The Effects of a Voice Treatment on Facial Emotional Expression in Parkinson's Disease: Expressivity, Experience, and Gender

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THE EFFECTS OF A VOICE TREATMENT ON FACIAL EMOTIONAL
EXPRESSION IN PARKINSON'S DISEASE: EXPRESSIVITY, EXPERIENCE, AND
GENDER

by

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THE CITY UNIVERSITY OF NEW YORK
ABSTRACT

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Advisor: Professor Joan C. Borod, Ph.D.

Individuals with Parkinson’s disease (PD) suffer from decreased ability to express emotion through facial expression, in what has been termed “masked facies” or hypomimia. Facial emotional expression is necessary for the accurate communication of needs, to obtain or maintain empathy from care-givers, and to be perceived by others in a way that matches the way that one feels. The current study provides a review of the deficits seen in Parkinson’s disease, an overview of the neurobehavioral disparity of spontaneous versus posed facial expression of emotion, and factors that influence the perception of emotion, such as gender and clinical variables. The relationship between the experience and expression of emotion is also discussed. Further, theorized neural mechanisms underlying a current treatment strategy for Parkinson’s disease (i.e., The Lee Silverman Voice Treatment – Loud [LSVT – LOUD®; Ramig et al., 2001]) is described. Finally, the neurobiological correlates between vocal production and facial expression were examined. The current study had three aims. The first one was to explore whether LSVT-LOUD® affects spontaneous facial emotional expression in PD compared to healthy control groups. Second, we examined whether the internal emotional experience of individuals with PD was related to their expression of facial emotion. The third aim was to explore whether there were gender differences in how men and women evaluated same- and opposite-gendered facial expressions of emotion.
Eighty-two individuals comprised the “the poser participants” (i.e., the participant groups of this study), which included PDs receiving the experimental treatment related to voice amplitude (LSVT-LOUD®; Ramig et al., 2001), PDs receiving a control therapy involving articulation (LSVT-ARTIC®; Ramig et al., 2015), PDs not receiving any therapeutic treatment (PD-untrx), and demographically-matched healthy controls (HCs). Using procedures from the New York Emotion Battery (Borod, Cicero, et al., 1998; Borod, Welkowitz, & Obler, 1992), all participants, while being videotaped, were asked to recall a previously experienced emotional event that was happy, sad, or angry, as well as a neutral non-emotional event. Participants’ self-reported experience of each emotion was also recorded. Twenty-four undergraduate-student rater participants, naïve to the hypotheses of the study, viewed 15-second silent video clips of posers as they recalled the previously experienced emotional or non-emotional events. The “raters” evaluated each video on a 7-point Likert scale, from 1 (“very little”) to 7 (“extreme amount”), for facial emotional expressivity, in terms of emotional frequency, emotional intensity, and emotional variability, as well as social engagement and facial mobility (a non-emotional measure).

Our results indicated that the PDs in our sample demonstrated impaired facial expressivity relative to HCs. Contrary to our expectation, we did not find an effect of LSVT-LOUD® treatment on PDs’ spontaneous facial emotional expressions. Second, when exploring whether PDs and HCs experience monologue emotions similarly, we found no differences between the two participant groups. Finally, when viewing same- and opposite-gender facial expressions of emotion, male raters rated all posers as more facially expressive than did female raters. Female raters rated female posers as significantly more facially expressive than did male posers. The results of the current study further characterize the emotional deficits seen in PD and are discussed in terms of clinical implications.
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THE EFFECTS OF A VOICE TREATMENT ON FACIAL EMOTIONAL EXPRESSION IN
PARKINSON'S DISEASE: EXPRESSIVITY, EXPERIENCE, AND GENDER

CHAPTER I.

Introduction

Whether it is the rising of the corners of the lips in a smile or the furrowing of one’s brow in disdain, facial expressions are considered the primary way humans communicate emotion. Facial expressions act as social cues for the emotional content of an individual’s internal experience (Ekman, 1997). Appropriate displays of facial expression parlay necessary information needed for contextually accurate and appropriate social interactions. This applies not only to everyday encounters but may play an important role in patient-physician interactions, which are imperative for appropriate diagnosis and care. Very few diseases rob an individual of the ability to express emotion, specifically, facial expressions of emotion. Parkinson’s disease (PD), a disorder most widely characterized by the presence of a resting tremor and general motor impairments, is one such disease.

This review focuses on the facial emotional expressivity disturbances seen in Parkinson’s disease. To provide context and support for the proposed research, emotional processing and the relationship between gender and the expression and perception of emotion in PD are discussed. Posed and spontaneous facial emotional expressions are discussed separately due to their assumed neuroanatomical independence and behavioral disparity. Second, this review will also examine the relationship between one’s internal experience of emotion and external expression of that emotion. Next, fundamental to the proposed research, there will be a discussion of a current
treatment strategy to improve facial emotional expressivity in PD and the neural mechanisms proposed for their effectiveness. Finally, there will be a brief discussion of the neurobiological correlates between vocal production and facial expression, which are critical to the research proposed.

Specifically, the aims of this review are: 1) to explore whether the Lee Silverman Voice Treatment (LSVT-LOUD®; Ramig et al., 2001) affects spontaneous facial emotional expression in PD patients as compared to a treatment control group (i.e., LSVT-Articulation [LSVT-ARTIC®, Ramig et al., 2015]) and healthy controls (HCs), 2) to examine whether subjective internal emotional experience is related to facial expression of emotion, and 3) to explore whether there are gender differences in how men and women view same- and opposite-gendered facial emotional expressions.

**Emotion and Facial Expressivity**

Emotion has been conceptualized as a “feeling state” which encompasses several processes, including cognitive appraisal or perception, goal-directed activity, expressive behavior, physiological arousal, and subjective experience or feelings (Plutchik, 1984, 2000). Physiological arousal is when certain types of physiological activation (e.g., hormones, the autonomic nervous system, or neurotransmitters) are associated with emotional states (Péron, Dondaine, Le Jeune, Gandjean, & Vérin, 2012). Although the timing and antecedents of such processes are still debated among emotion theorists, most would agree that emotion is a process that includes these components. For a discussion of this issue, see Borod (1993). Put simply, emotions are reactions to real or imagined evocative stimuli (Plutchik, 1984).
The intricacies and variability of emotional states are contingent upon a core set of universal basic emotions, which include fear, anger, sadness, disgust, surprise, and expressions of enjoyment (Ekman, 1993). In addition to these universal emotions, human beings experience and express a number of more complicated social and self-referential emotions, such as embarrassment, guilt, pride, and shame (Izard, 1971; Tracy & Robins, 2004, 2007). Both complex and basic emotional states are generally accompanied by physiological arousal which most often includes either subtle or overt changes in facial expression. It is well-established that facial expression and the recognition of basic emotions displayed via the face are remarkably consistent globally and cross-culturally (Ekman & Friesen, 1971; Izard, 1971). It has been discovered that the facial muscles utilized for basic emotions vary little among all humans, whereas the facial muscles recruited for all other emotions vary greatly among individuals (Waller, Cray, & Burrows, 2008). This sheds new light on why basic emotions are produced universally and recognized a majority of the time. It is still not known whether more complex “self-conscious” emotions (e.g., shame, embarrassment, and pride) may also be universally processed (Tracy & Robins, 2004).

From a psychoevolutionary perspective, humans prosper in a social atmosphere contingent upon interpersonal relationships, where accurate displays of emotional expression and successful interpretation of such expressions are of great importance. The basic propensity to recognize and correctly interpret emotional expressions engenders greater reproductive fitness (Plutchik, 2000). Through various configurations of facial movements, individuals convey both emotional and non-emotional information to express or even disguise feelings and emotional responses. The natural environment creates survival problems for all organisms that must be successfully surmounted. This includes appropriate responses to prey and predators, to care solicitors and caregivers, and to
potential mates. Because social interactions affect the survival advantage of an individual, and, thus, their inclusive fitness, the goals and behaviors of an individual need to be accurately perceived, recognized, and understood by others for advantageous social welfare (Parr, Waller, & Fugate, 2005; Plutchick, 2000). Further, within these contexts, many individuals are able to parlay this knowledge into the more cognitively and developmentally advanced ability of being able to project one’s self into the mental state of another, referred to as Theory of Mind (Gallup, 1982).

**Variability in Facial Emotion Processing**

In spite of the universality of facial emotion recognition, proficiency and production of facial emotional displays are subject to individual variability, including the reported effects of race (e.g., Anthony, Copper, & Mullen, 1992; Elfenbien & Ambady, 2002a, 2002b; Matsumoto, 2002; Zebrowitz, Kikuchi, & Fellous, 2010), gender (e.g., Grunwald et al., 1999; Hall, 1978; Hall & Matsumoto, 2004; Zebrowitz et al., 2010), and age (e.g., Borod et al., 2004; Hillier, Beversdorf, Raymer, Williamson, & Heilman, 2007).

In a meta-analysis by Elfenbein and Ambady (2002a), researchers found that there appears to be an in-group advantage in perceiving emotional expressions of others, such that emotional communication is generally more accurately perceived when the expressor and perceiver share membership in the same national, ethnic, or cultural group. Further, in a meta-analysis by Anthony et al. (1992), it was found that individuals are more accurate in facial recognition tasks of same-race members as compared to other-race members, lending further support to an in-group advantage in the perception of facial emotional expressions. In fact, new research suggests that individuals perceive neutral facial expressions of other-race members as portraying more negative emotions as compared to neutral expressions of in-race members (Zebrowitz et al., 2010).
The processing of emotional stimuli may also be subject to changes across the adult lifespan (Grunwald et al., 1999). Research has indicated that older individuals perceive lexical emotional stimuli with less accuracy than younger individuals (Grunwald et al., 1999; Hillier et al., 2007). It has also been found that older individuals perceive posed-emotional facial expressions less accurately than younger individuals (e.g., Borod, Teague, Myers, & Kirch, 2010; Malatesta & Izard, 1984; Savage et al., 2013). In contrast, Levenson, Cartensen, Friesen and Eckman (1991) did not find significant age-related differences during the perception of emotional faces.

In terms of the production of emotional facial expressions, research indicates that older individuals may be less accurate in displaying posed emotional expressions as compared to younger individuals (Borod, Yecker, Brickman, Moreno, Sliwinski, et al., 2004; see, also, Levenson et al., 1991). In contrast, research examining spontaneous facial expression has not indicated changes in the accuracy of facial emotion as a function of age (Levenson et al., 1991; Malatesta, Izard, Culver, & Nicholoich, 1987; Malatesta, Jonas, Shepard, & Culver, 1992). Using odors to elicit facial expressions of emotion, it was determined that older individuals show decreases in spontaneous facial emotion, as compared to younger individuals (Simons, Ellgring, & Pasqualini, 2003). These findings are in support of previous research that finds decreases in the amount of facial expressivity in older adults (e.g., Diener, Sandvik, & Larsen, 1985; Lawton, Kleban, Rajogopal, & Dean, 1992).

**Gender and facial expressions of emotion.** In terms of gender, the literature indicates that emotional processing may differ between the sexes (for review, see Borod & Madigan, 2000). An abundance of research has reported that women, as compared to men, may be more accurate
perceivers of emotional stimuli (e.g., Grunwald et al., 1999; Hall, 1978; Otta, Ambrosia, & Hoshino, 1996; Shields, 1991). In fact, women may also be more emotionally expressive than men. When analyzing posed and spontaneous facial expressions of emotion, Simons et al. (2003) found that women were more facially expressive than men, regardless of experimental condition. More importantly, evidence suggests that women and men may be differentially effective at displaying certain emotions. When presented negatively-valenced images (i.e., gruesome pictures of skin disease and severe injuries), Walbott and colleagues found that women exhibited more facial activity than did men (Wallbott, Harald, Scherer, & Klaus, 1991). Hess and colleagues created avatars (i.e., digitally created human figures) of men and women with comparable facial features. They found that the female avatars were perceived as expressing more intense anger and that the male avatars were perceived as expressing more intense happiness (Hess, Adams, & Kleck, 2004). Research by Kring and Gordon (1998) found that after watching emotional films, women were more facially expressive than men for both positive and negative emotions. Both studies suggest a gender by valence interaction for facial emotional displays.

**Facial expressivity among clinical populations.** Within the clinical realm, there is large body of literature to suggest that facial emotional expressivity can be compromised in several neuropsychological disorders such as schizophrenia (e.g., Blonder, Burns, Bowers, Moore, & Heilman, 1991; Borod et al., 1990; Cohen & Minor, 2010; Kring & Moran, 2008; Martin, Borod, Alpert, Brozgold, & Welkowitz, 1990), depression (e.g., Borod et al., 1990; Jaegar, Borod, & Peselow, 1986), stroke (e.g., Borod, 1992; Borod, Koff, Lorch, Nicholas, & Welkowitz, 1988; Kazandjian, Borod, & Brickman, 2007; Montreys & Borod, 1998), Alzheimer’s disease (e.g., Henry, Rendell, Sciclun, Jackson, & Phillip, 2009), and Parkinson’s disease (Borod et al., 1990; Martínez-Corral et al., 2010; Miller et al., 2006; Pilowsky, 1988; Pitcairn, Clemie, Gray, &
Pentland, 1990; Smith, Smith, & Ellring, 1996). Of particular interest are the emotional processing deficits seen in Parkinson’s disease (PD; for review, see McCabe, Borod, Meltzer, Spielman, & Ramig, 2010). Patients with PD demonstrate a decreased ability to express emotion through their face and voice.

**Parkinson’s Disease**

Nearly 1 million Americans and 10 million individuals worldwide live with Parkinson’s disease (Parkinson’s Disease Foundation, 2016), and it is the second most common neurodegenerative disease after Alzheimer’s disease (e.g., Nussbaum & Ellis, 2003). PD is a progressive neurodegenerative movement disorder that is associated with the depletion of dopaminergic neurons in the substantia nigra pars compacta (e.g., Dauer & Przedborski, 2003). This leads to denervation of the nigrostriatal tract and the reduction of dopamine in the striato-pallidal and pallido-thalamic pathways. This is what is thought to be responsible for the motor deficits seen in PD patients. The telltale signature of PD is Lewy-body pathology (e.g., presence of Lewy bodies and Lewy neurites) seen upon neuroanatomical post-mortem examination (e.g., Kalia & Lang, 2015).

The etiology of PD appears to be a result of both environmental and genetic determinants and varying symptomology. There are no diagnostic tests to confirm the presence of the disease during the early stages. Quite often, during the early stages of the disease, a diagnosis of PD is ascribed based on a patient’s response to the dopamine agonist Levodopa (e.g., Jankovic, 2008).

The classic motor symptoms of PD include rigidity, resting tremor, postural instability, and bradykinesia (e.g., McCabe, Borod, Meltzer, Spielman, & Ramig, 2010; Spielman, Borod, &
A diagnosis of idiopathic Parkinson’s disease (IPD) is given when at least two out of the four cardinal motor features of PD are present (Ward & Gibb, 1990). Non-motor symptoms of PD include constipation, hyposmia (i.e., olfactory dysfunction), excessive daytime sleepiness, and rapid eye movement sleep behavior disorder (e.g., Chen et al., 2015), as well as the neuropsychological disorders of depression, anxiety, and cognitive impairment (e.g., Aarsland et al., 2009; Katsikitis & Pilowsky, 1991). Another non-motor symptom that can occur is apathy (e.g., Pagonabarraga, Kulisevsky, Strafella, & Krack, 2015; Pedersen, Alves, Aarsland, & Larsen, 2011; Zgaljardic et al., 2007). Of particular note, there is evidence to suggest that non-motor (NM) symptomology may occur significantly earlier than the more widely observable motor symptoms (e.g., Chen et al., 2015; Dashtipour et al., 2015; Kalia & Lang, 2015; Tolosa, Compata, & Gaig, 2007). Research by Sauerbier, Jenner, Todorova, and Chaudhuri (2015) suggests that at least 6 non-motor subtypes of PD exist. These NM PD subtypes are being discovered before the observable motor symptoms develop and are characterized by patients presenting with specific clusters of the following symptoms: pain, sleep dysfunction, anxiety, cognitive problems, anosmia, weight loss or gain, fatigue, gastrointestinal issues, and apathy (Sauerbier et al., 2015). These symptoms may also include excessive daytime sleepiness. One theory suggests that these symptoms occur due to the Brack hypothesis, which assumes that there is underlying Lewy body pathology of the enteric nerves of the olfactory bulb and lower brainstem (Chen et al., 2015). Another theory suggests that decreased physical activity is an early symptom of PD, but also a catalyst in the degeneration process (Tillerson et al., 2002). In fact, Fischer et al. (2008) found that the PD group that participated in high-intensity work-outs (on a treadmill) showed significant increases in step and stride length, gait speed, hip and ankle joint movement, and improved
weight distribution when going from sit-to-stand (Fischer et al., 2008); these improvements were not seen in the low intensity work-out group or in the control group.

