INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps. Each original is also photographed in one exposure and is included in reduced form at the back of the book.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.
Facial EMG and the subjective experience of emotion in idiopathic Parkinson’s disease in response to affectively laden visual stimuli

Dalby, Patricia Reed, Ph.D.
The University of Arizona, 1994

Copyright ©1994 by Dalby, Patricia Reed. All rights reserved.
FACIAL EMG AND THE SUBJECTIVE EXPERIENCE OF EMOTION IN IDIOPATHIC PARKINSON'S DISEASE IN RESPONSE TO AFFECTIVELY LADEN VISUAL STIMULI

by

Patricia Reed Dalby

Copyright © Patricia Reed Dalby 1994

A Dissertation Submitted to the Faculty of the DEPARTMENT OF EDUCATIONAL PSYCHOLOGY In Partial Fulfillment of the Requirements For the Degree of DOCTOR OF PHILOSOPHY In the Graduate College THE UNIVERSITY OF ARIZONA

1994
As members of the Final Examination Committee, we certify that we have read the dissertation prepared by Patricia Reed Dalby entitled Facial EMG and the Subjective Experience of Emotion in Idiopathic Parkinson's Disease in Response to Affectively Laden Visual Stimuli and recommend that it be accepted as fulfilling the dissertation requirement for the Degree of Doctor of Philosophy.

Alfred W. Kaszniak, Ph.D. Co-Director
John E. Obrzut, Ph.D.
Lawrence M. Aleson, Ph.D.
Gary E. Schwartz, Ph.D.
Erwin B. Montgomery, M.D.

Final approval and acceptance of this dissertation is contingent upon the candidate's submission of the final copy of the dissertation to the Graduate College.

I hereby certify that I have read this dissertation prepared under my direction and recommend that it be accepted as fulfilling the dissertation requirement.

Dissertation Director

Date
STATEMENT BY AUTHOR

This dissertation has been submitted in partial fulfillment of requirements for an advanced degree at The University of Arizona and is deposited in the University Library to be made available to borrowers under rules of the library.

Brief quotations from this dissertation are allowable without special permission, provided that accurate acknowledgement of source is made. Requests for permission for extended quotation from or reproduction of this manuscript in whole or part may be granted by the copyright holder.

SIGNED: Patricia Reed Kelley
ACKNOWLEDGEMENTS

I wish to thank the co-directors, Alfred Kaszniak and John Obrzut, as well as the other members of my committee, Gary Schwartz, Erwin Montgomery, and Lawrence Aleamoni, for their guidance and intellectual challenge. My co-directors deserve special recognition. I thank Alfred Kaszniak, a mentor of genuine character and keen intellect, for giving generously of his time and for providing valuable critical advice. Because of his thought-provoking questions and great insight, I developed a better conceptualization of the research issues under study. I thank John Obrzut, an advisor of high standards of excellence, for giving sincere encouragement and continual motivation, paying great attention to detail, and for stressing simplification and clarification of thought in my writing. In addition, I'd like to thank both Alfred Kaszniak and John Obrzut for their friendship. The creative ingenuity and expertise of systems engineer, Mark Bakarich, and the professional assistance of support systems analyst, Jonathan Forster, is also deeply appreciated. I thank Ziya Dikman and Rick Haan for their assistance, as well. I also wish to thank Aldine von Isser for her inspiration and optimism and my close friends for their encouragement and understanding. Mostly, I wish to thank my parents for the importance they placed on knowledge and for their unstinting support throughout my graduate education, making the completion of this dissertation meaningful.
# TABLE OF CONTENTS

LIST OF TABLES ............................................. 8
LIST OF FIGURES ........................................... 9
ABSTRACT .................................................... 10
BODY ......................................................... 12

## CHAPTER

1 INTRODUCTION ............................................. 12

2 LITERATURE REVIEW .......................... 15
   Historical Overview of Related Theories .... 15
   Peripheral Mediation/James-Lange
      Hypothesis ........................................... 17
   Central Mediation/Cannon-Bard Theory of
      Emotion .............................................. 19
   Attitude Theory of Emotion ....................... 22
   Facial Feedback Theory: Tomkins. ............. 23

Facial EMG and Self-reported Emotion Relative
   to Task Assignment .................................. 27
Self-induced Imagery ................................... 29
   Within-subject ....................................... 29
Visually Presented Affective Scenes ............. 30
   Between Groups & Between Conditions ....... 30
Voluntary Manipulations of Facial
   Expression ........................................... 32

Patterns of Facial EMG, Self-reported
   Emotion, and ANS Specificity ..................... 33
Spontaneous Facial Actions to Visual
   Stimuli ............................................... 36
   Use of the International Affective
      Picture System ................................... 38
Spontaneous and Voluntary Facial
   Actions .............................................. 40
Implications for the Facial Feedback
   Hypothesis .......................................... 43

Behavioral Characteristics of Parkinson's
   Disease ............................................... 44
Depression in PD ....................................... 44
Perception of Emotion and Consequent
   Displayed Affect in PD .......................... 47
Facial Expression in PD ............................. 48
3 METHOD

1. Subjects
2. Materials
   a. Stimulus Materials: International Affective Picture System
   b. Neuropsychological Assessment Instruments
   c. Screening Instruments
   d. Testing Instruments
   e. Apparatus and Physiological Response Measurement

4 RESULTS

1. Descriptive Statistics
2. Mental State vs Mood State
3. Validation of PD Motor Symptoms on the UPDRS
4. Primary and Secondary Hypotheses Tested
   a. Primary Hypotheses--Subjective Experience of Emotion
   b. Collapsing of Slide Set A & Slide Set B Data
   c. Valence
   d. Arousal
   e. Primary Hypotheses--Facial EMG Activity
      f. Zygomatic EMG Change Scores
      g. Corrugator EMG Change Scores
      h. Zygomatic and Corrugator EMG Pattern
   f. Secondary Hypothesis--Levodopa Medication Effects on EMG Activity
      g. Baseline EMG Activity
**TABLE OF CONTENTS--Continued**

