*
- *How often does the speaker's gaze shift? Does it appear appropriate?*

While you will use your own first impression of how engaging a face looks, you can use some of the guidelines just mentioned as a way of helping you make your rating. In approaching this task, do not only consider your own perspective, but also consider how others might react to the speaker. Try to disregard head movements and concentrate on the movements of facial muscles. Movements which appear to be random (such as excessive blinking, head shaking, and body tremor) should not be included in SE ratings.

You should not take into account the person's individual facial features or respond to qualities that you find attractive or unattractive. Since you are being asked to judge the extent of SE, you should not, for example, be influenced by facial features, such as a mole or a scar, since those are their natural features.

Don't worry; there are no absolute right or wrong ratings. In the initial part of today's session, what we will do, as a group, is try to come to a consensus rating for many faces. In this way, you will learn from each other. The goal is to be able to reach agreement on the rating of as many faces as possible. This is how we will figure out what is "right". Later, you will be rating faces on your own.

ANY QUESTIONS?

The scale you will be using to rate the SE of the faces is a 7-point scale. For this scale, "1" is the lowest degree of engagement, "7" is the highest, and "4" represents the mid-point.

RESEARCHER DIRECTS RATERS TO SCALE.

Let's review each scale point, and what it means.

<u>Social Engagement Scale:</u>

1 – "Minimally engaging." In other words, the face conveys almost no or minimal SE.

2 – "Slightly engaging." The face conveys some, but only slight, SE.

3 – "Somewhat engaging." The face conveys more than a slight degree of SE, but just less than the middle point on the scale.

4 – "Moderately engaging." This is the mid-point on the scale which indicates moderate SE.

5 – "Quite engaging." The face conveys just more than moderate SE.

6 – "Very engaging." The face is very engaging, but not quite at the top of the scale.

7 – "Extremely engaging." This is the highest degree of SE that a face can convey.

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