The expression of emotion by individuals with PD has been studied less extensively than the perception of emotion in others by those with PD. Recent research has seen an increased focus on the emotional expressivity deficits evidenced in PD. PD patients have characteristic masked or blunted facial expressions that manifest themselves as a reduction in spontaneous blink rate, reduced facial mobility, and decreased facial expression. This has been referred to as “masked facies” (e.g., McCabe et al., 2010), “hypomimia” (e.g., Mergl, Mavrogiorgou, Hegerl, & Juckel, 2005; Priebe et al., 2015), or “mimetic facial paralysis” (Rinn, 1984). These terms refer to a general reduction in the speed of facial motor movement with specific decreases in intensity and variability of facial movements (e.g., Borod et al., 1990; Bowers et al., 2006; Smith et al., 1996). Hypomimia results in a general outward impression of a flat countenance and affect (e.g., Dumer et al., 2014). This reduction of facial expressiveness has been shown to be most notably apparent during the process of non-verbal facial emotional expression (e.g., Smith et al., 1996). Patients’ reduced ability to express their emotions through facial expressions can lead to negative social interactions and impaired social communication.

**Emotional Expression and Parkinson’s Disease**

**Social consequences of disordered facial communication in PD.** The emotional expressivity deficits seen in PD lead to various problems in social communication and interpersonal relationships (e.g., Brozgold et al., 1998; Dumer et al., 2013; Gunnery et al., 2016; Pitcairn et al., 1990; Scott, Caird, & Williams, 1984; Smith et al., 1996). PD patients are also often perceived as relating poorly to others, as not enjoying social interactions, and as being less
likeable (Pentland, Gray, Riddle, & Pitcairn, 1988). In fact, PD patients notice others’ reactions to changes in their communication and, as a result, become embarrassed and withdrawn (Miller, Noble, Jones & Burn, 2006). Pentland and colleagues found that during social interactions, PD patients are more likely to be assigned negative attributions by others, such as being anxious, depressed, unhappy, tense, hostile, and suspicious. PD patients were also seen as more introverted, passive, sensitive, and less intelligent. Further, in a study by Tickle-Degnen and Lyons (2004), novice healthcare practitioners incorrectly perceived PD patients as being more neurotic and more introverted based on the amount of facial masking they observed. Specifically, patients with decreased facial expressivity were viewed as more neurotic and less extroverted. It is interesting to note that in earlier findings by Pentland et al. (1998), the negative attributes of “anxious” and “tense” are closely related to “neuroticism” in the Tickle-Degnen and Lyons study (2004). Further, Pentland et al. (1998) also found that PD patients were perceived as more introverted compared to healthy controls. In a more recent study by Tickle-Degnen and her colleagues (2011), American and Asian (i.e., Taiwanese) medical professionals viewed videos of PD patients and healthy control participants recalling a previously enjoyed event. All practitioners were blind to the true purpose of the study. The researchers found that the practitioners of both nationalities judged the patients with facial masking as less cognitively competent, depressed, less social, and less socially supportive relative to the healthy participants (Tickle-Degnen, Zebrowitz, & Ma, 2011). Further, the health professionals viewed American PD patients with facial masking as less sociable than Taiwanese PD patients with comparable deficits in facial expressivity. In general, American practitioners were more biased by facial masking, when judging sociability, as compared to the Taiwanese practitioners. The study found that Taiwanese practitioners, as compared to the American practitioners, were more negatively biased by facial masking when
judging the *cognitive competence* and *social supportiveness* of the Taiwanese individuals. Of note, the health professionals’ negative attributions towards facial masking were greater for female participants than male participants, and even more so for *American* women (Tickle-Degnen, Zebrowitz, & Ma, 2011). The findings of this study suggest that the practitioners’ negative bias to facial masking reflects one’s own explicit, implicit, gender and cultural norms, and stereotypes. Not only do these findings suggest that Eastern and Western practitioners demonstrate a negative bias towards facial masking, their attributions were moderated by the effects of the practitioners’ culture and gender of the PD patient.

Tickle-Degnen and colleagues were not the only investigators to examine the role of gender in impression formation involving PD patients with facial masking. When viewing videotaped interviews of men and women with PD and subsequent facial masking, individuals expressed less interest in potential relationships with women with greater facial masking, and they judged the women as less supportive than women without PD. The same results were not true for the men who displayed comparable facial masking (Hemmesch, Tickle-Degnen, & Zebrowitz, 2009). It is important to note that in this study, the individuals making the judgments were of the patient population’s peer group. More specifically, these were older adult observers who also made attributional judgments after viewing segments of videotaped interviews of each of the members of the study participants. These findings were further supported by a more recent study by Hemmesch (2014). In a similar view, older adults judged PD patients with greater facial masking as being more socially negative. Again, the negative bias toward facial masking was greater for the female patients, as compared to the male patients.
Investigating the role of valence and facial expressivity in PD, researchers demonstrated that positively charged interview questions facilitated, while negatively charged interview questions inhibited, facial expressivity in PD patients (Takahashi, Tickle-Degnen, Coster, & Latham, 2010). Further, researchers Simons, Ellring, and Pasqualini (2003) found that PD participants contract fewer facial muscles when reacting to unpleasant stimuli as compared to HCs.

In a recent study, Gunnery et al. (2016) investigated how hypomimia severity correlated with PD patients’ and caregivers’ interpersonal relationships and social lives. Both the PD group and the caregiver group answered questionnaires regarding either their own degree of hypomimia or the degree of hypomimia for the individual whom they care for. They also answered a variety of questions pertaining to their own quality of life. Both PD-reported and CG-reported hypomimia were positively correlated with both partners feeling social rejection. This positive correlation diminished when researchers controlled for depression. While still controlling for depression, researchers found that the greater the caregiver reported hypomimia, the less enjoyment they had interacting with their partner (Gunnery et al., 2016). This study elucidates some of the problems that occur in real interpersonal relationships for PD patients and their caregivers, who oftentimes are loved ones.

It is still unclear whether all PD patients suffer from masked facies symptomology, as the literature remains equivocal. Findings have been inconsistent and have varied as a function of elicitation condition (Borod et al., 2004). In a review of 23 studies, 57% of the studies examined found significant facial expressivity deficits in individuals with PD (Zgaljardic, Borod, Foldi, & Mattis, 2003). In a more recent review of the literature (i.e., between 2000 and 2006), McCabe et
al. (2010) reported that 60% of studies investigating emotional processing deficits in PD reported facial expressivity deficits.

However, it is important to better understand the nature of such expressivity deficits in order to develop tailored rehabilitative strategies and for comprehensive scientific understanding. Before the potential for such interventions are discussed, further delineation of the facial emotional expressivity deficits seen in PD patients is presented.

The relationship between facial emotional expression and experienced emotion. In healthy populations, facial movements provide accurate representations of the subjective experience of emotion (Ekman, Friesan, & Ancoli, 1980). In addition, the extent to which a person experiences an emotion can be measured and distinguished through one’s facial expressions (Ekman et al., 1980). Furthermore, contracting the same muscles that are utilized in making facial expressions during an emotional scenario leads to increased emotional experience (e.g., Duclos & Laird, 2001; Laird, 1974; Strack, Martin, & Stepper, 1988; Soussignan, 2002), whereas inhibiting facial expressions during an emotional scenario decreases the strength of emotional experience (e.g., Davis, Senghas, & Oschner, 2009; Duclos & Laird, 2001). This is thought to occur due to the Facial Feedback Hypothesis (FFH). According to the FFH, skeletal muscle feedback from facial expressions plays a causal role in regulating emotional experience (Buck, 1980). In the classic study by Strack et al. (1988), half of the participants were directed to contract muscles associated with smiling while reading humorous comic strips. Those who contracted the muscles that are engaged when smiling reported having an enhanced positive emotion in response to the comic strip, compared to the control group.
There are certain cases where facial expressions of emotion differ from the internalized experience of emotion. As stated a bit earlier, women tend to produce more facial emotional expressions than men, even though women’s experience and physiological response to emotion is equivalent to men (e.g., Kring & Gordon, 1998). When investigating patients with lateralized brain damage, Montreys and Borod (1998) found that right-brain-damaged patients produced less intense facial emotion and reported less intense experience of emotion compared to the left-brain-damaged group. However, there was not a positive correlation between experienced emotion and displayed emotion, for each of the brain-damaged groups. Despite exhibiting some deficits in the behavioral expressions of emotion, Alzheimer’s disease patients experience emotion the same as their age-matched controls (Henry, Rendell, Scicluna, Jackson, & Jackson, 2009).

Further research into this area has, for the most part, concluded that among healthy participants, facial expressions of emotion and the experience of emotion are correlated (e.g., Montreys & Borod, 1998) and that emotional expressions of the face can influence one’s internal experience (e.g., Duclos & Laird, 2001; Larsen, Kasimatis, & Frey, 1992). It is through this mechanism that it was first hypothesized that PD individuals might experience a reduction in their internal emotional experience due to their reduced ability to demonstrate facial displays of emotion.

Surprisingly, most studies suggest that Parkinson’s disease patients appear to experience emotion similarly, and with the same intensity, as healthy controls (e.g., Halfacre et al., 2009; Madeley et al., 1995; Smith et al., 1996; Zgaljardic et al., 2003). In a study by Mikos et al. (2009), researchers compared self-ratings of emotional expressivity to ratings made by a family member or a close friend. PD participants reported experiencing emotions as intensely as healthy
participants, despite rating their own facial expressions of emotion as less intense. They also found that PD participants accurately appraised their decreased facial expressivity (Mikos et al., 2009). These findings are in support of research by Borod et al. (2008) where it was found that PD patients experience emotion with the same intensity and accuracy as HCs. In addition, PDs’ emotional experience was positively correlated with their observed intensity of facial emotion and their frequency of facial emotions.

Simons and colleagues (2004) quantified spontaneous expressions of amusement and correlated them with self-report measures of how amused PD participants felt while watching a humorous video. Just as in the Borod et al. (2008) and Mikos et al. (2009) studies, a link between emotional experience and expressed emotion in PD was demonstrated. Specifically, the amount of spontaneous facial expression demonstrated by the PD group while watching a humorous video positively correlated with self-reported measures of how amused the PD participants felt during the video. Despite the PD group’s overall reduction in facial emotional intensity, a link between emotional expression and experience was present. Of note, this finding was not present during the other conditions of emotional posing, non-emotional posing, or pleasant conversations with a spouse or stranger (Simons et al., 2004). It was speculated that the same result for the spontaneous humor condition would not be found during the emotional posing and non-emotional posing conditions, as posing or voluntary expressions are not being produced from a genuine internal place of feeling or emotion (Simons et al., 2004). Furthermore, it is possible that for the pleasant conversation condition, with either a spouse or a stranger, this condition did not produce a strong enough emotion in the PD group. These findings support other research (e.g., Madeley et al., 1995; Smith et al., 1996) where emotional experience in PD seems to be dissociated from facial expression.
However, despite convincing evidence, not all research has supported that PD patients experience emotion in the same way as HCs. In a review of the literature, Peron et al. (2012) suggest that PD patients demonstrate blunted physiological arousal, startle-reflex, and emotional appraisal.

**Hypomimia: Volitional (posed) vs. spontaneous facial emotional expression.**

Voluntary (i.e., also termed posed) and spontaneous (i.e., involuntary) facial expressions of emotion rely on relatively distinct neuroanatomic pathways (e.g., Borod, Haywood, & Koff, 1997). Given that PD is characterized primarily by motoric deficits, it has been proposed that voluntary/posed and spontaneous facial emotional expressions would be differentially affected in PD patients with masked facies (e.g., Heilman, Blonder, Bowers, & Crucian, 2000). Spontaneous movement is largely controlled by extrapyramidal and subcortical regions, such as the basal ganglia (which also controls involuntary movement); whereas, voluntary/posed movements are controlled by cortical areas in the frontal lobe (e.g., Blair, 2003; Borod & Koff, 1984). Therefore, it has been postulated that PD patients would have difficulty producing spontaneous facial expressions (i.e., subcortical/basal ganglia) as compared to posed facial expressions (i.e., frontal cortex). In one of the first studies to investigate spontaneous facial expression in PD, Buck and Duffy (1980) found that when PD participants viewed emotionally evocative stimuli, raters viewed their facial expressions as less expressive than those of the control group. Second, the raters were not able to accurately discern which emotions the PD participants were displaying. Using an anatomically based coding system, called FACS (The Facial Action Coding System; Ekman & Friesen, 1978), Smith et al. (1996) and Simons et al. (2003) found that, relative to controls, PD participants demonstrated decreased intensity in muscle movement, less facial mobility, and fewer “sincere smiles” (i.e., what is referred to as the “Duchenne smile”). One of
the easiest ways you can determine the difference between a Duchenne smile and a non-Duchenne smile is that a Duchenne smile will also show certain movements around the eyes. During a Duchenne smile, there is wrinkling that occurs at the outer corners of each eye and, sometimes, the eyes are slightly squinted. Within pop culture, on the popular reality television show America’s Next Top Model, Tyra Banks referred to these very specific eye movements as “smizing,” meaning smiling with your eyes. Since then, “smizing” has become a widely-recognized word by millions (Graham, 2012).

McCabe et al. (2010) reported that the total number of smiles between PD individuals and HCs do not seem to differ; however, PDs exhibited fewer Duchenne smiles. Halfacre et al. (2009) posit that PD individuals are aware of their diminished spontaneous smiling behavior and, therefore, compensate by purposely posing a forced (or non-spontaneous smile). Alternately, spontaneous smiling behavior in PD may appear non-Duchenne due to the way the hypomimia affects the muscles of the face. It is quite possible that PD-induced hypomimia favors the lower portions of the face while causing more pronounced deficits in the upper regions of the face (McCabe et al., 2010). Investigating both spontaneous and posed facial expressions in the same group of participants, Brozgold, Borod, Rosen, and Alpert (1999) found that PD participants were less expressive than controls when engaged in spontaneous displays of facial emotion; however, the PD group and control group did not differ in expressivity when engaged in posed facial emotions. These results suggest that spontaneous facial expressions are impaired in PD patients, relative to healthy controls.

In a slightly different study, investigating spontaneous emotional facial expressions in PD, Livingstone, Vezer, McGarry, Lang, and Russo (2016) assessed the mimicry behavior in PD
individuals. When healthy individuals view emotional facial expressions, although usually unaware, they often react with brief automatic facial movements that mimic the face that they are viewing. These movements are thought to play a role in social communication and have been termed “facial mimicry” (Livingstone et al., 2016). Mimicry is not exactly the same as spontaneous movement, as spontaneous mimicry relies on the recruitment and firing of mirror neurons in the premotor cortex. Twenty-seven non-depressed PD individuals and matched controls viewed calm, happy, sad, angry, and fearful emotional facial expressions. Overall, the PD patients displayed less mimicry compared to the controls. The deficits seemed to vary as a function of valence, where mimicked frowns were decreased somewhat but were nowhere near the profoundly weakened and delayed mimicry smiles.