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zygomatic EMG Change Scores and Slide Presentation</td>
<td>120</td>
</tr>
<tr>
<td>Corrugator EMG Change Scores and Slide Presentation</td>
<td>120</td>
</tr>
<tr>
<td>Summary</td>
<td>121</td>
</tr>
<tr>
<td>5 DISCUSSION</td>
<td>123</td>
</tr>
<tr>
<td>Implications for Future Research</td>
<td>127</td>
</tr>
<tr>
<td>APPENDICES</td>
<td>132</td>
</tr>
<tr>
<td>APPENDIX A: Means, Standard Deviations, and Significance Levels of Individual Profile of Mood State Scales for the Parkinson's (PD) and Normal Control (NC) Groups Relative to Drug Condition</td>
<td>132</td>
</tr>
<tr>
<td>APPENDIX B: Significance Levels for Individual Movement Items on the Unified Parkinson's Disease Rating Scale Between Groups</td>
<td>133</td>
</tr>
<tr>
<td>APPENDIX C: Method for Deriving Positive, Neutral, and Negative Slide Types (PON)</td>
<td>134</td>
</tr>
<tr>
<td>APPENDIX D: Mean Valence Ratings by Group, On/Off Drug Condition, and Slide Type (PON)</td>
<td>135</td>
</tr>
<tr>
<td>APPENDIX E: Mean Arousal Ratings by Group, On/Off Drug Condition, and Slide Type (PON)</td>
<td>136</td>
</tr>
<tr>
<td>APPENDIX F: Mean Zygomatic and Mean Corrugator EMG Change Scores by On/Off Drug Condition and Slide Type (PON) Within the Parkinson's Group</td>
<td>137</td>
</tr>
<tr>
<td>APPENDIX G: Mean Zygomatic and Mean Corrugator EMG Change Scores by On/Off Drug Condition and Slide Type (PON) Within the Normal Control Group</td>
<td>138</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>139</td>
</tr>
</tbody>
</table>
LIST OF TABLES

TABLE 1, Means and Standard Deviations for Descriptive Characteristics of the Parkinson's (PD) Group and Normal Control (NC) Groups by Sex ................................. 95

TABLE 2, Means, Standard Deviations, and Significance Levels for the Beck Depression Inventory (BDI) for the Parkinson's (PD) and Normal Control (NC) Groups Relative to Drug Condition ................................. 97

TABLE 3, Means and Standard Deviations of Valence Ratings by Slide Type (PON) and Slide Set (A, B) for the Parkinson's (PD) and Normal Control (NC) Groups .................. 101

TABLE 4, Means and Standard Deviations of Arousal Ratings by Slide Type (PON) and Slide Set (A, B) for the Parkinson's (PD) and Normal Control (NC) Groups ............. 103

TABLE 5, Means and Standard Deviations of Valence Ratings by Slide Type (PON) for the Parkinson's (PD) and Normal Control (NC) Groups ........................................ 105

TABLE 6, Means and Standard Deviations of Arousal Ratings by Slide Type (PON) for the Parkinson's (PD) and Normal Control (NC) Groups ........................................ 108

TABLE 7, Means and Standard Deviations of Zygomatic and Corrugator EMG Change Scores by Slide Type (PON) for the Parkinson's (PD) and Normal Control (NC) Groups ............ 114
LIST OF FIGURES

FIGURE 1, Paper and pencil adaptation of the Self-Assessment Manikin (Lang, 1980) used to rate valence and arousal dimensions .. 78

FIGURE 2, Overall coordinates for the valence and arousal dimensions for slide set A ... 81

FIGURE 3, Overall coordinates for the valence and arousal dimensions for slide set B ... 81

FIGURE 4, Male and female coordinates for the valence and arousal dimensions for slide set A . 82

FIGURE 5, Male and female coordinates for the valence and arousal dimensions for slide set B . 82

FIGURE 6, Location of bilateral surface EMG electrode placement . . . . . . . . . 88

FIGURE 7, Mean valence ratings by group and slide type (PON) . . . . . . . . . . . . . . 106

FIGURE 8, Mean arousal ratings by group and slide type (PON) . . . . . . . . . . . . . . 109

FIGURE 9, Mean zygomatic EMG change scores by group and slide type (PON) . . . . . . 112

FIGURE 10, Mean corrugator EMG change scores by group and slide type (PON) . . . . . 113

FIGURE 11, Pattern of mean EMG change score responses by slide type (PON) and EMG site for the Parkinson's group . . . . . . . . . 116

FIGURE 12, Pattern of mean EMG change score responses by slide type (PON) and EMG site for the normal control group . . . . . . . . . 117

FIGURE 13, Mean baseline EMG activity by on/off drug condition for the Parkinson's (PD) and normal control (NC) groups . . . . . 119
ABSTRACT

The purpose of the study was to investigate the possible role of facial musculature movement in the subjective experience of emotion. Nineteen nondemented, nondepressed patients with idiopathic Parkinson's disease and 19 demographically matched control subjects were asked to rate valence and arousal dimensions after viewing emotionally laden slides. The patients with Parkinson's disease viewed one set of slides at their peak levodopa dose and one set of slides after at least a 12 hour abstention from their levodopa medication. Normal control subjects underwent two similar testing sessions, although no drug was administered. Mean valence and mean arousal ratings of slides within groups were determined. During the viewing of the slides, bilateral facial electromyographic activity in the zygomatic and corrugator muscle regions was recorded. EMG change scores relative to individual slide presentation were determined. Comparisons were made between and within groups of the mean valence, arousal, and EMG change scores relative to the slide valence type (i.e., positive, neutral, or negative slide content) and on/off drug condition. Results suggest that a subgroup of Parkinson's Disease patients experience similar emotional valence and arousal, to that of normal controls, when confronted with emotional visual stimuli. However, they display significantly less
facial muscular movement in the zygomatic muscle region and somewhat less facial muscular movement in the corrugator region than the normal controls. Implications of these results are discussed relative to the James-Lange theory that posits emotional experience to be dependent upon a peripheral "feedback" system versus the Cannon-Bard theory that posits emotion to be mediated centrally. Although the present results lend support to the Cannon-Bard theory of emotion, future research is necessary to determine the role of the skin of the face (with blood and temperature components), rather than the facial musculature per se, in the subjective experience of emotion. It may be that the skin of the face and the sound of one's own voice (among other factors) play important roles in the subjective experience of emotion as posited by S. S. Tomkins. If so, a modified peripheral mediation theory of emotion would be supported.
CHAPTER 1
INTRODUCTION

Numerous researchers have attempted to determine the role of facial expressions in the experience of emotion (e.g., Ekman & Friesen, 1975; Izard, 1977; Laird, 1984; Tomkins, 1962; and Zajonc, 1985). As a result, varied hypotheses have been proposed: the activity of facial muscles may affect brain functioning through peripheral feedback (Ekman, Levenson, & Friesen, 1983; Izard, 1977; Tomkins, 1962), by altering the blood flow to the brain (Tomkins, 1982; Zajonc, 1985), by the direct excitation of the hypothalamus by the motor cortex (Ekman, et al., 1983), or by the use of cues of facial expression in forming attributions about the self (Laird, 1984).

One of the most controversial theories put forth, in regard to the subjective experience of emotion, has been an adaptation of James' (1884/1922) feedback hypothesis by Tomkins (1962, 1963). In Tomkin's proposal, the action of facial musculature plays a primary role in inducing felt emotion. Tomkins (1982) has since modified his theory to posit that the facial musculature plays a lesser role in inducing specific affects, secondary to the skin in general and the skin of the face in particular, as well as voice feedback. That is, he purports that shifts in blood flow and temperature causes a change in the density of neural
firing, and that "the patterned changes in facial muscle responses serve as self-masturbatory stimulation to the skin and its own sensitized receptors" (Tomkins, 1982). He posits that the feedback of this set of changes provides the "feel" of specific affects.

The facial musculature component of Tomkins' theory has been tested by a number of researchers. However, few studies have tested Tomkins' theory using both subjective and physiological components within a study design that included both within subjects and between groups analyses. The present study purports to be among the first to do just that using facial electromyography (EMG) in subjects with idiopathic Parkinson's disease (PD) and demographically matched controls.

PD patients were used as the study population because mask-like facies and poverty of facial expression have been recognized as one of the hallmark characteristics of parkinsonian patients (Parkinson, 1817). Consequently, it was presumed that PD patients would show less movement of the facial muscles than normal controls (NCs) in response to emotional stimuli and would show proportionately reduced subjective experience. That is, when applying Tomkins' adaptation of James' feedback theory to PD patients, it was predicted that PD patients as a group would feel emotions less intensely because of their more rigid facial
expression.

Thus, the present study has a twofold primary purpose: (1) to determine whether PD patients with some degree of masked facies experience emotions less intensely than NCs, and (2) to determine whether PD patients show less facial musculature movement than NCs while viewing affect laden visual stimuli. A secondary purpose is to determine levodopa medication effects on facial efference and subjectively felt emotion in PD patients. Facial EMG studies in PD patients have shown that levodopa therapy results in a considerable decrease in resting EMG activity and reestablishes a reciprocal muscular activation (Hunker, Abbs, & Barlow, 1982; Leanderson, Meyerson, & Persson, 1971, 1972). Whether PD patients have greater facial mobility and therefore greater self-reported emotion at their peak levodopa dose is unknown.
CHAPTER 2
LITERATURE REVIEW

Following an overview of historical theories related to emotion and facial expression, selective reviews of studies correlating facial EMG and the subjective experience of emotion will be provided. Research concerning known and speculated deficits among PD patients that correlate facial expression, oral/facial impairment due to brain lesions, and facial EMG in PD will also be reviewed. Subsequently, definitions of relevant terms will be provided, followed by the stated primary and secondary hypotheses.

Historical Overview of Related Theories

Bartlett and Izard (1972) outlined two approaches to the study of emotion as it relates to subjective experience. The dimensional approach, which grew out of Spencer's (1890) concept of a pleasantness-unpleasantness continuum, suggests that emotion is not a special state in the organism but rather a more general process, commonly termed activation or arousal. Wundt (1896), a proponent of this approach, proposed that emotional experience varied along the dimensions of pleasantness-unpleasantness, excitement-quiet, and tension-relief. Since then, pleasantness and intensity or activation have appeared in every study of the dimensions of emotional experience.

The topological approach to the study of emotion is
distinguished from the dimensional approach by the fact that proponents of the former approach assume the existence of discrete emotions. Woodworth (1938) proposed that discrete emotions such as surprise, fear, and disgust can each be characterized by a continuous linear scale of intensity. Ekman (1989) has since been instrumental in clarifying discrete emotions, although Darwin (1872/1965) initiated the first systematic search for fundamental emotions. This approach suggests that there are different types of emotions that are qualitatively distinct relative to expression and possibly relative to appraisal, antecedent events, probable behavioral response, and physiology (among other factors) (Ekman, 1898). Thus, each discrete emotion is thought to constitute a special state or process in the organism and to have particular motivational and experiential properties (Ekman, 1973; Ekman & Friesen, 1975; Tomkins, 1962, 1963; Izard, 1972).

Currently there is agreement that there are three aspects of an emotion: the expressive, experiential (subjective experience), and physiological. Contemporary thought is concerned with distinctions between emotions across the three aspects of emotion, whether these three aspects of emotion are interrelated, and whether one of the three aspects of emotion is more important in the elicitation of an emotional reaction.
Peripheral Mediation/James-Lange Hypothesis

One line of research is concerned with the subjective experience of emotion and the bodily feelings and overt changes that accompany it. The James-Lange hypothesis has been instrumental in motivating subsequent theories in this area of emotion research.