Using odors to elicit spontaneous unpleasant facial emotions, Simons et al. (2003) found that the PD patients exhibited less spontaneous facial expressivity relative to healthy controls. The PD patients also demonstrated a reduction in facial expressivity under a combined expression task. Participants were asked to pose either a negative or positive facial expression while simultaneously smelling an unpleasant odor to produce a mixed expression. In the congruent-posing condition, the participant would pose a negative expression while simultaneously posing an unpleasant odor. In the incongruent-posing condition, the participant would pose a positive facial expression while smelling an unpleasant odor. Having participants smell an unpleasant odor either increased their negative facial expression or masked their negative facial expression. Interestingly, the congruent-posing condition (i.e., smelling an unpleasant odor while posing a negative facial expression) elicited the greatest amount of facial expressivity from the PD participants; however, overall, the PD group demonstrated a significant reduction in posed facial expressivity compared to the control group (Simons et al., 2003). It is important to note that the
elicitation procedures of the Simons et al. (2003) study utilized noxious odorants to elicit facial expressions of disgust and discontent and a pleasant smell condition was not employed. Moreover, other more naturalistic studies have employed the manipulation of mood by either having the participant watch a video that induces a feeling state or having the participant recall an event that causes them to experience emotion. The varying methods of emotional elicitation may be why there is conflicting evidence in the literature regarding whether spontaneous facial emotion in PD is preserved or impaired. In fact, Bowers et al. (2006b) found that PD patients demonstrated blunted reactivity to aversive stimuli and speculated that this muted effect could reflect a “bradylimbic” disturbance that is due, in part, to connections between the amygdala and the basal ganglia (Bowers, 2006b).

However, all of the research in this area remains equivocal. There are studies which have not found significant differences in spontaneous facial expressivity between PDs and healthy controls (e.g., Madeley, Ellis, & Mindham, 1995; Smith et al., 1996).

Again, since posed movements are largely controlled by cortical areas (which control voluntary movement), it has been postulated that posed facial emotional expressions would be relatively spared in PD (Blair, 2003). Yet, Borod and colleagues (1990) found that PD patients were less accurate in displaying posed emotional facial expressions as compared to HCs. Bowers et al. (2006a) argued that posed facial expressions would be influenced by PD in the same way intentional movements of the limbs are affected by bradykinesia. Their study concluded that relative to healthy controls, PD patients’ posed facial emotional expressions were generated more slowly and were of lower expressive amplitude. Yet, Bowers et al. (2006a) conclude that their study should serve as evidence that there is not a neuroanatomical dissociation between voluntary
and spontaneous facial expressions in PD, despite conflicting evidence (e.g., Blair, 2003; Borod & Koff, 1984; Brozgold et al., 1999). Jacobs, Shuren, Bowers, and Heilman (1995) demonstrated that PD patients posed less intense facial emotional expressions than did healthy controls. More specifically, the PD and HC groups differed significantly in producing sad and angry expressions. In order to examine the relationship between posed smiling and abnormalities of voluntary movement, Marsili et al. (2014) compared the differences in facial expressivity of PD patients during a posed-smiling condition and a matched lower-face voluntary-control condition (i.e., participants were asked to voluntarily show their teeth as quickly and widely as possible). The authors found that facial movements in the PD group were decreased during both the posed-smiling and voluntary-grinning conditions. The amount of movement for the posed smiles correlated with those of the volitional control condition, but only for the PD group. Abnormal movement during the posed or volitional condition was related to overall PD motor severity but, interestingly, did not improve whether or not patients were on or off L-dopa therapy (Marsili et al., 2014).

In order to clarify the role that elicitation procedure has on facial expressivity, researchers analyzed posed and spontaneous facial expressions of PD and HC participants across a number of experimental elicitation conditions (Simons, Pasqualini, Reddy, & Wood, 2004). Participants were shown humorous video clips in order to elicit the feeling of amusement. They then engaged in conversation on a positive/pleasant topic and were then asked to pose a happy, sad, angry, fearful, sad, surprised, or disgusted facial expression or to imitate a non-emotional facial movement. Researchers found that the PD participants demonstrated a reduction in spontaneous facial expressivity, relative to healthy controls, across all of the experimental conditions (Simons
et al., 2004). Furthermore, the PD participants were less expressive than the controls when posing emotional expressions and imitating non-emotional facial movements.

In one of the most recent studies published on facial expressivity in PD, Priebe et al. (2015) examined facial expressions of pain in PD. Similar to the findings of Simons et al. (2004), facial displays of pain were decreased compared to controls. Interestingly, there were noticeable qualitative facial expressivity differences between the PD and HC groups during the experience of pain. In the HC group, narrowing of the eyes was the most frequent movement while experiencing pain; yet, this movement was demonstrated least frequently by the PD group, out of all of the pain-related movements. Further, the most frequently displayed movement for the PD group, while experiencing pain, was opening the mouth, which is not considered to be a pain-related movement. Priebe et al. (2015) found that the movements of the upper face (i.e., the narrowing of the eyes) was affected the most while the movements of the lower face, although atypical, were relatively preserved (Priebe et al., 2015). These findings, coupled with the findings mentioned earlier on non-Duchenne smiling in PD (McCabe et al., 2010), suggest that hypomimia may differentially affect the upper and lower face.

Rehabilitation of Hypomimia in Parkinson’s Disease

A variety of behavioral and neurological studies have looked at the expressive deficits in PD; however, very few have attempted to provide a treatment for hypomimia in PD (Dumer et al., 2014; Elefant, Lotan, Baker, & Skeie, 2012; Katsikitis & Pilowsky, 1996; Ricciardi et al., 2016; Spielman et al., 2003). The Katsikitis and Pilowsky (1996) treatment study investigated the effects of orofacial physiotherapy, specifically, by brushing the facial muscles of participants. Their findings were weak, at best. They found 1 statistically significant effect out of the 12
possible effects; PD participants could open their mouths wider, after treatment. This result could have simply been due to client/clinician interaction. In Elefant et al. (2012), they discovered that music therapy improved hypomimia in PD patients; however, the lack of a control group for comparison weakened the study’s methodology and veracity of their findings. A study by Ricciardi et al. (2016) involved a multi-modal approach in rehabilitating hypomimia. However promising, their study suffered from small sample sizes, thus preventing firm conclusions regarding the study’s results. In 2003, Spielman and colleagues first reported the preliminary data and results they obtained from treating 22 PD patients with LSVT versus a matched control group of 22 PD patients treated with a respiratory therapy (Spielman et al., 2003). The PD patients who received LSVT were rated as having greater facial mobility and higher levels of social engagement than the control group. These results were encouraging, and further studies have investigated the role of LSVT’s effectiveness in treating hypomimia.

**Vocal Deficits in Parkinson’s Disease**

The emotional deficits seen in PD are not limited to just the face, but are evidenced in the voice, as well. Nearly 90 percent of the 1.5 million patients with PD exhibit speech and voice symptoms, and at least 75% of patients with PD are classified as having a speech disorder; yet, only 3-4% of patients receive speech therapy (Logemann, Fisher, Boshes, & Blonsky, 1978; Ramig, Countryman, O’Brien, Hoehn, & Thompson, 1996; Ramig, Countryman, Thompson, & Hori, 1995). Hoarse voice quality, soft voice (i.e., hypophonia), monotone voice, breathy phonation, reduced and imprecise articulation, reduced prosody (i.e., hypoprosodia), and vocal tremors contribute to the dysarthria and communication issues seen in a large number of PD patients (Fox et al., 2006; Sapi, Spielman, Ramig, Story, & Fox, 2007). Additional speech
abnormalities, such as longer pauses during speech, decreased vocal sound pressure (SPL), shorter duration of sustained phonation, and an overall reduction in speech intelligibility has also been demonstrated (e.g., Ramig et al., 2004; Sapir, Ramig, & Fox, 2011; Sapir et al., 2007).

Unlike their facial expressivity deficits, PD patients are usually unaware of their own hypophonic deficits. Interestingly, when they are prompted to speak with increased volume, PD patients often report feeling as if they are speaking too loud (Liotti et al., 2003). Similar to the facial expressivity deficits seen in PD, their disordered speech limits their ability to function normally in society (Ramig et al., 1996).

The Lee Silverman Voice Treatment – LOUD (LSVT- LOUD®)

The Lee Silverman Voice Treatment (LSVT - LOUD®; Ramig et al., 2001) is an intensive speech treatment that trains PDs with hypophonia to speak with greater amplitude while self-monitoring the effort it takes to produce such vocalization (Ramig et al., 1996). LSVT-LOUD has been shown to be particularly effective in increasing vocal intensity/amplitude in PD individuals demonstrating hypophonia. The LSVT- LOUD treatment is designed to target inadequate muscle activation underlying hypokinesia (i.e., reduced amplitude of movement) and bradykinesia (i.e., slowness of movement) in PD (Fox et al., 2006). The program focuses on maximizing phonatory and respiratory functions by instructing patients to produce sustained loud phonations, using maximum effort (Sapir et al., 2011), in a variety of speech tasks (Ramig et al., 2001). It is hypothesized that intensive highly effortful voice therapy teaches the patient to rescale the magnitude of speech-motor output (Ramig et al., 1996). The LSVT capitalizes on the known effect for high effort (i.e., demonstrated through either complexity or difficulty) tasks, over an extended period of time (i.e., repetition), to result in rehabilitative cortical reorganization.
(Farley, Fox, Ramig, & McFarland, 2008). This effect has been successfully demonstrated in animal models of PD and transcranial magnetic stimulation (TMS) studies in humans (for a review, see Farley et al., 2008). The LSVT-LOUD treatment encourages increased vocal amplitude on the respiratory and articulatory systems of speech (e.g., Sapir et al., 2007). It is important for patients to have knowledge of how they are doing and to use that information going forward in treatment (Ramig et al., 1996). Feedback is given directly to the patient since he/she must rely on internal cueing to maintain the desired vocal amplitude changes over time (as opposed to relying on external cues). As a result, the training requires learning, memory, motivation, and self-reliance (Sapir et al., 2007). The entire treatment consists of sixteen 60-minute sessions, for a total treatment protocol of one month.

Voice abnormalities in PD patients have been attributed to asymmetric vocal fold tension or movements, stiffness or rigidity of the vocal folds and respiratory muscles, glottal incompetence, reduced laryngeal muscle activation or synergy, dysfunctional vocal fold adduction, and muscle atrophy or fatigue (Ramig et al., 2001; Sapir et al., 2007). Given that there is pathology observed in laryngeal and respiratory function in PD patients (Ramig et al., 1996) and that orofacial and extrinsic laryngeal muscles ultimately regulate the vocal tract and vocal amplitude (Sapir et al., 2007), vocal loudness serves as a trigger for distributed effects across the speech production system (Fox et al., 2006). The loud and phonatory tasks of LSVT-LOUD have been designed to improve vocal fold adduction, vocal tract configuration, respiratory drive, supralaryngeal articulatory movements, and laryngeal muscle activity and synergy (Ramig et al., 2001). Such exercises have been shown to improve voice quality, resonance, prosody, amplitude, articulation, and speech intelligibility. The increased vocal fold adduction allows patients to generate adequate intensity (Ramig et al., 1996). Loudness is generated largely from the
interaction between aerodynamic and vocal fold visoelastic forces (Sapir et al., 2007).

Individuals with PD integrate improved speech into functional communication (Fox et al., 2006).

However, the role of self-monitoring is a key aspect of the treatment which is critical.

In Narayana et al. (2009), PD patients displayed abnormalities in several speech regions, yet through the use of the LSVT-LOUD treatment, functional imaging showed that changes to these regions occurred. Using H20-Positron Emission Tomography (PET), Narayana and colleagues (2009) found a post-treatment related shift in neuronal activity toward the right hemisphere. Further, they found greater correlated activity within the right dorsolateral prefrontal (DLPFC), temporal (i.e., auditory), and speech-motor regions. Specifically, 1) the DLPFC is not an area activated during speech tasks in healthy individuals, 2) as speech became more normal in the PD group, regional cerebral blood flow (rCBF) in the DLPFC increased, and 3) rCBF is absent at the level of the basal ganglia. Taken together, these results suggest that the LSVT-LOUD intervention caused its effect via a top-down mechanism (Narayana et al., 2009). The LSVT-LOUD treatment, through a neuroplastic rehabilitative mechanism, increased recruitment of the right auditory cortex and improved communication among right-hemisphere (RH) speech-motor regions (Narayana et al., 2009). Such improvements to the sensory-motor speech system are thought to cause an auditory recalibration in which increased self-awareness of loudness is normalized (Narayana et al., 2009). The shift in activation to the RH demonstrates increased recruitment among the regions of the brain devoted to global aspects of prosodic speech, such as loudness, pitch, and tone (Narayana et al., 2009).

As of 2009, only 2 studies had investigated the differences between the speech motor areas of patients with IPD and those of HCs (i.e., Liotti et al., 2003; Pinto et al., 2004). During
speech tasks, PD patients in both studies had significantly greater rCBF in prefrontal and premotor cortices during speech tasks, as compared to matched healthy controls. In response to the decrease in blood flow for the areas involved in the planning of motor movements (i.e., premotor and prefrontal), both studies reported altered rCBF in the motor areas of the primary motor cortex and cerebellum, finding both increases (Liotti et al., 2003) and decreases (Pinto et al., 2004).

LSVT-treated patients improved or maintained vocal intensity at or above pre-treatment levels one year post-treatment. The respiratory control group’s vocal intensity deteriorated significantly at 12 months (Ramig et al., 1996). What has been the most interesting and surprising finding has not only been how LSVT has improved vocal intensity in PD patients but how it has contributed to increases in facial expressivity (Spielman, Borod, & Ramig, 2003). Initially, LSVT-LOUD was developed to treat voice and speech deficits in PD; the benefit of increasing facial expressivity (as demonstrated in Spielman et al., 2003) was, at first, an unexpected serendipitous finding.

**The Lee Silverman Voice Treatment – Articulation (LSVT-ARTIC®)**

The LSVT Articulation Therapy (LSVT – ARTIC®; Ramig et al., 2015) is another voice treatment developed by Ramig and colleagues (Spielman et al., 2012) intended to help improve articulation in PD patients. Due to several factors, including the same intensive schedule of treatment as the LSVT-LOUD program, LSVT-ARTIC is also similar to LSVT-LOUD in the amount and type of feedback given to a patient, as well as the amount of patient and therapist interaction. However, instead of focusing the treatment on increasing vocal amplitude, as in LSVT-LOUD, patients undergoing LSVT-ARTIC treatment focus on articulatory effort. The
treatment involves over-articulating vowel, consonant, and vowel consonant sounds. Due to their similar schedules of treatment and focus on the voice, it was hypothesized that LSVT-ARTIC might also have a positive effect on facial expressivity. However, recent studies have shown that such improvements are not seen after LSVT-ARTIC therapy. When considered from a neuroevolutionary perspective, post-treatment increases in facial expressivity accompanying improved vocal loudness suggests that a treatment aimed at increasing vocal amplitude may be stimulating phylogenetically-old neural centers of the brain, such as thalamic, limbic, and reticular connections (Spielman, 2000). Whether it’s a mating call or a warning that danger is approaching, phonation plays a large role in the vocalization of birds and primates (Petkov & Jarvis, 2012). Birds and primates have the same phylogenetically-old areas of the brain as we have (Petkov & Jarvis, 2012). However, articulation, an aspect of voice production specific to human beings, is controlled by the neocortex (Spielman, 2000), a phylogenetically-newer neural area. Since LSVT-ARTIC is acting at the cortical level, it is expected that it would not have the same effect on the subcortical emotional centers of the brain, thus, not impacting facial expressions of emotion.

Since LSVT-LOUD and LSVT-ARTIC share similarities such as high-effort, intensive treatment, clinician feedback, clinician-patient interaction, and a high focus on the same channel of communication (i.e., the voice), LSVT-ARTIC is the perfect treatment control group for the LSVT-LOUD group (Spielman, 2000). As such, the LSVT-ARTIC therapy will be used as a treatment control in the proposed study.

The Neuroanatomical Link Between the Voice and Face
The finding that a voice therapy could improve facial expressivity is supported by numerous theories linking vocal and facial expression (for review, see Bono & Borod, 2016). In one such study by McClean and Tasko (2002), correlations were found between lip, tongue, and jaw movement speed and breathing volume. These results suggest that there is an underlying neural substrate coordinating mouth movements for speech and breathing rate for vocalization. It has been suggested that the voice and face are linked components of a complex coordinated system designed to express one’s internal state (Kaiser & Scherer, 1998). According to Damasio (1995), there is an underlying common neural network between regions that mediate our emotional responses; these include the basal ganglia and the anterior cingulate cortex. In addition, there is a common facial musculature which expresses the functions of both language and facial expressions (Rinn, 1984). Both the larynx and face are innervated by the same lower motor neuron tract (i.e., the seventh cranial nerve or facial nerve; Rinn, 1984). Given these commonalities, it is not improbable to suggest that improvements in one system (e.g., the voice) may facilitate improvements in the other (e.g., the face).

If we are to understand the connectivity between the voice and face, we must first understand their shared neural mechanisms. The brain innervates the muscles of the face via the corticospinal tract (Blumenfeld, 2010). The corticospinal tract is composed of upper and lower motor neurons. Upper motor neurons carry electrical impulses from the primary motor area of the frontal lobe to the lower motor neurons located in the brain stem or spinal cord. These lower motor neurons consist of cranial nerves V and VII, which then carry the impulses to the face (Blumenfeld, 2010). Cranial nerve V (i.e., the trigeminal nerve) controls chewing and facial, mouth, and tongue sensations, while cranial nerve VII (i.e., the facial nerve) controls facial expressions as well as taste, lacrimation, and salivation (Blumenfeld, 2010). The facial nerve (i.e.,
cranial nerve VII) has five main branches that are responsible for different functions; further, different branches of this cranial nerve innervate the lower, middle, and upper portions of the face (Blumenfeld, 2010; Rinn, 1984).

Although the function and anatomy of the trigeminal and facial nerves are distinct and independent of one another, there is growing evidence to suggest connectivity between them (e.g., Felizardo et al., 2010; Tanaka, Yu, & Kitai, 1971). In an electrophysiological study, Felizardo et al. (2010) were able to demonstrate that the trigeminal nerve (responsible for somatosensation of the tongue) modulates the neurons arising from the facial nerve (responsible for taste) at the level of the Nucleus of the Solitary Tract (i.e., medulla and lower pons). The connectivity between the 5th and 7th cranial nerves may be a partial mechanism to explain the relationship between the face and voice.

However, some of the most convincing evidence comes from studies that have investigated the cortical and subcortical substrates involved in regulating vocal and facial expressions of emotion (e.g., Hopf et al., 1992; Jürgens & Zwirner, 1996; Özeren, Sarica, & Efe, 1994; Ross & Mathiesen, 1998). The anterior cingulate cortex (ACC) projects to the peri-aqueductal grey (PAG), an area that is thought to integrate motor systems for vocalizations (Blumenfeld, 2010). The medial dorsal nucleus of the thalamus (MDN) projects to the larynx directly and also via the PAG (Devinsky et al., 1995; Rinn, 1984). The MDN, as well as many other thalamic nuclei, communicate to and from the ACC, which is believed to be involved in automatic emotional vocalizations, as well as deliberate vocalizations, through its connection to motor neurons in the larynx (Devinsky et al., 1995; Ploog, 1987). It is through these thalamo-cortico connections that Devinsky et al. (1995) proposes that the striatum (receiving projections
from the basal ganglia) projects to the MDN which then projects to the ACC, in preparation for vocalization (Devinsky, 1995). This theory suggests that the thalamus is the main link between facial expressions of emotion and vocal production. In fact, a number of studies using electrical stimulation (e.g., Jürgens & Zwirner, 1996) or thalamic lesions (e.g., Hopf et al., 1992; Özeren, Sarica, & Efe, 1994; Ross & Mathiesen, 1998) lend support to this theory. In fact, lesions of the striatum have been known to cause emotional facial paresis, where spontaneous smiling or weeping can occur, but volitional movements of the face are preserved (Trosh, Sze, Brass, & Waxman, 1990). However, Davis, Zhang, Winkworth, and Bandler (1996) hypothesize that the PAG is the crucial brain site for mammalian voice production, not only for emotional vocalization or involuntary sounds, but for larynx control and respiration that are essential for human speech. Moreover, it has been discovered that mammals’ facial motor nucleus, which controls facial expressions, has widespread projections to respiratory-related areas of the brainstem (Li, Guan, Chan, & Zheng, 2004).

Jürgens and Zwirner (1996) discovered two vocal control pathways, one neocortical pathway and one limbic pathway, using electrical stimulation. Vocalizations by electrically stimulating the ACC and the hypothalamus ceased when a blocking agent was injected into the peri-aqueductal grey (PAG); however, this injection had no effect on vocal fold movements elicited by electrically stimulating the motor cortex of the squirrel monkey (Jürgens & Zwirner, 1996). These findings can be further supported by the findings of Brown, Ngan, and Liotti (2008); when using fMRI, researchers were able to map the movements of the larynx onto the motor strip of human primary motor cortex just slightly superior to where the lips are represented (Brown, Ngan & Liotti, 2008). In a more recent study, researchers discovered that injury to the
cerebellum can severely impair a person’s facial emotional expressivity and prosodic ability (Heilman, Leon, Burtis, Ashizawa, & Subramony, 2014).

Further evidence comes from studies investigating the relationship between facial and vocal expressivity. Borod, Koff, Lorch, and Nichols (1985) found significant correlations between facial expressivity and vocal intonation for both brain-damaged and healthy control groups. Another study evaluating correlations between vocal and facial expression of emotion with patient groups with PD, right-sided brain damage, depression, and schizophrenia, as well as healthy controls reported similar findings (Borod et al., 1990). Borod et al. (2000) was able to demonstrate support for a general processor for emotional perception tasks, across modalities (i.e., face, voice, and speech content) in 100 healthy men and women. Taken together, these studies support the idea of a central mechanism regulating emotional expression across various channels of communication.

Hypomimia limits PD patients’ ability to express themselves accurately. This leads to misattributions by others, PD patients feeling embarrassed to communicate (Miller, Noble, Jones, & Burn, 2006), loss of interest from caregivers (Gunnery et al., 2016), and overall decline in quality of life for the individuals affected. Therefore, it is imperative that we understand the nature of such expressivity deficits in order to develop tailored rehabilitative strategies and to further our scientific understanding of emotion.

To date, LSVT-LOUD has provided patients with a state-of-the-art (Trail et al., 2005) multi-dimensional treatment option to treat hypomimia in PD. LSVT-LOUD is based on principles derived from motor learning, muscle training, neurology, physiology, and neuropsychology (Trail et al., 2005). Researchers have learned that training a single motor control
parameter of vocal loudness can result in widespread and distributed effects, as seen in improvements in facial emotional expressivity. It is hypothesized that through neuroplasticity, a variety of motor behaviors, including those from seemingly different motor systems, such as speech and spontaneous facial expression, can show improvement (Fox et al., 2006). After LSVT-LOUD treatments, functional imaging revealed cortical activations more closely resembling activations seen in HCs; this included the additional recruitment of right anterior insular cortex, caudate, putamen, and the DLPFC (Liotti et al., 2003).

The purpose of the present study is to examine the effects of LSVT-LOUD on measures of facial expressivity in individuals with idiopathic Parkinson’s disease. In order to discern that LSVT-LOUD is responsible for the changes in facial expressivity post-treatment, participants will be randomly divided into a treatment group (LSVT-LOUD), an articulation control treatment group (LSVT-ARTIC), an untreated PD control group (PD-untrx), and a matched healthy control group (HC).

Aims

**Aim 1a.** To examine whether the Lee Silverman Voice Treatment (LSVT-LOUD®; Ramig et al., 2001) affects facial emotional expression in Parkinson’s disease patients during the recollection of past emotional experiences compared to demographically-matched healthy controls (HCs). Preliminary research (Spielman et al., 2003) showed that PD patients treated with LSVT-LOUD, compared to a control respiratory therapy, displayed a significant increase in facial mobility and social engagement after treatment. However, this preliminary study included a small sample and was limited in scope. The current study seeks to replicate these findings using a larger sample, while examining a wider range of parameters of facial emotional expressivity (i.e.,
emotional frequency, emotional intensity, and emotional variability), as well as social
engagement and facial mobility (i.e., a non-emotional control measure). Examining the effects of
LSVT-LOUD on these measures may provide insight into whether facial emotional expressivity
is mediated by a motoric, potentially neuroanatomical, mechanism or whether emotional
regulation is being more directly affected. We hypothesize that PD patients will show greater
increases in measures of facial emotion, from baseline to post-LVST-LOUD treatment relative to
post-LSVT-Articulation (LSVT-ARTIC®; Ramig et al., 2015) and healthy controls. Based on
previous findings (Spielman et al., 2003), we also predict that PD patients will demonstrate
increases in Social Engagement post-LVST-LOUD treatment relative to the LSVT-ARTIC group
and healthy controls.

**Aim 1b.** In an attempt to better characterize the facial emotional expressivity deficits seen
in PD, we utilized a recently developed (Borod et al., 2007) and more refined systematic rating
system where we divided the overall construct of Emotional Expressivity into three distinct
components: 1) Emotional Frequency (EF), 2) Emotional Variability (EV), and 3) Emotional
Intensity (EI). We anticipate that these measures will provide us with a more comprehensive way
to delineate the nature of the facial emotional expressivity deficits seen in PD and in other clinical
disorders. Moreover, we wanted to explore a unique aspect of the psychosocial impairment seen
in dyadic interactions between PD patients and the perceiver by including the variable “Social
Engagement (SE).” By including a measure of social engagement, we assumed a more
ecologically valid approach that might better characterize the everyday psychosocial and
communication deficits experienced by PD patients during dyadic encounters.
Aim 2. To examine whether subjective emotional experience (i.e., how intensely participants feel the emotion) during the recollection of past emotional events is related to the three measures of facial emotional expressivity. Based on the literature (e.g., Borod & Montreys, 1998), we hypothesize that there will be a moderate correlation between HCs’ self-reported experiences of emotion and observed ratings of their facial expressions of emotion. For the PDs in both treatment groups, a less strong, or a negligible, relationship between these variables is anticipated.

Aim 3. To examine whether there are gender differences in how men and women view facial emotional expressions of the same and opposite gender. The majority of studies in the literature have shown that women are more accurate perceivers of emotion than are men (for reviews, see Borod & Madigan, 2000; Grunwald et al., 1999). As such, we hypothesize that male and female raters may differ in how they perceive the facial emotional expressions of the male and female participants in our experimental and control groups. Further, these differences may vary depending on the gender of the facial stimuli viewed, lending further support to the existing evidence for an “in-group advantage” in emotional perception (e.g., Elfenbien & Ambady, 2002a).

Method

Participants

Two sets of study participants will be utilized in this study: Posers and Raters.

Poser participants. The poser participants consisted of 64 individuals with idiopathic Parkinson’s disease (IPD), ranging in age from 48 – 85 years old ($M = 66.2$ years, $SD = 9.1$), and
20 demographically-matched healthy adult controls ranging in age from 46 – 80 years old ($M = 64.6 \text{ years}, SD = 9.2$). Poser participants’ demographic and clinical information is summarized in Table 1.

All poser participants, or “posers,” were recruited from the Boulder and Denver, Colorado area through physician referrals, support groups, and aging centers. Approved advertisements were posted in newspapers, on email listservs, in newsletters, on websites, and in research and educational facilities. Prior permission was obtained from each recruitment site via IRB approval. Each poser was paid $10 for the initial screening phase and $30 for experimental participation. All posers were treated in accordance with the guidelines and standards set forth by the Colorado Multiple Institutional Review Board (COMIRB) at the University of Colorado, as well as the IRB at Queens College of the City University of New York.

Posers were given a series of medical, cognitive, and physiological tests to ensure that they met the overall inclusionary criteria. Exclusionary criteria included the following: severe depression (cut-off score $> 28$; measured by the Beck Depression Inventory-II; Beck, Steer, & Brown, 1996); mild, moderate, or severe dementia (cut-off score $< 24$; measured by the Mini-Mental Status Examination; Folstein, Folstein, & McHugh, 1975); symptoms of a neurological condition other than PD; substance abuse; head or neck cancer; significant history of gastrointestinal disease or surgery (as defined by an otolaryngologist); speech-language disorders unrelated to PD (as defined by a speech-language pathologist); neurological surgical treatment if it was not a surgical treatment specifically for Parkinson’s symptoms; laryngeal surgical treatment, laryngeal pathology (as defined by an otolaryngologist); participation in any intensive speech treatment within the past two years; smoked in the last four years; severe
temporomandibular joint (TMJ) disorder; or pregnancy. No participant had hearing loss unexpected for his/her chronological age (based upon audiological screening). Patients did not change medications or dosages during the speech treatment period and were neuropharmacologically stable during speech treatment period. Inclusion criteria included the following: 45-85 years; PD Stage I-IV (Hoehn & Yahr, 1967); none or mild dementia; none, mild, or moderate depression; and mild, moderate, or severe speech and voice disorder. Written informed consent was obtained from each poser participant through the Colorado IRB.

**Rater participants.** Four groups of six Caucasian rater participants (12 male and 12 female) were recruited via IRB-approved flyers at Queens College in Flushing, NY. All 24 rater participants, or “raters,” were Queens College students who ranged in age from 18-39 years old. They were recruited as study participants and were screened for inclusion/exclusion criteria (see Appendix A) using one of the screening instruments from the New York Emotion Battery (NYEB; Borod, Welkowitz, & Obler, 1992). One of the inclusionary criteria is that all raters be Caucasian. This was necessary due to the fact that the posers in the larger study were almost exclusively Caucasian due to the geographical area in which the experimental data were collected. As stated, recent literature has demonstrated an effect of ethnicity on the perception of emotional expression (i.e., the “in-group advantage;” Anthony et al., 1992; Elfenbein & Ambady, 2002a). Further inclusionary criteria for the rater participants included: being right-handed, being native-speakers of English, and having no history of neurological disorder, head injury, learning disability, psychiatric disorder, or substance abuse. Raters were compensated at the rate of $9 per hour for participation in this study. All rater participants were treated in accordance with the guidelines and standards set forth by the Queens College IRB, and written informed consent was obtained from each participant.
**Materials and Procedures**

**Poser procedures.**

*LSVTLOUD® and LSVT-ARTIC® treatment protocols.* All poser participants took part in the research protocol in the lab of Dr. Lorraine Ramig at the University of Colorado-Boulder. Poser participants with PD were randomly assigned to one of the following three groups: 1) LSVT–LOUD, 2) LSVT-Artic, or 3) PD untreated. The HC participants were demographically matched.

Those assigned to the LSVT-LOUD® (Ramig et al., 2001) group were required to attend sixteen 50-60–minute private treatment sessions in a month. Within each week, participants had to attend four consecutive days of treatment. During LSVT-LOUD treatment sessions, poser participants were instructed to sustain the vowel “ah” loudly for as long as they can, for a maximum of 12-15 times. Then the poser participants were instructed to sustain the vowel “ah” for 5 seconds in the highest and lowest pitch that they can produce, for a maximum of 12-15 times, for each pitch. The loudness was trained by the clinician and appropriate feedback was given. In each session, poser participants used their newly trained “loudness” in speaking 10 phrases repeated 5 times each. This part of the treatment took approximately 30 minutes. For the remaining 30 minutes, the clinician worked the patient through a Speech Hierarchy that started with phrases and progressed gradually to conversation. The clinician began with phrases and sequenced to sentences, then reading, and finally conversation. This was completed all while the participants were speaking loudly. Participants were cued to “be loud” for each task. Homework was assigned to help carryover the skills learned in treatment to everyday life.