The first systematic attempt to describe the relationship between the subjective experience of emotion and the corresponding bodily changes as independent of one another was attempted by James in 1884. He suggested that when one experiences an emotion a "feedback" system is evoked: an object which is externally present or imagined (e.g., objects associated with rage, love, fear) stimulates one or more sense organs; afferent impulses pass to the cortex and the object is perceived; subsequently impulses extend down to the muscles and viscera and alter them; afferent impulses from these disturbed organs course back to the cortex, and the object that was initially simply apprehended becomes the object emotionally felt (1884/1922). In other words, the feeling of the peripheral changes as they occur is the emotion; "...the bodily changes follow directly the perception of the exciting fact, and that our feeling of the same changes as they occur is the emotion" (James 1884/1922, p. 100). In James' view, even subtle emotional changes could be due to a variation in the
feedback from bodily physiologic changes. In essence, no emotion could be said to exist if the bodily experience of it was removed.

Lange (Lange & James, 1885/1922) shared James' basic conception of the root of the emotional experience, but narrowed the concept by suggesting that felt emotions were due to changes in the circulatory system alone. His ideas were put forth less clearly than James, but he evidently did not claim that the "feeling" of bodily changes was the emotion. Rather, he implied that vasomotor activity (vasomotor disturbances, varied dilatation of the blood vessels, and consequent excess of blood in the separate organs) are the real, primary effects of emotion. The other phenomenon such as abnormal motor movements, sensation paralysis, subjective sensations, disturbances of secretion, and intelligence were only secondary disturbances, due to abnormal vascular innervation.

Needless to say, these theories caused quite a stir in the scientific community and debate continues today. Researchers attempted to discredit the subjective aspects of James's theory by devising studies that appeared to show that subjective feelings are independent of peripheral changes. In addition, other research continued in an attempt to delineate the temporal relationships between patterns of physiological changes and emotional experience.
Central Mediation/Cannon-Bard Theory of Emotion

Cannon (1927) posited that the neural substrates for emotional expression reside in subcortical centers and that these centers are discharged instantaneously when they are properly stimulated. He suggested that an external situation stimulates receptors that in turn start impulses toward the cortex. Cannon posited that neurons concerned with emotional expression are within and near the thalamus and near the relay in the sensory path from the periphery to the cortex. Once these neurons are discharged in a particular combination, they innervate muscles and viscera and excite afferent paths to the cortex by direct connection or by spreading activation. Thus, according to Cannon, when the thalamic discharge occurs, the bodily changes occur almost simultaneously with the emotional experience. Lader and Tyrer (1975) suggested that the fundamental distinction between James's and Lange's and Cannon's theories is that the former two researchers' theories suggest that emotion is primarily peripherally mediated whereas the latter suggests emotion is centrally mediated.

At one time Cannon (1915) thought the same visceral changes occur in different emotional states as in non-emotional states. The visceral changes he referred to are brought on by sympathetic stimulation via accelerated heart rate, contracted arterioles, dilated bronchioles, increased
blood sugar, inhibited activity of the digestive glands, inhibited gastro-intestinal peristalsis, sweating, epinephrine discharge, widened pupils, and erected hairs. Cannon (1915) proposed that these changes are seen in great excitement under any circumstances, and that the viscera are too uniform to allow for emotion specificity in relation to intensity of subjective report of felt emotion.

However, in 1927 Cannon revised his theory, proposing that although the chief part of "felt" emotion originated from the viscera, a back flow of impulses from the periphery, arising from all parts of the organism (i.e., the muscles, skin, and viscera), play a contributory part in the experience of emotion.

In an effort to further delineate the source of the emotional experience, the two sources of the afferent processes--the visceral and the vasomotor--began to be researched (Sherrington, 1900). That is, researchers thought that if all sympathetic channels were removed in experimental animals, all sensations of felt emotion would be abolished eliminating the possibility of return impulses by these channels to the cortex. Therefore, if the viscera were separated from the central nervous system, according to James' theory the "felt" emotion should largely disappear, and according to Lange's modified theory the "felt" emotion should entirely disappear. However, when presented with an
emotional stimulus, dogs appeared to have a normal "emotional experience" in that they displayed facial efference, head and foreleg movements, and vocalization.

Whether such reactions were indeed emotionally felt experiences remains in question. Total separation of the viscera from the central nervous system in animals was found not to alter emotional behavior (Cannon, Lewis, & Britton, 1927; Sherrington, 1900). However, Angell (1916), Perry (1926), and others, suggested that there was no real basis for either affirming or negating the presence of "felt emotion" in surgically transected animals.

Cannon (1931) reinterpreted the functional significance of peripheral physiological changes in emotional states by suggesting that bodily changes during acute emotional arousal were due to sympathetic nervous discharge. He maintained that the sympathetic division of the autonomic nervous system was activated at times of "flight or fight" whereas the parasympathetic (cranial and sacral) division was responsible for fortifying the bodily reserves to withstand the stress of the situation. Accordingly, Cannon appeared to consider the peripheral bodily changes as being adaptive in nature.

In 1928 Bard expanded Cannon's 1927 revised theory by providing evidence that the diencephalon and associated structures were necessary for the expression of emotional
behavior.

Subsequently, Papez (1937) discounted the thalamus as having the chief role in emotion and proposed what became known as the Papez circuit—the fornix, mammillary bodies, anterior thalamic nuclei, parahippocampal, and cingulate gyri of the brain (known today as the "limbic system")—as the primary functional connection between the cerebral cortex, hypothalamus, and reticular formation.

Attitude Theory of Emotion

As an outgrowth of Cannon and Bard's theories, more contemporary physiologically—based theories of brain functioning evolved. Nina Bull (1951) argued that confusion as to which comes first—bodily changes or subjective experience—was due to a failure to separate emotional efference into its component parts. She suggested that James was mistaken only in that he focused on the action component rather than on the preparatory motor attitude: "We feel angry as a result of readiness to strike, and feel afraid as a result of readiness to run away, and not because of actually hitting out or running, as James explained the sequence" (p.6).

Bull postulated that the spontaneous postural attitudes taken prior to action are accompanied by appropriate organic changes, and that "feelings of these organic changes combine with the feelings of the orienting posture itself—and with
some awareness of the original exciting stimulus—to produce the familiar experience known as emotion" (p. 5). She further stated that feeling "...may follow and accompany a motor attitude, but does not necessarily do so; and cannot possibly precede it--cannot in fact appear at all without an antecedent motor attitude to fire the afferent pathways from the muscles and viscera to the brain" (p. 19). Feeling, according to Bull, was shown as "...belonging to an intermediary state, being dependent on a delay occurring after the set-up of the preliminary motor attitude. It is actually the feeling or consciousness of the motor attitude, and indicates a holding up of the consummatory action" (1951, p. 13).

Bull considered her attitude theory of emotion to be an extension of the James-Lange's "peripheral" theory. Within this context, Bull did not assign a special role to the face. But during the next decade, emotion theorists began to postulate a specific and central role of the facial musculature in the experience of emotion.

**Facial Feedback Theory: Tomkins**

Tomkins (1962, 1963) thought of emotion as either immediately rewarding or punishing experiences that are mediated by receptors activated by an individual's own responses. Tomkins proposed that emotion is "activated" by an increase, steady level, or decrease in the density of
neural firing or stimulation (i.e., the number of neural firings per unit in time). He posited that both positive and negative affects (interest, startle, and fear) are activated by stimulation increase, negative affects (distress, anger) are activated by a steady level of stimulation, and a positive affect (joy) is activated by a decrease in stimulation. Adelmann and Zajonc (1989) paraphrased Tomkins feedback cycle:

"... a stimulus activates an innate, subcortical "affect program," which emits messages through the motor and circulatory pathways to the entire body. The responses of the affected motor and glandular targets--the face primarily, other sites secondarily--supply sensory feedback to the brain, which, if it reaches consciousness, is subjectively experienced as emotion. Tomkins argued that this feedback may be acted upon whether or not it reaches awareness, that voluntary facial efference may not accurately duplicate the innate pattern, and that the sequence may be initiated by retrieved conscious affect or central imagery as well as by an external emotion stimulus." (p. 257).

In the early 1960s Tomkins laid out the foundation of the feedback theory as being tied to the facial musculature in a two-volume set of books (1962, 1963). He regarded the inner bodily responses, after the James-Lange theory, to be
important but secondary to the expression of emotion through the face; "...the face expresses affect, both to others, and to the self, via feedback, which is more rapid and more complex than any stimulation of which the slower moving visceral organs are capable" (1962, pp. 205-206). The tongue and facial muscles, the sound of one's own voice in the ears, and the changes in blood-flow and temperature of the face were considered by Tomkins as factors providing feedback from the face. Thus, he proposed that the face was primary over other bodily physiological changes in eliciting emotion because the nerves and the muscles of the face are more finely differentiated than the viscera, allowing for the face to be the most sensitive and dominant part of the body with a high density of "neural representation and firing" (1962, p. 208), and, therefore, more capable of rapid and flexible response than the slower moving viscera.

In 1980 Tomkins modified his theory concerning the role of the face in felt emotion by suggesting that facial muscles are specialized for action and not for affect. Specifically, he proposed that receptors that are normally hidden in the skin change position in response to the facial muscle patterns in the expressive face and that, therefore, feedback results from these cutaneous receptors rather than the muscles of the face.

More recently, Tomkins (1982) postulated eight specific
innate affects (interest/excitement, enjoyment/joy, surprise/startle, distress/anguish, fear/terror, shame/humiliation, contempt/disgust, and anger/rage) that are revealed in facial and skin responses (due to blood flow and temperature changes), and that the blood flow and temperature changes of the skin of the face rather than the facial musculature is the major mechanism involved in the response.

Tomkins also modified his theory of affect as amplification (1982), in that he no longer expects a gain in electronic amplification (e.g., electromyography (EMG), galvanic skin response (GSR), or heart rate (HR) activity) to correspond with increased affective amplification, because affects are separate mechanisms involving bodily responses quite distinct from other bodily responses they are presumed to amplify. In essence he proposed the following:

"...increased gradients of rising neural firing activate the specific affect as the slope of increasing density of neural firing becomes steeper. Enjoyment is activated by a decreasing gradient of neural firing; distress is activated by a decreasing gradient of neural firing, which exceeds an optimal level by an as yet undetermined magnitude; and anger is also activated by a nonoptimal level of neural firing but one that is
substantially higher than that which activates distress. Increase, decrease, or level of neural firing are in this model the sufficient conditions for activating specific affects" (p. 384).

Tomkins still regards the voice and face to be major contributors to the feedback process experienced as affect, but now rather than considering the facial musculature to be of primary importance, he deems the skin of the face (with blood flow and temperature components) to be of greatest importance in producing the feeling of affect. In addition, Tomkins continues to stress that facial affective responses are not necessary (e.g., imagery can conjure up experience of affect) or sufficient (other bodily responses occur as well) conditions for the conscious experience of affect. Also, innate vocalization programs and patterns of breathing that are specific to affect continue to be major factors in the experience of affect, according to Tomkins.

**Facial EMG and Self-Reported Emotion Relative to Task Assignment**

A number of EMG studies of facial expressions of emotions and patterns of emotions have evolved over the years and may be categorized as falling in one or more of the following categories: facial EMG studies of affective imagery and affective disorder, facial EMG studies of overt expressions, and facial EMG studies of social interaction.
Because the latter category deals primarily with the perception of emotion, a review of facial EMG studies of social interaction will not be imparted here (see Fridlund & Izard, 1983 and Tassinary & Cacioppo, 1992 for reviews).

In the correlational literature on facial feedback, external stimuli such as films or slides have usually been used although other techniques such as imagery have been used as well. Correlational studies have focused primarily on the physiological whereas experimental studies have focused on the subjective experience as it relates to facial efference. A few studies, however, have collected both physiological and subjective emotional data from the same subject population. Since the subjective experience of emotion relative to facial efference is more directly pertinent to the facial feedback hypothesis, all studies related here have a subjective experience component.

Isolated uses of facial EMG recordings can be found in the late 1950's and early 1960's (Sumitsuji, Matsumoto, & Kaneko, 1965; Whatmore & Ellis, 1959, 1962), but most facial EMG research has been conducted since the mid 1970's. Whatmore and Ellis (1959, 1962) were instrumental in that they found a relationship between invisible facial action and mood state. Specifically, they showed that those with retarded depression to the point of being mute or almost mute with overt immobility evidenced significantly higher
"invisible" residual motor activity than the control group did in four motor regions (forehead, jaw/tongue, forearm, and leg). Yet, in spite of these findings, the study of facial EMG and subjective experience of emotion did not regain vigorous activity until the 1970s and '80s.