The LSVT-ARTIC® (Ramig et al., 2015) treatment program was designed to parallel the
LSVT-LOUD program. The same treatment schedule (i.e., duration and number of visits) was used for the LSVT-ARTIC therapy as was for the LSVT-LOUD therapy. Poser participants were instructed to over-articulate and repeat a single phoneme such as “ta”, “pa”, or “ka.” The Iowa Performance Instrument (IOPI) was used periodically during this task to measure tongue strength and endurance. The IOPI device has a soft rubber bulb that is placed in the front of the mouth of the participant, while the poser participant says each phoneme. In accordance with the manufacturer’s instructions (i.e., Northwest Company, LLC), each bulb was disinfected in Cavicide before the first use and washed with soap and water between uses. Each participant was provided with their own new bulb; bulbs were not shared among participants. This first task of phoneme repetition continued for 10 minutes. During the next 10 minutes of treatment, the poser participants repeated and over-articulated, with high effort, contrasting phonemes such as t/k. The clinician used the word “enunciate” to cue the participant. All homework, carryover tasks, and sensory training was equivalent for both treatment groups and was taught during the treatment session. Much care and attention was placed in treating the LSVT-LOUD and the LSVT-ARTIC patients in a similar manner and in providing the same amount of positive reinforcement to each group.

**Facial expression protocol.** Posers were seated comfortably in a dental examining chair with a headrest in a sound-resistant booth (Industrial Acoustics Company [IAC]). All individuals were video-taped using a Canon XL1S mini DV video-camera. The camera was positioned approximately 9 feet away from the participant, centered, and zoomed-in to capture the face and head of each poser from the neck up. The experimenter was seated next to the video-camera in order to provide instructions and prompting, if necessary. Posers were asked to produce monologues based on procedures adapted from the NYEB (Borod et al., 1992; Montreys &
Borod, 1998). All posers were asked to recollect experiences pertaining to 3 different emotions (i.e., anger, sadness, and happiness), separately, with the order of monologue type counterbalanced among all poser participants. The neutral monologue was always produced first in order to control for any potential emotional carry-over effects that could contaminate non-emotional expression.

Each poser was given the following instructions: “We are interested in studying the kinds of emotional events that are important to people. I would like you to talk about emotional experiences involving each of three different emotions, which I will present to you one at a time, such as fear. Try to remember and actually relive a time that you felt the emotion that I ask you to talk about. I would like you to recall the time that you felt this emotion with the greatest intensity. Put yourself back into the situation and try to re-experience it with as much real feeling and intensity as when it actually occurred. Describe what happened and how you felt in detail. I am going to leave the room for a moment, let me know when you are ready, and I will come back in the room. When I return, begin to speak as soon as you feel you can vividly remember and feel the experience. Please speak for at least 90 seconds. Here is an example of how you might describe an event involving the emotion of fear.” Posers were then provided with a sample emotional monologue. If the participant was having difficulty, the examiner provided standardized probes (Borod et al., 1992; Montreys & Borod, 1998). This procedure was repeated for happy, angry, and sad emotional experiences. For control purposes, each poser was also asked to talk about a neutral, non-emotional experience (i.e., watching television). For the neutral monologue, the poser participants were given the following instructions: “We are interested in studying everyday activities, in other words, things that you do regularly. I would like you to talk for a few minutes about an activity, which I will present to you, such as watching television. Try
to remember and reconstruct the last time that something happened related to the topic that I ask you to talk about. Put yourself back into the situation and try to reconstruct it with as much accuracy and precision as when it actually occurred. Describe the event in detail. Please begin to speak as soon as you can remember what happened. Try to speak for at least 90 seconds. Please make sure that the situation you describe is not an emotional one. Here is an example of how you might describe an event involving watching TV.” The posers were then provided with a sample narrative to help them. Again, standard probes were used by the examiner, if needed.

After completing each emotion monologue, participants were asked to report their subjective experience of emotion during the monologue on a 7-point Likert scale, from “1” (minimal) to “7” (maximal). The experiential ratings were not collected for the neutral monologues. Using procedures developed by Borod et al. (2008), participants were first instructed to report the intensity of their feelings immediately following the monologue with the prompt “On a scale of 1-7, please indicate how happy, sad, or angry you feel right now” (i.e., immediate experience). Next, accuracy was assessed with the prompt: “How accurately were you able to express the specific emotion that you experienced during the situation which you just described” (i.e., accuracy of experience). Finally, intensity of their emotional experience, during the monologue, was assessed via the prompt: “Please indicate how intensely you relived the experience and felt the emotion that you were talking about just now” (i.e., intensity of feelings).

These data were recorded at the University of Colorado, Boulder and were then sent on DVD to Dr. Joan C. Borod’s Emotion Lab, at Queens College, for processing and analysis.

**Rater procedures.** The experimental data (i.e., the poser monologues) were split into approximately 8-12 fifteen-second segments (depending on the original length of the monologue),
for each of the four monologue types. This resulted in a total of approximately 6,000 video segments across the 84 posers. Using an in-house video-stimulus presentation software (programmed in Microsoft Access v. 2007), 24 raters (12 female and 12 male) were presented with the experimental poser data on a 17-inch flat panel LCD computer monitor. Each poser-video segment was 3.75” x 3.5”. In order to control for order effects (i.e., poser group, emotion type, and poser gender), all segments were completely randomized and presented to the raters individually. Segments were viewed without audio so that the raters were not aware of which emotion they were viewing. Further, the raters were naïve to the study’s hypotheses and subject-group characteristics. The raters were asked to evaluate each video segment in accordance with procedures developed by Dr. Borod and colleagues (Borod et al., 2007; Canino, Borod, Madigan, Tabert, & Schmidt, 1999). The rater participants worked individually, in a quiet room, at a Dell Desktop computer. They were asked to evaluate the facial expressivity in each 15-second video segment. The raters evaluated each video segment using a 7-point Likert scale (from “1” [minimal] to “7” [maximal]), in terms of facial movement (i.e., facial mobility), emotional expressivity (i.e., emotional variability, emotional frequency, and emotional intensity), and social engagement. Each facial variable (e.g., emotional expressivity or emotional frequency) was rated individually until all video segments were completed. Using the computer mouse, raters viewed each video segment, entered their value from 1-7, and advanced to the next video segment by clicking “play next.” All raters were able to watch the video segment up to three times, if needed, to make their rating.

A set of approximately 1,500 video segments were rated, separately, in terms of the five facial variables of interest (i.e., Facial Mobility [FM], Emotional Variability [EV], Emotional Frequency [EF], Emotional Intensity [EI], and Social Engagement [SE]). Each rater participant
was extensively trained on how to rate each variable in group sessions, and all raters carried out their ratings for each corresponding variable, individually, with no single rating session exceeding 3 hours. Multiple rating sessions were required in order to complete all of the ratings for a single variable (i.e., approximately two weeks). Once the ratings for the first variable were completed, the rater participants were then trained on the next variable, and those rating sessions began. The variables were trained and rated in the following order: FM, EV, EF, EI, and SE. Once the raters completed the training and rating procedures for all five variables, six new raters were recruited to undergo the exact same process as described above. This was done four times (i.e., four groups of raters), due to the amount of video data that needed to be evaluated. If we were to use one group of participant-raters to rate all of the data, we feared we would lose raters due to attrition. It would have been too large of a task for any 6 rater participants.

**Rater cohorts.** Due to the large volume of poser-participant video data, we chose to use four groups of six raters (3 male, 3 female) to evaluate the poser-participant data. The four groups of raters were comparable in terms of ethnicity (i.e., all Caucasian), age, and years of education. Each rater cohort evaluated a subset of poser-participant data. All 6 raters rated pre- and post-video data, PD patients (i.e., LSVT-LOUD; LSVT-ARTIC, and PDuntrx), healthy controls, and all monologue emotions (i.e., happy, angry, sad, and neutral). Rater Cohort 1 was established first. When the rater participants completed their ratings for all variables (i.e., FM, EF, EV, EI, and SM), a new cohort of raters was recruited and formed. Each of the Rater Cohorts 1-4 rated the variables in the order of FM, EF, EV, EI, and SE.

**Rater training procedures.** For each variable, all raters participated in an extensive group training session. The training sessions were run by an advanced doctoral student (i.e.,
training session leader) in Dr. Borod’s Emotion Laboratory. First, the training session leader defined relevant terms and operationalized the variable on which they were to be trained, such as “Facial Mobility.” Second, the 7-point Likert scale that was used to make the actual ratings was reviewed and explained to the raters. Third, the training session leader described all of the different facial features of a face and how movement of such features (e.g., wrinkling of the forehead, widening of eyes, pursing of lips, etc.) should be attended to with respect to each facial rating, disregarding any extraneous head movements, tremors, or excessive blinking. These descriptions were based on the literature regarding prototypical characteristics of facial expression (e.g., Ekman & Friesen, 1975; Izard 1983; Scherer, 1989) and were developed specifically for this project.

Exemplar presentation. For the next phase of training, raters were shown two 15-second exemplar video segments (i.e., one male and one female) which clearly represented each point on the scale (i.e., what a “1” would look like, what a “2” would look like, etc.), totaling 14 exemplars. Superlab v. 4.5 Pro (Cedrus Corporation) was used for all video stimulus presentation during variable training. The exemplar video segments and subsequent poser training materials consisted of digitized VHS data, of both PD and HC subjects, from previous pilot work by Borod and colleagues (Borod et al., 2007). The video exemplars used for training were previously evaluated by three judges, and only where there was very high agreement among the judges, was an exemplar to be used for training. During exemplar presentation, each segment was shown without audio, and the training session leader explained each segment in terms of the characteristics that qualified it as being an example of a 1, 2, 3, etc. on the Likert Scale. Two exemplars for each value on the Likert scale (1-7) were shown to the raters.
**Conferencing.** After all exemplars were presented to the raters, a “Conferencing Stage” took place. The training session leader presented 12 video segments that allowed for practice, ratings, discussion, and review. These video segments were presented in a randomized order with respect to gender, group, and emotion. Raters were asked to write down their rating on a sheet provided. The ratings were then spoken aloud and shared with the group, so that the raters were able to see how their ratings compared to the ratings of the other 5 rater participants. If all of the individual ratings were consistent with one another (i.e., not differing by more than 2 points), the next poser video segment was presented. If discrepant ratings occurred (i.e., ratings that differ by greater than 2 points), a discussion was held among the group and opinions and thoughts were discussed. The descriptions and instructions provided at the start of the training session were reinforced, and the experimenter focused the discussion on rating techniques that would encourage group agreement and consensus. Once the discussion was over, raters were given the opportunity to watch the video segment again and re-rate it, as the discussion may have led them to a different evaluation of the video segment. The experimenter recorded all of the raters’ original ratings and any ratings that changed after discussion. This process continued for each of the 12 conferencing video segments.

**Practice ratings.** In the last stage of training, forty practice poser video segments were presented to the raters. The raters were instructed to rate the segments individually and to write down their responses on the sheet provided. No feedback or discussion was given during this stage of training. After all ratings were complete, the training session leader calculated the average one-way random Intra-Class Correlations (ICC) for the 40 practice items. This was done to ensure sufficient internal consistency among the raters. Based on standards used in other studies, we determined that group consensus would be considered sufficient at a minimum of 80%
agreement among raters’ responses (ICC ≥ .80). If the criterion was not met, additional training was continued until sufficient consensus was reached.

**Pre-Data Processing**

**Poser video cleaning and segmentation.** All poser monologue videos, as originally sent from Colorado, were first inspected for any extraneous talking by or to the experimenter, long pauses without speech, and any additional extraneous video interruption. These parts of the taped monologue sessions were removed using Corel Video Studio ProX 3 via a process we termed “cleaning.” The remaining monologue were then split into 15-second segments for all participants and emotions. These “cleaned” and “segmented” video segments were then used as the poser experimental data set.

**CHAPTER III.**

**Results**

**Statistics**

A significance level of $p = .05$ was used for all statistical tests. The Bonferroni procedure was used to correct for multiple comparisons for all main effects (i.e., where there were greater than 2 levels) and interactions.

**Preliminary Analyses**

**Evaluation of the distributions.** The 4 poser groups (i.e., LSVT-LOUD, LSVT-ARTIC, HC, and PD-untrx) were first inspected for normality using the Kolmogorov-Smirnov statistic with Lilleifors significance level (i.e., $p < .05$). All groups were normally distributed ($ps < .05$).
Bivariate normality was assessed between group and monologue emotion using the Kolmogorov-Smirnov test. The distributions for the 4 poser groups (i.e., LSVT-LOUD, LSVT-ARTIC, HCs, and PD-untrtx) for each of the 4 monologue types (i.e., Happy, Angry, Neutral, and Sad) were normally distributed, except for the Sad monologue for the LSVT-ARTIC group. The overall percent of normally distributed bivariate data was 15/16 distributions, or 94%. All observations in the sample for all combinations of variables were analyzed for multivariate normality (Stevens, 1992). The data (i.e., 5 facial variables x 4 monologue types x 2 time points = 40 distributions) for each of the 4 treatment groups (i.e., 160 distributions) were analyzed for normality using the Kolmogorov-Smirnov test. The percentage of normally-distributed distributions for each treatment group were as follows: LSVT-LOUD = 88%, LSVT-ARTIC = 75%, PD-untrtx = 93%, and HCs = 78%.

The data (i.e., 5 facial variables x 4 monologue types x 2 time-points) for each of the 4 poser groups were then examined for multivariate homoscedasticity using Levene’s test. Eighty-eight percent of the distributions (i.e., 140/160) demonstrated homogeneity of variance (i.e., $p > .05$). For these distributions, all normal probability plots (i.e., Q-Q plots) were visually inspected for variability and outliers (Mertler & Vannatta, 2005).

Taking all three of these analyses into consideration, we felt very comfortable proceeding with parametric statistical procedures as the Analysis of Variance (ANOVA) procedure is robust to violations of homoscedasticity (e.g., Lix & Keselman, 1998; Mertler & Vannatta, 2005; Ramsey, 1994).

**Poser group characteristics.** Posers were stratified and matched across the 3 experimental groups (i.e., LSVT-LOUD, LSVT-ARTIC, and PD-untreated) on the factors of age,
education, stage of disease, and gender. Posers in the HC group were carefully matched to the 3 experimental groups by the same factors. Demographic and clinical information for all four poser groups can be seen in Table 1. A one-way ANOVA was performed for each of the demographic factors. The analyses revealed that there were no statistically significant differences among the four poser groups for age, $F(3, 76) = 1.21; p = .32$, and education, $F(3, 76) = 1.76; p = .162$.

Further, there were no statistically significant differences among the three PD groups for stage of disease, according to the Hoehn and Yahr scale, $F(2, 59) = .248; p = .781$, and time since diagnosis, $F(2, 59) = .021; p = .97$. Finally, the Chi-square test revealed that there were no statistically significant differences between the PD and HC groups for gender, $X^2 (2) = .321, p > .571$.

**Rater group characteristics.** Four groups of 6 raters (3 male and 3 female) were selected to rate the poser facial expressions. The mean age for the 12 male raters was 21.7 years and 21.7 years for the 12 female raters. The average years of education for the male and female raters was 14.3 and 14.7, respectively. Rater group demographic characteristics (i.e., gender, age, and education) are summarized in Table 2.

**Inter-rater reliability.** Four groups of 6 raters ($N = 24$) were trained to rate each of the 5 facial expressivity variables: Facial Mobility (FM), Emotional Frequency (EF), Emotional Intensity (EI), Emotional Variability (EV), and Social Engagement (SE). For each group of four raters, we analyzed the group’s average One-way Random Intra-Class Correlations (ICCs; Shrout & Fleiss, 1979) for the 12 conferencing ratings and for the 40 practice ratings (see Methods section for details). The ICCs can be seen in Table 3. The inter-rater agreement for each training variable needed to meet or exceed 80% (i.e., ICC ≥ .80) in order to proceed to the experimental
ratings. The correlations showed that agreement among the raters was high for all variables. All correlations for conferencing and practice ratings were ≥ .85. The conferencing ICCs were always higher than the practice ratings for each variable. This is to be expected because the conferencing ratings were performed aloud as a group. When raters rated the practice items, individually, consistency was not as high.

Group Comparisons

Statistical analyses by aim.

Aim 1. Determine whether the Lee Silverman Voice Treatment (LSVT-LOUD) affects facial emotional expression in individuals with Parkinson’s disease (PD), during the recollection of past emotional experiences, as compared to 3 demographically-matched groups: Untreated PDs, HCs, and a PD group receiving an equivalent treatment. In order to examine the effect of Treatment (i.e., LSVT-ARTIC, LSVT-LOUD, PD-untrx, and HC) on facial emotional expression, we performed a 4 x 4 x 2 (Group [LSVT-LOUD, LSVT-ARTIC, PD-untrx, and HC] x Monologue Type [Angry, Sad, Happy, and Neutral] x Time [Pre and Post]) mixed-model ANOVA, separately, for each of the five facial variables (i.e., FM, EV, EF, EI, and SE).

We expected to find an interaction between Group and Time, demonstrating that the LSVT-LOUD group improved post-treatment compared to the 3 control groups. We did not find a significant Group x Time (ps > .05) or Group x Time x Monologue Emotion (ps > .05) interaction for any of the 5 facial variables. These results indicate that there was no significant change in the facial expressivity of PDs after being treated with LSVT-LOUD. However, as expected, the facial expressivity of the LSVT-ARTIC and the untreated groups (untreated PDs and HCs) did not change over time.
The results of the ANOVAs indicated that there were no significant main effects for timepoint \((p < .05)\); there were no significant differences between Time 1 (Pre) and Time 2 (Post) for any of the 5 facial expressivity variables.

Significant main effects were found for Group for EF, \(F(3,72) = 4.4, p = .007\); SE, \(F(3, 72) = 4.17, p = .009\); and FM, \(F(3, 72) = 3.13, p = .031\). For the measures of EF and SE, the HC group was rated as having greater emotional expressivity and social engagement compared to the 3 PD groups. For FM, LSVT-ARTIC was the only PD group that was significantly different from the HC group. However, the average facial ratings for LSVT-LOUD and PD-untrx were lower than the HC group although they did not reach significance. These results are summarized in Figure 1. Further, although not significant, a trend towards a main effect for Group was found for EI, \(F(3, 72) = 2.54, p = .06\). A comparison of the group means demonstrated the same pattern as above. As expected, none of the facial ratings for the PD groups (LSVT-LOUD, LSVT-ARTIC, or PD-untrx) were significantly different from one another for any of the 5 facial expressivity variables.

Significant within-subject main effects were found for Emotion for each of the 5 facial expressivity measures, FM, \(F(3, 70) = 11.51, p = .000\); EI, \(F(3, 70) = 7.46, p = .000\); EF, \(F(3, 70) = 7.94, p = .000\); EV, \(F(3, 70) = 11.64, p = .000\); and SE, \(F(3, 70) = 23.1, p = .000\). The pairwise comparisons for the main effects of Emotion are summarized in Figure 2. Overall, for each measure, the order from highest to lowest ratings was Happy, Angry, Sad, and Neutral. In terms of significance, posers’ Happy monologues were rated significantly higher than posers’ Neutral or Sad monologues for each of the 5 facial expressivity variables. For EI, EF, and SE, posers’ Happy monologues were rated significantly higher than Angry monologues. Angry monologues were
rated as having significantly greater emotional expressivity (i.e., EF, EV, and EI) than the Neutral monologues; however, the reverse was found for FM and SE, with neutral monologues rated significantly higher than Angry monologues. Further, Angry monologues were rated as having significantly greater EV and SE as compared to Sad monologues. Finally, for SE, Neutral monologues were rated significantly higher than Sad monologues.

**Aim 2. Explore whether the emotional intensity of one’s facial expression relates to one’s experience of emotion (i.e., how intensely participants *experience* emotion) during the production of monologues recalling past emotional experiences.** Does the relationship between emotional experience and expression differ between PDs and HCs? We performed Pearson Product-Moment Correlations, separately, for the PD and HC groups, for the facial expressivity ratings of emotional intensity (EI) versus each of the three self-reported emotional experience measures: 1) immediate experience (i.e., how subjects felt, emotionally, immediately after talking about an emotional event [i.e., “the monologue”]), 2) accuracy of experience (i.e., how accurately they felt they expressed the emotion during each monologue), and 3) intensity of experience (i.e., how intensely they felt the emotion during the monologue). These correlations were performed on the ratings of Happy, Angry, and Sad monologues, separately, as well as for pre and post ratings. If a correlation was significant, we performed the Fisher r-to-z transformation (Chapman & Chapman, 1998) to determine if one group (i.e., PDs or HCs) had stronger correlational effects than the other. The results of all of the correlational analyses are presented below and summarized in Table 4.

**PD group correlations.**
For the PD group, ratings of facial emotional intensity were positively correlated to the experience of each emotion, both at Time 1 (Pre) and Time 2 (Post). Only 6/18 correlations were significant; however, out of the 6 possible correlations for the Sad emotion, 5/6 were significant (see Table 5 for a summary of results). The correlations between the expression of sadness and the experience of sadness were significantly correlated ($r = .40, r = .47, r = .24, r = .29, r = .22; ns., r = .19$). The emotional intensity of posers’ faces, while producing a Sad monologue, was positively correlated to their feelings of emotional intensity during the monologues and to how they felt immediately after the monologues. This pattern of results is seen at Time 1 (Pre) and Time 2 (Post), although the correlations are not as strong at Time 2. Facial expressions of sadness were less related to perceived accuracy in producing the emotion. The correlation between facial EI for sadness and self-assessed accuracy was only significant at Time 2 (i.e., $r = .29$) as opposed to Time 1 (i.e., $r = .19$).

Only 1/6 total possible correlations was significant between the experience of happiness and facial expressivity for happiness. For Time 1, there was a significant positive correlation between the expression of happiness and perceived accuracy in displaying happiness ($r = .23$). This finding is difficult to interpret given the overall findings of 5/6 non-significant correlations for the happy monologue and that the same relationship was not present at Time 2. The positive correlations between the experience of Anger and the facial expressivity for Anger did not reach significance.

**Healthy-Control correlations.**

For the Healthy Control group, 12/18 of the correlations were positive. Three significant inverse correlations (i.e., $r = -.51, r = -.58, r = -.68$) were found between the expression of Anger
and the experience of Anger (i.e., Intensity, Accuracy, and feelings “Immediately” following the monologue) for Time 1. These findings suggest that, for Angry monologues, greater demonstration of facial EI was related to a reduction in the experience of Anger intensity. Conversely, and more likely, these findings could also suggest that greater experiences of Anger were related to reductions in facial EI. It is unclear as to why Time 2 did not show a similar correlational pattern (see Table 5 for a summary of the results). One inverse correlation ($r = -.03$) was found between the expression of happiness and the experience of happiness, and one was found between the experience of happiness and perceived accuracy of happiness ($r = -.27$). Since neither of these correlations reached significance, these results cannot be explained in a meaningful way. Finally, none of the remaining positive correlations for the Happy or Sad condition reached significance.

**Correlation comparison between groups.**

In order to examine if there were stronger correlational effects between the PD and HC groups, the Fisher $r$-to-$z$ transformation (Chapman & Chapman, 1998) was used to test the significance between the group correlations. A summary of these analyses can be seen in Table 6. The results indicate that the correlations for the HC group were significantly greater for the Angry emotion at Time 1 than were those for the PD group. Specifically, we found a significant difference in the relationship between the facial expressivity of Anger and experienced EI of Anger ($p = .02$), perceived accuracy of Anger ($p = .01$), and experience of Anger immediately following the monologue ($p = .00$). Likely driving these results are the significant inverse correlations found between the experience of Anger and facial expression of Anger, for Time 1,
for HCs. These findings indicate that there is a significant difference in the relationship between experienced and expressed Anger between PDs and HCs.

**Group differences in emotional experience.**

We also examined whether there were differences in emotional experience between PDs and HCs. A 2 x 3 x 2 (Group [PD and HC] x Monologue Emotion [Angry, Happy, and Sad]) x Gender [Male and Female]) mixed-model ANOVA was performed for each of the three emotional experience measures (i.e., “Intensity” of Experience, “Accuracy,” and Experience “Immediately” Following). Even though the literature (e.g., McCabe et al., 2010) typically does not find differences in emotional experience between PDs and HCs, we examined this in our own sample of PD and HC participants. The results from these ANOVAs are summarized in Table 7.

As expected, we did not find a main effect of Group (ps > .05) or any interaction between Group and Emotion for any of the 3 experience variables. These findings are comparable with previous research (e.g., McCabe et al., 2010) that suggests that PDs and HCs experience emotion similarly. However, we did find a main effect for Emotion for Intensity, $F(2, 66) = 8.67, p = .000$, Immediate, $F(2, 66) = 14.2, p = .000$, and Accuracy, $F(2, 66) = 3.21, p = .047$. Pairwise comparisons indicate that Sad emotions resulted in significantly higher experienced EI, perceived accuracy, and greater feelings immediately following the monologue compared to Angry emotions. Posers reported feeling significantly greater intensity for Sad monologues than Happy monologues. Immediately following the monologue, posers felt significantly more emotion after Happy monologues than Angry monologues. The group means can be seen in Figure 3.
Aim 3. Examine whether there are gender differences in how men and women view facial emotional expressions of the same and opposite gender. We conducted a 2 x 2 x 2 x 4 (Rater Gender [Male and Female] x Poser Gender [Male and Female] x Time [Pre and Post] x Monologue Type [Angry, Happy, Sad, and Neutral]) mixed-model ANOVA for each of the five facial variables (i.e., FM, EV, EF, EI, and SE). The Bonferroni procedure was used to correct for multiple comparisons for all main effects and interactions. See Table 8 for a summary of results.

The results of the gender analyses revealed significant main effects for Rater Gender for EF, \( F(1, 74) = 5.90, p = .018 \); EV, \( F(1, 74) = 56.1, p = .000 \); and EI, \( F(1, 74) = 8.64, p = .004 \) (see Figure 4). Men rated posers as having significantly greater emotional expressivity (EF, EV, and EI) than did the female raters. However, women rated the posers as having greater Social Engagement compared to male raters [SE, \( F(1, 74) = 18.7, p = .000 \)]. For the control variable of FM, both genders were rated as having equivalent amounts of FM (\( p > .05 \)). Further, we found an interaction between poser gender and rater gender for SE, \( F(1, 74) = 10.6, p = .002 \); see Figure 4. Although both male and female raters judged female posers to be more facially expressive than male posers, female posers were rated as significantly more expressive than men by female raters than by male raters (see Figure 6). For EV, we found a 3-way interaction among poser gender, rater gender, and emotion (Figure 14), \( F(3, 74) = 3.55, p = .027 \). However, after the Bonferroni correction, the effect did not reach significance (\( ps > .05 \)). We also discovered a 4-way interaction among poser gender, rater gender, emotion, and time, for EF, \( F(3, 74) = 3.74, p = .012 \), and FM, \( F(3, 74) = 6.09, p = .001 \). We did not make any predictions regarding the interaction of time and valence. However, in an attempt to explore these findings, we visualized the data by creating Figures 5a & 5b and 6a & 6b. Unfortunately, the figures did not yield any meaningful results. However, the interactions show that female posers, as compared to male
posers, were rated as having greater EF and FM for Happy Monologues; of note, this effect was greatest when rated by female raters at Time 1.

For all of the facial expressivity variables of EF, EV, EI, SE, and FM, we found an interaction between poser gender and emotion ($p$s $>$ .05). After pairwise comparisons and a visual inspection of the data (see Figures 7-11), it is clear that female posers were rated higher than male posers, in general. However, when the ratings were broken down by monologue emotion, there were some emotions where male and female posers did not differ significantly in terms of how they were rated. This was especially true for the Sad monologues. For instance, for EF, female posers were rated as significantly more expressive than male posers during Happy, Angry, and Neutral monologues but not for the Sad monologues. These findings, however, do not represent true interactions between poser gender and emotion, rather, they are demonstrative of regression towards the mean and greater variability among the female posers’ ratings.

We found a significant main effect for Poser Gender for each of the 5 facial expressivity variables: EF, $F(1, 74) = 21.9, p = .000$; EV, $F(1, 74) = 22.1, p = .000$; EI, $F(1, 74) = 24.7, p = .000$; SE, $F(1, 74) = 6.39, p = .014$; and FM, $F(1, 74) = 20.9, p = .000$. For each of the facial expressivity variables, female posers were rated as having significantly more facial emotional expressivity and facial mobility as compared to male posers. The group means can be seen in Figure 12.

There was a significant main effect of emotion for EF, $F(3, 74) = 11.6, p = .000$; EV, $F(3, 74) = 11.7, p = .000$; EI, $F(3, 74) = 8.27, p = .000$; SE, $F(3, 74) = 20.53, p = .000$; and FM, $F(3, 74) = 14.5, p = .000$. These results are summarized in Figure 13. Posers were rated as significantly more facially expressive during Happy monologues compared to Sad ($p < .05$), Angry ($p < .05$), and Neutral monologues ($p < .05$) for EV, EI, SE, and FM ($p$s $< .001$). For EF,
EI, and FM, posers were rated as significantly more facially expressive during Sad monologues as compared to Neutral monologues ($p < .05$). For EV, EI, and FM, Angry monologues produced significantly more facial expressivity than Neutral monologues ($p < .05$).

We did not find a main effect for time ($p > .05$) or any two-way interactions for time x poser gender ($ps > .05$), time x rater gender ($ps > .05$), or time x emotion ($ps > .05$) for any of the facial expressivity variables.

CHAPTER IV

Discussion

Through various configurations of facial muscles, individuals convey a wealth of emotional information. Parkinson’s disease is one of few illnesses that is marked by the decreased ability to communicate emotions facially. The research conducted in this project examines the ways in which the facial communication channel is compromised in PD and explores the potential benefit of LSVT-LOUD in remediating hypomimia.

Summary of Findings

LSVT-LOUD treatment effects.

Contrary to our predictions for Aim 1, we did not find an effect of LSVT-LOUD treatment on PDs’ facial emotional expressions. There could be some reasons for this. In 2003, Spielman and colleagues first reported the preliminary data and results they obtained from treating 22 PD patients with LSVT-LOUD. The PD patients who received LSVT-LOUD were rated as having greater facial mobility and higher levels of social engagement than a PD group receiving respiratory treatment (Spielman et al., 2003). It is important to point out the differences in facial expression elicitation between the Spielman et al. study and the current study. In the Spielman et
al. (2003) study, PD patients were asked to “talk about anything you want for the next 20-30 seconds.” This resulted in self-generated speech and facial expressions that were not necessarily emotional. Further, the content of their monologue could have been in the future, present, or past. The methods utilized in the current study asked PD patients to recall an emotional event that happened to them previously and to talk about it. Our task involved retrieval of an autobiographical event, holding the retrieved memory in the attention window (Jacobs et al., 1995), reconstruction of stored internal representations (Dudai, 2002), sequencing, recruitment of limbic regions, and reporting what happened. Further, evidence suggests that the recollection of a visual scene results in the activity of visual cortices (Wheeler, Petersen, & Buckner, 2000). In sum, the task used in the current study was neurocognitively demanding, requiring the activation and integration of numerous brain regions. It is plausible that our task was more challenging than the task from the Spielman et al. (2003) study and, thus, less amenable to the therapeutic effects of LSVT-LOUD.