During the '70s and '80s, researchers investigated the subjective experience of emotion within the context of facial muscular movement in response to self-generated imagery or visually presented affective laden stimuli—even when no apparent overt expression could be perceived—and in response to voluntary manipulation of facial expressions.

Self-Induced Imagery

Within-Subject. To date, numerous within-subject studies have used a self-generated imagery paradigm which asks subjects to imagine happy or sad thoughts while EMG activity is monitored. Results have been fairly consistent, showing greater EMG activity in the corrugator muscle region during unhappy imagery and greater EMG activity in the zygomatic region during happy thoughts (Brown & Schwartz, 1980; Clark, 1986; Fridlund, Schwartz, & Fowler, 1984; Teasdale & Bancroft, 1977). In addition, intensities of different emotions have been found to evoke differential patterns of self-reports and EMG activity (Brown & Schwartz, 1980). Moreover, gender effects have been found using EMG and the imagery paradigm; females showed greater EMG
activity and stronger experience of emotion than men, and both the zygomatic major and corrugator muscle regions across genders showed greater sensitivity in the imagery condition and more association with the subjective experience of emotion than other muscle sites (i.e., masseter and frontalis) (Schwartz, Brown, & Ahern, 1980).

Visually Presented Affective Scenes

Between Groups & Between Conditions. Because affectively laden visual stimuli are used in the present study, a number of studies using visual stimuli to evoke facial EMG activity and the subjective experience of emotion will be reviewed. Most of these studies tested EMG activity between groups and/or between conditions.

Conclusions based on Dimberg's (1990a) review of data collected in his laboratory on facial EMG responses to different external emotional stimuli, concur with the theory that the facial muscle reaction is a general component of the emotional response, and that the facial EMG technique is a sensitive tool for measuring emotional reactions. His findings from conditioning experiments (Dimberg, 1983, 1988a) went so far as to suggest that it is possible to aversively condition facial reactions within a Pavlovian conditioning paradigm, and his work supports the hypothesis that humans are biologically prepared to associate angry faces with aversive outcomes or prewired to react with a
"negative" emotional response to angry faces.

Another experiment by Dimberg (1988b) required subjects to rate their own specific emotions immediately after exposure to angry or happy faces. Results revealed that subjects reported more fear after being exposed to angry as compared to happy faces, and that happy faces induced significantly more happiness. The remaining rating dimensions were not affected although angry stimuli tended to induce more surprise. These data, according to Dimberg, demonstrate that spontaneously elicited facial EMG reactions are accompanied by a corresponding change in perceived emotion. McHugo (1983) agreed finding that zygomatic major activity while watching positive films correlated with positive self-reported emotion whereas corrugator activity during negative films correlated with higher self-reported anger.

Results of additional studies by Dimberg (1990) indicated that the facial EMG technique appears to be sensitive in detecting different response patterns among subjects suffering from specific fears. This extends past findings that facial EMG detects differences between depressed and nondepressed patients (Schwartz, Fair, Salt, Mandel, & Klerman, 1976a, 1976b).

Dimberg pointed out, however, that specific correlational data at the individual level (in addition to
between-group, between condition studies) are needed in order to more thoroughly evaluate the relation between EMG responsiveness and other aspects of the emotional response system.

**Voluntary Manipulations of Facial Expressions**

Those studying the effects of voluntary manipulation of facial expression (e.g., inhibited or exaggerated expressions—Cacioppo, Petty, Losch, & Kim, 1986; McCanne & Anderson, 1987; or posed expressions—Smith, McHugo, & Lanzetta, 1986) on subjective experience of emotion found consistent changes in facial EMG relative to positive emotional stimuli and mixed changes in facial EMG relative to negative emotionally laden stimuli. For example, Cacioppo et al. (1986) showed that EMG activity over the corrugator (mid brow region) and orbicularis oculi (periocular region) muscle sites vary as a function of valence and intensity of the subjects' affective reactions to the visually presented scenes; the more subjects liked a scene, the lower the level of EMG activity over the brow region and the higher the EMG activity over the periocular region. Also, EMG activity was greater over the cheek region (zygomatic major) for liked than disliked scenes. In contrast, McCanne and Anderson (1987) found no support for the effects of suppression of facial muscle tension on negative affective experiences.
It is important to note that McCanne and Anderson's (1987) subjects were able to produce reliable changes in corrugator and zygomatic EMG without visible facial changes in eight trials or fewer. The paradigm involved having subjects imagine positive and negative affective scenes in three counterbalanced conditions: (1) simply imagine the scene, (2) imagine the situation and enhance the muscle tension in one or two muscle groups (zygomatic or corrugator), and (3) imagine the situation and suppress the muscle tension. Findings suggested that subjects experience less enjoyment and more distress during positive affective trials when they suppressed zygomatic EMG activity. These results are consistent with Izard's (1927, 1977) and Tomkins' (1962) respective theories that purported changes in facial musculature may alter brain functioning through afferent feedback in that the alterations in facial musculature played an important role in producing changes in emotional responding. These results also lent support to Laird's (1984) contention that subjects use facial muscle cues in making inferences about their own behavior.

**Patterns of Facial EMG, Self-Reported Emotion, and ANS Response Specificity**

Ekman, et al. (1983) suggested facial muscular activity triggers the autonomic reactions directly, either by means of facial feedback or through the direct excitation of the
hypothalamus by the motor cortex. Therefore, although James assigned only an auxiliary role to autonomic arousal in his original feedback concept, Ekman and others are currently studying facial efference patterns in the context of patterns of autonomic arousal.