Posed and spontaneous facial emotional expressions rely on relatively distinct neuroanatomic pathways (e.g., Borod, Haywood, & Koff, 1997) and, therefore, can be affected in PD differently. Spontaneous facial expressions are controlled by subcortical regions, whereas voluntary/posed facial expressions are controlled by cortical areas in the frontal lobe (e.g., Blair, 2003; Borod & Koff, 1984). It has been postulated that the facial expressivity benefits gained from LSVT-LOUD might be due to improved motoric functioning, therefore, spontaneous facial expressions, not posed, would improve after LSVT-LOUD. The improvements in spontaneous expressions during conversational speech seen in Spielman et al. (2003) support this theory. However, does the current task of recalling a previously experienced emotional event result in spontaneous expression? The task is performed spontaneously and extemporaneously, however,
it required greater neurocognitive recruitment than Spielman’s (2003) task. In a compromised population, such as PD, it’s plausible that our emotional monologue task required greater cognitive effort (i.e., increased neurocognitive recruitment) than it did for the HCs. Increased cognitive activity has been shown to reduce emotional processing (Blair, Smith, Mitchell, Morton, Vythilingam, et al., 2007). As such, the cognitive demands of the emotional monologue task may have resulted in an overall reduction in emotion processing for the PD group as compared to other emotional tasks.

Although not an aim of the current study, our results indicate that the PDs in our sample demonstrated impaired facial expressivity relative to HCs. Emotional Frequency and Social Engagement were significantly reduced in the PD groups compared to HCs. Despite seeing reductions in all PD groups, only the LSVT-ARTIC group was significantly decreased on the measure of Facial Mobility. Similarly, all three PD groups demonstrated a reduction in Emotional Intensity compared to the HCs; however, the difference did not reach significance.

**Effect of Monologue Emotion.**

Although we did not have specific predictions about how different emotions would be perceived by raters, we found that for all of the facial expressivity variables, Happy monologues were rated significantly higher than Sad monologues across all participant groups.

In general, there was a pattern of Happy > Angry > Sad > Neutral, with the exception of SE, where Neutral > Sad. Generally, these findings legitimized the Neutral condition as our non-emotional control variable. Using a Neutral condition as a non-emotional control, we expected

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1 Not all pairwise comparisons reached statistical significance after the Bonferonni correction.
that ratings of facial emotional expressivity would be lower for neutral monologues compared to the emotional monologues, and this was the case.

As described in the previous paragraph, there was an overall pattern of facial emotional expressivity such that posers were rated as more expressive as a function of monologue type as follows: Happy > Sad > Angry > Neutral. These results are consistent with the preponderance of findings in the literature regarding emotional valence, such that positive facial expressions of emotion are judged as more expressive than are negative expressions (e.g., Borod et al., 2004). These results may be best understood in terms of positivity theory and socioemotional selectivity theory. According to positivity theory, older adults are driven to reduce the amount of energy and time spent on negative events, particularly unpleasant social encounters (Carstensen, Fung, & Charles, 2003). According to socioemotional selectivity theory, when reliving negative emotional events from the past, older adults report less negative emotion and greater positivity than younger adults (Charles & Carstensen, 2008). The emotional expressions of the posers in our study are in line with these findings, such that older adults were more expressive for positive emotions (e.g., Happy) than negative emotions (e.g., Sad and Angry). As expected, the non-emotional control condition of Neutral resulted in the least amount of facial expressiveness.

**Group differences: The relationship between experienced and expressed emotion.**

Since evidence suggests that PDs experience emotion in a similar way as HCs (e.g., Mikos et al., 2009), yet have emotional expression deficits, we hypothesized that the correlation between experienced and expressed emotion would be lower for the PDs than for the HCs. However, we did not see a pattern in the correlation matrix to support this hypothesis.

For the PD group, the correlations between the expression of sadness and the experience of sadness were significant. The emotional intensity of posers’ faces, while producing a Sad
monologue, was positively correlated with their feelings of Emotional Intensity during the monologues and how they felt Immediately following the monologue. Facial expressions of sadness were less related to Accuracy of experience, since only 1 of the 2 correlations was significant.

The literature suggests that PD patients with hypomimia can be perceived as unhappy and depressed (e.g., Pentland, Gray, Riddle, & Pitcairn, 1988). It is possible that raters perceived PD posers in our sample as appearing depressed or unhappy. This could have led to higher correlations between the experience/expression of sadness compared to the other emotions. All posers in this sample were screened using the BDI, and any individual meeting the criterion for severe depression was excluded from participation. However, it is unknown if the HCs and PDs differed in terms of depression. As such, we ran follow-up analyses comparing BDI scores between PDs and HCs using the independent samples t-test. We found that the PDs and HCs differed significantly for the BDI (PDs [\(\bar{x} = 8.8\)] and HCs [\(\bar{x} = 2.6\)]), \(t(1, 78) = 4.47, p < .001\) (see Figure 15). It has been previously reported that depressed patients have a difficult time expressing positive facial emotions (Jaeger, Borod, & Peselow, 1984) and display more intense sad emotions (Mergl et al., 2005). It is possible that the higher BDI scores represent sub-clinical depression in the PD group and are moderating the higher correlations between the expression and experience for sadness.

We expected to see a moderate/high correlation between the experience of emotion and the expression of emotion in the HC group. We found, mostly, low/moderate positive correlations that were not statistically significant for the Happy and Sad monologues. However, at Time 1, there was a significant inverse correlation between the expression/experience of anger for the HC group. When comparing the correlations between the HC and PD groups, we found a
significant difference between the two group’s correlations for Angry monologues, such that the correlations for the HCs were lower than the correlations for the PDs. Research by Comblain, D’Argembeau, and Van der Linden (2005) found that older adults tended to reappraise negative events in a more positive light than did younger adults. Since our population of posers was made up of older individuals, it is possible that our HC group also reappraised negative events more positively. Alternately, the HC group could have been subconsciously acquiescing to social display rules. According to Ekman and Friesen (1969), social display rules are the “learned, culture-specific rules governing the management and control of emotional expression depending on social circumstances.” Buck et al. (1992) found that the expression of negative emotions is inhibited or reduced when in the presence of an unfamiliar person. It is plausible that the HCs were experiencing intense anger, but due to the presence of an unfamiliar experimenter in the room, HCs reflexively masked their facial expressions of anger. In support of this theory, we did not find the same pattern of correlations for Anger at Time 2. It is possible that the posers were more familiar and comfortable with the experimenter at Time 2, thus, attenuating the effect of the presence of an unfamiliar person. Further, it makes sense that PD patients did not demonstrate the same adherence to display rules as HCs. Individuals with PD have nonverbal communication deficits. In the presence of others, PD patients’ facial expressions are masked; therefore, facial and verbal feedback from others are perceived and interpreted differently, if not more negatively. Oftentimes, PD patients feel excluded from conversation, leading to feelings of loneliness and isolation (Miller et al., 2006). Due to these feelings of social isolation, even in the presence of others, it is possible that individuals with PD do not interpret the social scenario as one that requires emotional monitoring or social display rules. Further, research suggests that patients with PD cannot interpret how others feel when compared to healthy age-matched controls. PD
patients have demonstrated a decreased ability to accurately perceive *theory of mind* tasks when compared to young and elderly controls (e.g., Saltzman, Strauss, Hunter, & Archibald, 2000). Taken together, evidence suggests that PD patients may not be as susceptible to the effects of social display rules as HCs. Therefore, this could explain the discrepant facial expressivity findings for HCs when producing angry monologues.

We compared the PD and HC groups on the 3 measures of emotional experience: “Emotional Intensity” felt during the monologue, their feelings “Immediately Following” the monologue, and the extent to which they felt they produced the emotion “Accurately.” As hypothesized, there were no significant differences between the HCs and PDs for evaluations of emotional experience; both groups experienced the monologue emotions similarly.

We did, however, find a main effect of emotion. Posers experienced anger to a lesser degree in Emotional Intensity and to a lesser degree Immediately Following the monologue compared to their experience for Sad monologues. Also, Immediately Following the Angry monologues, posers experienced a lesser degree of emotion than after happy monologues. It is possible that posers found it more difficult in becoming angry as compared to other emotions. Again, this can be best understood in terms of social display rules. Posers may have subconsciously felt it socially inappropriate to display anger in the presence of the experimenter. As such, they had greater difficulty in feeling angry from a past event. The differences in the degree to which varying emotions are relived is an area for further research.

**Gender analyses.**

One of the major aims of this study was to examine how facial masking in PD is evaluated by others and how these judgments differ based on the gender of the individual and the gender of the observer. We expected that raters would evaluate posers of the same gender as more
expressive compared to opposite-gendered posers; this prediction was partially substantiated. Our gender analyses revealed that for each of the facial emotional expressivity variables (i.e., EF, EV, and EI), female posers were rated as having significantly more facial expressivity and Facial Mobility as compared to male posers. This finding corroborates the results from previous research that has found that women are more facially expressive than men (e.g., Kring & Gordon, 1998). Although all raters judged female posers to be more facially expressive, as expected, female posers were rated as significantly more expressive than male posers by only female raters. Given that women are better at detecting facial emotions (e.g., Montagne, Kessels, Frigerio, de Haan, & Perrett, 2005) and that they are more facially expressive (e.g., Brody & Hall, 1993; Kring & Gordon, 1998) than are men, it is not surprising that we found that female posers were rated as significantly more facially expressive by female raters.

Our gender analyses revealed that men and women rate facial expressions differently. For the facial expressivity measures of Emotional Frequency, Emotional Variability, and Emotional Intensity, male raters rated posers as more facially expressive than did female raters. This is a surprising finding. Given that men are less sensitive to emotional facial expressions (e.g., Montagne et al., 2005), we would expect that if any rater-gender effect occurred, it would be that women rated posers as being more expressive. We did, however, find that female raters rated posers as being more socially engaged than did the male raters. Interestingly, male and female raters rated all of the emotional expressivity (i.e., EF, EV, EI) variables differently, however, they rated measures of Facial Mobility similarly for both male and female posers. This lends further support for the use of Facial Mobility as a non-emotional control.
Limitations

One limitation of the present study was that the facial expressions of the posers yielded a small range of rated values. Very few posers were rated as 6s or 7s. One reason for this could be due to the demographic region from which these participants were recruited. As stated in the Methods section, all poser participants were recruited from the Colorado area. It is plausible that variations in emotional expressivity exist among regions in the United States, such that Midwesterners are more likely to dampen negative expressions of emotion in comparison to those in the NY Metro area. Further, those living in NY Metro areas may be less frequent in showing positive facial expressions (e.g., saying hello, smiling, etc.) compared to other regions in the US. It is possible that the emotional expression differences could be a result of how strongly individuals adhere to social display rules and propriety. We hypothesize that decreases in facial expressivity from individuals within these regions may have resulted in a floor effect within the dataset, such that differences between PDs and HCs could not be captured. While this explanation is purely speculative, further investigation into regional differences in facial emotional expression would be of interest.

Another limitation of the present study is that sub-clinical depression may have been present affecting facial expressivity. Future research should try to take into account any sub-clinical depression present in the PD groups (e.g., via covariate analyses) that could account for any blunted facial affect. Further, matching the HC group to the PD group on their score on the Beck Depression Inventory - II could better control for any underlying sub-clinical depression.

Rather than using a system like the *Facial Action Coding System* (*FACS*; Ekman & Friesen, 1978), the current study examined facial emotional expressions as evaluated by human raters. Evaluation by human raters is more subjective in nature than when using *FACS* and,
therefore, could be considered not as empirically rigorous, despite high inter-rater reliability. However, human ratings/evaluations have greater ecological validity and improved generalizability of results. Future research may want to use both methods of evaluation within a given sample.

**Implications and Conclusion**

Hypomimia limits PD patients’ ability to express themselves accurately and affects the ability to navigate their social world. Communication and inadequate social relationships are two of the most frustrating complaints among PD patients (e.g., Borod et al., 1990). This leads to misattributions by others, PD patients feeling embarrassed to communicate (Miller, Noble, Jones, & Burn, 2006), loss of interest from caregivers (Gunnery et al., 2016), and overall decline in quality of life for the individuals affected. Therefore, it is imperative that we understand the nature of such expressivity deficits in order to develop tailored rehabilitative strategies and to further our scientific understanding of emotional expression.

One of the major aims of this study was to examine how facial masking in PD is evaluated by others and how these judgments differ based on the gender of the patient and the gender of the observer. We garnered further support to the previously demonstrated finding that women are more facially expressive than men. In line with our hypothesis, female raters rated female posers as more expressive than male posers. However, we did not find the same congruency effect for male raters rating male posers; this is likely because female facial expressivity was higher than male expressivity.
Contrary to what we predicted, we did not find evidence to support that LSVT-LOUD improves facial masking in PD patients. However, our study has several important clinical implications. We provided additional evidence to support previous findings (e.g., Borod, Rogers, et al., 2008) that PDs experience emotion similarly to HCs, despite hypomimia symptomology. Expressivity deficits in PD due to depression, sub-clinical or otherwise, require further examination. Given that hypomimia is a feature of both depression and PD, diagnosing depression in PD can be difficult. Future research should focus on factors related to depression, self-worth, and social isolation and on how these factors interact with the hypomimia symptomology in PD. Understanding all dimensions of these factors will help patients with PD maintain an active role in our social world. The results of the current study further characterize the facial deficits seen in PD and explore the possibilities for clinically meaningful future research.
Appendix A
(Borod, Welkowitz, & Obler, 1992)

Screening Questionnaire

ID # _______________

1. Demographics:

   Gender: ☐ Male   ☐ Female
   Age: ______   Birthdate: _____________
   Ethnicity (What is your ethnicity?): _______________________
   Education: ______________________

   First language: ________________
   (If anything other than English, from what age have you been speaking Eng.? ___ years-old)

2. Medical History:

   Have you ever had any neurological problems (e.g. stroke, tumor, head injury etc.)?  
   ☐ Yes ☐ No

   Have you ever seen anyone for emotional problems (e.g. counselor, psychiatrist, psychologist, member of the clergy) or have you ever had any diagnosable psychiatric disorders (such as depression, substance abuse, bipolar disorder, etc.)?  
   ☐ Yes ☐ No

   Have you ever been hospitalized for any psychiatric problems or for substance abuse?  
   ☐ Yes ☐ No

   If yes, why and for how long?
   ____________________________________________________________
   ____________________________________________________________

   Have you ever had any problem with your vision?  
   ☐ Yes ☐ No
   If yes, what was the problem and how long did it last?
   ____________________________________________________________

   Do you have 20/20 vision with or without correction?  
   ☐ Yes ☐ No

3. Neurological History:

   Have you ever had any of the following diseases?

   ☐ Alzheimer’s Disease
   ☐ Arteriosclerosis
   ☐ Congenital Abnormalities
   ☐ Creutzfeldt - Jakob disease
   ☐ Dementia
   ☐ Epilepsy
   ☐ Huntington’s Chorea
   ☐ Korsakoff’s Syndrome
   ☐ Multiple Sclerosis
   ☐ Normal Pressure Hydrocephalus
   ☐ Parkinson’s Disease
   ☐ Pick’s Disease

67
Have you ever had a head injury or head trauma? Did you lose consciousness?
____________________________________________________________________________
____________________________________________________________________________
____________________________________________________________________________

4. Learning Disability:

Do you have a history of a learning disability?  □ Yes  □ No

Was it ever diagnosed by a professional?  □ Yes  □ No

Do you or have you had serious problems with reading, writing, spelling, or arithmetic?  □ Yes  □ No

5. Substance Use:

Have you ever considered cutting down on your alcohol intake?  □ Yes  □ No

Do people annoy you by criticizing your drinking?  □ Yes  □ No

Have you ever felt bad or guilty about your drinking?  □ Yes  □ No

Have you ever had an alcoholic drink first thing in the morning (eye-opener) to steady your nerves or get rid of a hangover?  □ Yes  □ No

Have you ever used recreational drugs?  □ Yes  □ No

Has drug use ever interfered with your daily functioning?  □ Yes  □ No

6. Handedness:

Do you consider yourself to be:  □ Right handed  □ Left handed  □ Ambidextrous?