Buck (1980) proposed that two versions of the facial feedback hypothesis have been tested relative to autonomic activity and emotion, causing some confusion in the literature: (1) a between subjects version (i.e., that individual differences in the feedback from emotional expression are related to individual differences on other emotional indices—"...that for a given emotional stimulus, Person A who freely expresses his or her feelings will have a greater response on other indices of emotion than will a different Person B who shows little expression" (p. 813)), and (2) a within-subjects version (i.e., "...Person A confronted by a given emotional stimulus will have greater response on other affective indices if he or she freely expresses an emotion than if that same Person A were to show little experience" (p. 813)). Thus, a review of studies showed it is possible to simultaneously have negative intersubject and positive intrasubject relationships between emotional expression and other affective indices (Buck, 1980). For example, "...Person A may have a larger GSR when he or she is expressive than when he or she is not, but
Person A who in general is expressive tends to have a smaller skin conductance response than Person B who in general is not expressive (p. 816).

More recently Levenson and colleagues (Levenson, 1988; Levenson, Carstensen, Friesen, & Ekman, 1991; Levenson, Ekman, & Friesen, 1990) successfully attempted to distinguish emotion specific autonomic nervous system (ANS) activity in humans of all ages. However, the physiological correlates of spontaneous facial efference have been mixed when within-subjects designs (as defined by Buck, 1980) have been used; GSR and HR have not consistently discriminated positive from negative facial affect although GSR and HR have seemed to increase with intensity of facial efference (see Adelmann & Zajonc, 1989 for review). Yet, in many of these studies researchers found an inverse relationship between the degree of individual's emotional expressivity (e.g., facial) and autonomic arousal.

Of importance, relative to the feedback hypothesis, is that this inverse relationship between subjects does not rule out the possibility of a positive correlation between expressivity and arousal within subjects, as predicted by the facial feedback hypothesis. In most within-subjects studies this association has been found.

Since individuals in the present study were asked to respond spontaneously to visual stimuli, a number of within-
subject facial EMG studies using visual stimuli have been reviewed here. Several studies using The International Affective Picture System (IAPS) (Lang & Greenwald, 1988; Lang, Greenwald, & Bradley, 1988, 1990; Lang, Ohman, & Vaitl, 1988) have been presented as well, since portions of the IAPS stimuli was used in the present study.

**Spontaneous Facial Actions in Response to Visual Stimuli**

In the past and more recently, researchers have used positive and negative film stimuli, in an attempt to elicit differential patterns of facial EMG relative to GSR and HR changes. As a result, a consistent pattern has evolved relative to GSR activity whereas inconsistent patterns have been noted relative to HR changes. During spontaneous facial actions to film stimuli, researchers found augmented electrodermal activity in response to both positive and negative film presentation (Hubert & deJong-Meyer, 1990; Zuckerman, Klorman, Larrance, & Spiegel, 1981). In contrast, an inconsistent pattern has been noted relative to HR changes in response to emotionally laden films. Hubert and deJong-Meyer (1990) found HR to correlate positively with facial EMG when positive film stimuli were presented but correlate negatively when negative stimuli were shown. However, Ancoli (1979) showed HR to increase "before" facial response to pleasant films but increase "during" facial response to unpleasant films.
Of interest, the pattern of consistently higher GSR changes in response to visual stimuli has not been replicated when voluntary (i.e., imitated) facial actions have been requested (McHugo, Lanzetta, Sullivan, Masters, & Englis, 1985); in such instances a lower GSR occurred during happy facial configurations when subjects reported feeling joyful and "warm" (i.e., the Comforted and Supportive triads averaged) and a higher GSR was noted during angry facial configurations when subjects reported a negative emotional state. McHugo and colleagues (1985) interpreted these findings as consistent with the view that facial expressive displays (i.e., that which is then imitated) appear to evoke patterned visceral and facial muscle changes in observers that are in part independent of self-reported reactions.

McHugo and colleagues also noted that self-reported emotion following presentation of visual stimuli consistently showed a more relaxed, joyful, less anxious mood state following positive films and slides and more anxious, disgusted, and less relaxed feelings following negative stimuli (McHugo et al., 1985; McHugo, Smith, & Lanzetta, 1982).

In addition, facial EMG activity has differentiated both valence and intensity of the affective reaction following exposure to slides and tones devised to evoke mildly to moderately positive and negative affect (Cacioppo,
et al., 1986). In Cacioppo et al.'s study (1986) facial EMG activity differentiated the pleasantness and intensity of individual's affective reactions to the visual stimuli even though the subjects' expressions of emotion were not apparent on videotape analysis. These results suggest that facial EMG can provide objective and continuous probes of affective processes that are too subtle or fleeting to evoke visible expressions during normal social interaction.

**Use of the International Affective Picture System.**

Lang and colleagues introduced variations of the International Affective Picture System (IAPS) (see Materials section for description of stimulus) to ascertain affective, facial, visceral, and behavioral reactions. As a result, a better understanding of laterality and gender effects relative to facial expression has evolved.

Several studies have monitored EMG activity of the corrugator, zygomatic major, and orbicularis oculi muscle regions; heart rate; and skin conductance in an effort to determine the dependence or independence of valence, arousal, interest, and attention while subjects viewed objects in the foreground of slide presentations (e.g., a rabbit standing in the foreground with shrubbery and grass in the background). Results showed a consistent linear pattern when analyzing the corrugator EMG data; the more positively valent the stimuli the less corrugator activity
and the more negatively valent the stimuli the greater the corrugator activity. However, when analyzing zygomatic EMG data a quadratic trend was found largely due to movement of the zygomatic muscles upon grimacing in response to disgusting slides (e.g., mutilated face or body) (Greenwald, Cook, & Lang, 1989; Lang, Greenwald, Bradley, & Hamm, 1993). Regression analyses showed that differential facial EMG activity and peak HR acceleration are both specific and sensitive to valence (pleasure-displeasure) ratings, while skin conductance is specific and sensitive to arousal ratings (Greenwald, et. al., 1989).

Additionally, researchers showed significant gender effects relative to EMG and ANS activity in college populations. Males showed greater concordance between arousal and skin conductance and females demonstrated greater overall zygomatic EMG (Greenwald, et al., 1989). Lang et al. (1993) found that women produced greater mean corrugator response than men across all pictures. Also, almost twice as many women as men had significant correlations between valence judgements and corrugator response. The zygomatic activity of women varied more closely with valence than did that of men as well; women had a significantly larger mean quadratic correlation \( r = .36 \) than did men \( r = .04 \) between valence and zygomatic tension. Furthermore, three times more women (76\%) than men
(26%) showed a significant within-subject covariation. No differences in concordance between valence ratings and HR acceleration were noted.