In childhood or as an adult, did you ever switch, or were you ever forced to switch your handedness?  □ Yes  □ No

Are (or were) either of your parents or any of your full siblings left-handed or ambidextrous?  □ Yes  □ No
Table 1
Demographic Characteristics for Poser Participants (N=82)

<table>
<thead>
<tr>
<th></th>
<th>PD Group (N=64)</th>
<th>Total PDs (N=64)</th>
<th>Healthy Controls (N=18)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LSVT-LOUD® (n=22)</td>
<td>LSVT-ARTIC (n=19)</td>
<td>Untrx-PD (n=23)</td>
</tr>
<tr>
<td></td>
<td>Men (n=15) Men (n=7)</td>
<td>Men (n=14) Women (n=5)</td>
<td>Men (n=14) Women (n=9)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>67.9 (9.0) Men (n=15)</td>
<td>66.7 (6.8) Women (n=7)</td>
<td>66.9 (8.3) Men (n=14) Women (n=5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>16.5 (2.6) Men (n=15)</td>
<td>14.8 (3.3) Women (n=7)</td>
<td>17.2 (2.2) Men (n=14) Women (n=5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage of Disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.2 (0.5) Men (n=15)</td>
<td>2.5 (0.45) Women (n=7)</td>
<td>2.3 (0.58) Men (n=14) Women (n=5)</td>
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<td></td>
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</tr>
<tr>
<td>Time since diagnosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3.5 (2.6) Men (n=15)</td>
<td>6.8 (11.0) Women (n=7)</td>
<td>5.4 (5.9) Men (n=14) Women (n=5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>7.8 (4.5) Men (n=15)</td>
<td>13.6 (6.1) Women (n=7)</td>
<td>9.71 (6.4) Men (n=14) Women (n=5)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MMSE score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>28.8 (1.3) Men (n=15)</td>
<td>28.7 (1.6) Women (n=7)</td>
<td>28.7 (1.2) Men (n=14) Women (n=5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. BDI = Beck Depression Inventory (<15 mild depression, 15-30 moderate depression, >30 severe depression; Beck et al., 1996); MMSE = Mini Mental Status Examination (a 30-point scale where 20-24 = mild dementia, 13-20 = moderate dementia, and < 12 indicates severe dementia; Folstein et al., 1975); Hoehn-Yahr stage of disease ranges from 0 (minimal) to 6 (most severe; Hoehn-Yahr, 1967); Means are presented for each group. Standard Deviations are in parentheses.
Table 2
Demographic Characteristics for Rater Participants (N=24)

<table>
<thead>
<tr>
<th></th>
<th>Age $\bar{x}$ (in years)</th>
<th>Education $\bar{x}$ (in years)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Men (n=3)</td>
<td>Women (n=3)</td>
</tr>
<tr>
<td>Cohort 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n=6)</td>
<td>26.0</td>
<td>22.3</td>
</tr>
<tr>
<td></td>
<td>(11.3)</td>
<td>(2.5)</td>
</tr>
<tr>
<td>Cohort 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n=6)</td>
<td>20.0</td>
<td>21.3</td>
</tr>
<tr>
<td></td>
<td>(0.0)</td>
<td>(1.5)</td>
</tr>
<tr>
<td>Cohort 3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n=6)</td>
<td>21.3</td>
<td>21.7</td>
</tr>
<tr>
<td></td>
<td>(3.2)</td>
<td>(4.6)</td>
</tr>
<tr>
<td>Cohort 4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n=6)</td>
<td>19.3</td>
<td>21.3</td>
</tr>
<tr>
<td></td>
<td>(0.6)</td>
<td>(1.2)</td>
</tr>
<tr>
<td>Means</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>21.7</td>
<td>21.7</td>
</tr>
<tr>
<td></td>
<td>(3.8)</td>
<td>(2.5)</td>
</tr>
</tbody>
</table>

Note. Standard Deviations are in parentheses.
### Table 3
*Interrater Reliability: Intra-class Correlations for Conferencing and Practice Ratings*

<table>
<thead>
<tr>
<th>Training Variables</th>
<th>COHORT 1</th>
<th>COHORT 2</th>
<th>COHORT 3</th>
<th>COHORT 4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FM</td>
<td>EV</td>
<td>EF</td>
<td>EI</td>
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<tr>
<td><strong>Conferencing</strong></td>
<td>0.96</td>
<td>0.90</td>
<td>0.92</td>
<td>0.98</td>
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<tr>
<td><strong>Practice Ratings</strong></td>
<td>0.92</td>
<td>0.85</td>
<td>0.91</td>
<td>0.94</td>
</tr>
</tbody>
</table>

*Note.* FM = Facial Mobility; EV = Emotional Variability; EF = Emotional Frequency; EI = Emotional Intensity, SE = Social Engagement. The ICCs for conferencing are based on the original rating evaluation given by each rater, not the re-rating value that *may* have changed after the discussion part of conferencing.
Table 4
Group by Time by Emotion (4 x 2 x 4) ANOVA for Emotional Expression, Significance of Effects

<table>
<thead>
<tr>
<th></th>
<th>EF</th>
<th>EV</th>
<th>EI</th>
<th>SE</th>
<th>FM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emotion (4)</td>
<td>&lt; .001**</td>
<td>&lt; .001**</td>
<td>&lt; .001**</td>
<td>&lt; .001**</td>
<td>&lt; .001**</td>
</tr>
<tr>
<td>Time (2)</td>
<td>.457</td>
<td>.619</td>
<td>.621</td>
<td>.542</td>
<td>.882</td>
</tr>
<tr>
<td>Group (4)</td>
<td>.007*</td>
<td>.102</td>
<td>.063</td>
<td>.009*</td>
<td>.031*</td>
</tr>
<tr>
<td>Emotion x Group</td>
<td>.490</td>
<td>.546</td>
<td>.936</td>
<td>.705</td>
<td>.470</td>
</tr>
<tr>
<td>Time x Group</td>
<td>.758</td>
<td>.657</td>
<td>.316</td>
<td>.660</td>
<td>.679</td>
</tr>
<tr>
<td>Emotion x Time</td>
<td>.505</td>
<td>.853</td>
<td>.744</td>
<td>.870</td>
<td>.856</td>
</tr>
<tr>
<td>Emotion x Time x Group</td>
<td>.149</td>
<td>.480</td>
<td>.519</td>
<td>.621</td>
<td>.532</td>
</tr>
</tbody>
</table>

Note. * p ≤ .05, ** p ≤ .001
Table 5
**Correlations Between Emotional Experience and Emotional Expression**

<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Emotion Type</th>
<th>Experience (PRE)</th>
<th>Experience (POST)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Intensity</td>
<td>Accuracy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Healthy Controls</td>
<td>Happy</td>
<td>.37</td>
<td>.26</td>
</tr>
<tr>
<td></td>
<td>Angry</td>
<td>-.51*</td>
<td>-.58*</td>
</tr>
<tr>
<td></td>
<td>Sad</td>
<td>.30</td>
<td>.29</td>
</tr>
<tr>
<td>PDs (all 3 groups)</td>
<td>Happy</td>
<td>.11</td>
<td>.23*</td>
</tr>
<tr>
<td></td>
<td>Angry</td>
<td>.08</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>Sad</td>
<td>.40**</td>
<td>.19</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Emotion Type</th>
<th>Experience (PRE)</th>
<th>Experience (POST)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Intensity</td>
<td>Accuracy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Healthy Controls</td>
<td>Happy</td>
<td>.16</td>
<td>-.27</td>
</tr>
<tr>
<td></td>
<td>Angry</td>
<td>.03</td>
<td>.28</td>
</tr>
<tr>
<td></td>
<td>Sad</td>
<td>.02</td>
<td>.07</td>
</tr>
<tr>
<td>PDs (all 3 groups)</td>
<td>Happy</td>
<td>.14</td>
<td>.16</td>
</tr>
<tr>
<td></td>
<td>Angry</td>
<td>.02*</td>
<td>.01*</td>
</tr>
<tr>
<td></td>
<td>Sad</td>
<td>.24*</td>
<td>.29</td>
</tr>
</tbody>
</table>

*Note.* *p* ≤ .05, **p** ≤ .001

Table 6
**Correlation Comparisons Between Groups using the Fisher r-to-z Transformation**

<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Emotion Type</th>
<th>Experience (PRE)</th>
<th>Experience (POST)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Intensity</td>
<td>Accuracy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Healthy Controls</td>
<td>Happy</td>
<td>p = .17</td>
<td>p = .46</td>
</tr>
<tr>
<td></td>
<td>Angry</td>
<td>p = .02*</td>
<td>p = .01*</td>
</tr>
<tr>
<td></td>
<td>Sad</td>
<td>p = .35</td>
<td>p = .36</td>
</tr>
<tr>
<td>PDs vs. HCs</td>
<td>Happy</td>
<td>p = .47</td>
<td>p = .07</td>
</tr>
<tr>
<td></td>
<td>Angry</td>
<td>p = .48</td>
<td>p = .27</td>
</tr>
<tr>
<td></td>
<td>Sad</td>
<td>p = .23</td>
<td>p = .22</td>
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</tbody>
</table>

*Note.* *p* ≤ .05, **p** ≤ .001. Calculation according to Eid, Gollwitzer & Schmidt, 2011, pp. 547; single sided test
Table 7  
*Group by Emotion by Gender (2 x 3 x 2) ANOVAs, Significance of Effects*

<table>
<thead>
<tr>
<th>Emotional Experience</th>
<th>Intensity</th>
<th>Accuracy</th>
<th>Immediate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>.951</td>
<td>.771</td>
<td>.887</td>
</tr>
<tr>
<td>Emotion</td>
<td>.002*</td>
<td>.045*</td>
<td>&lt; .001**</td>
</tr>
<tr>
<td>Gender</td>
<td>.142</td>
<td>.552</td>
<td>.246</td>
</tr>
<tr>
<td>Group x Emotion</td>
<td>.830</td>
<td>.151</td>
<td>.421</td>
</tr>
<tr>
<td>Group x Gender</td>
<td>.191</td>
<td>.274</td>
<td>.060</td>
</tr>
<tr>
<td>Emotion x Gender</td>
<td>.346</td>
<td>.415</td>
<td>.663</td>
</tr>
<tr>
<td>Group x Emotion x Gender</td>
<td>.153</td>
<td>.724</td>
<td>.447</td>
</tr>
</tbody>
</table>

*Note. *p ≤ .05, **p ≤ .001*
Table 8
*Rater Gender by Poser Gender by Time by Emotion (2 x 2 x 2 x 4) ANOVA, Significance of Effects*

<table>
<thead>
<tr>
<th></th>
<th>EF</th>
<th>EV</th>
<th>EI</th>
<th>SE</th>
<th>FM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poser Gender</td>
<td>&lt; .001**</td>
<td>&lt; .001**</td>
<td>&lt; .001**</td>
<td>.014*</td>
<td>&lt; .001**</td>
</tr>
<tr>
<td>Rater Gender</td>
<td>.018*</td>
<td>.000**</td>
<td>.004**</td>
<td>.000**</td>
<td>.075</td>
</tr>
<tr>
<td>Time (2)</td>
<td>.801</td>
<td>.696</td>
<td>.869</td>
<td>.912</td>
<td>.479</td>
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<tr>
<td>Emotion (4)</td>
<td>&lt; .001**</td>
<td>&lt; .001**</td>
<td>&lt; .001**</td>
<td>&lt; .001**</td>
<td>&lt; .001**</td>
</tr>
<tr>
<td>PG x RG</td>
<td>.349</td>
<td>.665</td>
<td>.117</td>
<td>.002**</td>
<td>.238</td>
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<tr>
<td>PG x T</td>
<td>.679</td>
<td>.548</td>
<td>.400</td>
<td>.978</td>
<td>.438</td>
</tr>
<tr>
<td>PG x E</td>
<td>.005*</td>
<td>.038*</td>
<td>.025*</td>
<td>.005*</td>
<td>.011*</td>
</tr>
<tr>
<td>RG x T</td>
<td>.362</td>
<td>.556</td>
<td>.144</td>
<td>.247</td>
<td>.966</td>
</tr>
<tr>
<td>RG x E</td>
<td>.085</td>
<td>.305</td>
<td>.162</td>
<td>&lt; .001**</td>
<td>.026*</td>
</tr>
<tr>
<td>T x E</td>
<td>.960</td>
<td>.463</td>
<td>.949</td>
<td>.797</td>
<td>.201</td>
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<tr>
<td>PG x RG x T</td>
<td>.767</td>
<td>.398</td>
<td>.056</td>
<td>.791</td>
<td>.835</td>
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<tr>
<td>PG x RG x E</td>
<td>.655</td>
<td>.027*</td>
<td>.538</td>
<td>.374</td>
<td>.307</td>
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<td>PG x T x E</td>
<td>.366</td>
<td>.480</td>
<td>.123</td>
<td>.811</td>
<td>.118</td>
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<tr>
<td>RG x T x E</td>
<td>.057</td>
<td>.526</td>
<td>.379</td>
<td>.484</td>
<td>.957</td>
</tr>
<tr>
<td>PG x RG x T x E</td>
<td>.012*</td>
<td>.661</td>
<td>.325</td>
<td>.092</td>
<td>.001**</td>
</tr>
</tbody>
</table>

Note. * significant at $p \leq .05$; ** significant at $p \leq .001$. 
Figure 1

Aim 1: Main Effects of Group: Pairwise Comparisons

Note. The brackets indicate which groups are significantly different from one another at the .05 level after the Bonferroni correction.
**Figure 2**
Aim 1: Main Effects of Emotion: Pairwise Comparisons

Monologue Emotion

*Note.* The brackets indicate which groups are significantly different from one another at the .05 level after the Bonferroni correction.

**Figure 3**
Aim 2: Main Effects of Emotion: Pairwise Comparisons

Monologue Emotion

*Note.* The brackets indicate which groups are significantly different from one another at the .05 level after the Bonferroni correction.
**Figure 4**  
*Interaction: Poser Gender $\times$ Rater Gender*

### Social Engagement

<table>
<thead>
<tr>
<th></th>
<th>Male Rater</th>
<th>Female Rater</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male Poser</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female Poser</td>
<td>2.5</td>
<td>3.5</td>
</tr>
<tr>
<td>Male Poser</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Female Poser</td>
<td>3.5</td>
<td>4.5</td>
</tr>
</tbody>
</table>

*Note.* *Main effects are significant at $p \leq .05$.*
Figure 5a-b. Rater Gender x Poser Gender x Emotion x Time

5a. Emotional Frequency (Time 1)

5b. Emotional Frequency (Time 2)

Note. * Indicates significance at $p \leq .05$ after the Bonferroni correction
Figure 6a-b. Rater Gender x Poser Gender x Emotion x Time

6a. Facial Mobility (Time 1)

6b. Facial Mobility (Time 2)

* Indicates significance at $p \leq .05$ after the Bonferroni correction
Figures 7-11
Interaction: Poser Gender x Emotion
Figure 12
Main Effects of Poser Gender: Pairwise Comparisons

![Bar chart showing main effects of poser gender for various measures such as emotional variability, frequency, intensity, social engagement, and facial mobility.]

Note. All main effects are significant at $p < .01$ after the Bonferroni correction.

Figure 13
Main Effect of Emotion

![Bar chart showing main effect of emotion for various measures such as emotional variability, frequency, intensity, social engagement, and facial mobility.]

Note. The brackets indicate which groups are significantly different from one another at the .05 level after the Bonferroni correction.
Figure 14
Main Effects of Rater Gender: Pairwise Comparisons

Rater Gender

Note. * Main effects are significant at p ≤ .05 after the Bonferroni correction.
Figure 15

*Group Comparison of BDI scores*

Beck Depression Inventory Means

*Note. * Significant at $p \leq .05$. 
References


Parkinson’s disease and healthy control participants. Poster presented at the meeting of the International Neuropsychological Society, Portland, OR.