**Spontaneous and Voluntary Facial Actions**

Given that voluntary and spontaneous emotional expressions are controlled by different neural pathways (i.e., voluntary facial actions receive their impulses from the cortical motor strip, passing through the corticobulbar projections whereas spontaneous facial actions are mediated by the extrapyramidal motor system), it is not surprising that numerous studies emerged testing voluntary versus spontaneous facial expression and subjective experience of emotion. However, these studies, investigating inhibition or exaggeration of facial expression, congruent poses of facial expressions, and direct facial movements (i.e., coached muscle-by-muscle movement), showed conflicting patterns relative to self-reported emotion and ANS activity.

Lanzetta, Cartwright-Smith, and Kleck (1976) tested the effects of nonverbal dissimulation on emotional experience and ANS activity. Subjects either concealed or exaggerated the facial display associated with the anticipation and reception of painful shocks that varied in intensity. Both self-reports of shock painfulness and skin conductance measures of emotional response showed significant changes paralleling the changes induced in expressive behavior; that
is, attenuation of expressive responses to shocks of varying intensity appears to consistently produce a decrease in the magnitude of autonomic arousal and in subjective reports of shock painfulness, whereas the free expression of pain-related affect produces either a decrement or an increment in arousal, depending on the absence or presence of a camera. Such findings lend partial support to Tomkins' facial feedback theory in that greater facial muscle movement produced greater self-reported emotion.

In contrast, Tourangeau and Ellsworth (1979) found that for fearful and sad films, a congruent facial pose did not increase self-reported emotion of the matched emotion above that reported by the unmanipulated (spontaneous facial efference) and nonemotional-faced groups. Also, the incongruent poses did not decrease self-reports of the filmed emotion. However, posed fearful and sad facial expressions corresponded with a greater drop in heart rate than seen in the nonemotional-faced group across all films. Still, this decrease was less than that seen in the spontaneous expresser group. Also, a larger galvanic skin response was noted in the spontaneous expresser group whereas the lowest galvanic skin response was evident in the subjects posing fearful expressions.

Levenson et al., (1990) found different ANS response patterns existed in voluntary facial actions, as well.
Subjects received muscle-by-muscle instructions and coaching to produce facial configurations for anger, disgust, fear, happiness, sadness, and surprise while heart rate, skin conductance, finger temperature, and somatic activity were monitored. Results indicated that autonomic distinctions among emotions are found both between negative and positive emotions and among negative emotions, are consistent between group and individual subjects' data across gender, and are stronger when the voluntary facial configurations most closely resembled actual emotional expressions. Also, the voluntary activity produced significant levels of subjective experience of the associated emotion. This may have been the first study to reveal the capacity of voluntary facial expressions to generate emotion-specific ANS activity without mimicking facial expressions.

More recently, Levenson and colleagues (Levenson, et al., 1991) tested these results in an older population (N = 20; ages 71-83, median 77) using the same muscle-by-muscle coaching paradigm as well as an imagery condition while ANS activity was monitored. Most of the results resembled that found with younger subjects (Levenson, et al., 1990) although the magnitude of change in ANS measures was smaller in older than in younger subjects. In the directed facial action task, however, differences were found between elderly and young subjects in both subjective report and facial
expression. The mean quality rating of facial configurations was lower for elderly subjects than younger and elderly subjects reported experiencing the target emotion much less often than did the younger subjects in spite of the finding that neither group had more difficulty than the other in forming the instructed expressions.

In the relived emotions task no subjective or expressive differences between elderly and younger subjects were noted. Levenson and colleagues argued that the reasoning for the differential effects between age groups on the directed facial action task may be due to a lessening of voluntary muscular control with age or that this artificial task may not have been engaging for elderly subjects. However, Carstensen's selectivity theory (1987, 1989) suggests that the appearance of less emotionality in facial expression may be due to lowered levels of social activity representing an affect regulation strategy that is prominent in old age. That is, limiting social interaction to people and issues of great importance to the individual can optimize positive emotional experiences and minimize negative emotional experiences; this lessening of negative affect, according to Carstensen, could then create the appearance of lowered emotionality in the elderly.

**Implications for the Facial Feedback Hypothesis**

The between-subjects studies as a group show that
facial efference is negatively correlated with autonomic arousal. However, within-subjects comparisons show that increased facial efference is correlated with increased physiological arousal as the facial feedback hypothesis would suggest.

In regard to subjective experience (self-reported emotion) relative to spontaneous facial actions, the degree of self-reported emotion seems to correspond almost consistently to facial efference patterns of the corresponding emotion (i.e., a greater degree of happiness is reported when a happy facial efference is displayed). Voluntary facial efference, whether produced, inhibited, or exaggerated on request, seems to change the subjective experience as well. Most studies suggest that the more intense a congruent facial pattern the more intense the subjectively felt emotion. Conversely, the more inhibited the congruent pose or a pose of an incongruent emotion, the less the subjective experience if felt. These results also support the Tomkins' facial feedback hypothesis that purports the facial muscles play a role in the subjective experience of positive versus negative emotion.

**Behavioral Characteristics of Parkinson's Disease**

**Depression in PD**

Depression is often among the characteristic symptoms of PD. After reviewing 14 studies, Gotham, Brown, and
Marsden (1988) found a mean estimate of 46% of PD patients to be concordantly depressed. Some suggest that depression in PD reflects biochemical and neuroanatomical changes intrinsic to PD while others claim that depression in PD is a reaction to the illness (i.e., individual coping styles and availability of support).

Fibiger (1984) suggested that damage to the reward-related systems (degeneration of mesolimbic and mesocortical dopamine projections) may contribute directly to the high incidence of depression reported in PD.

Another theory suggests a subtype of PD in whom a decrease of serotonin levels, as well as dopamine levels, leads to depressive illness (Mayeux, Stern, Cote, & Williams, 1984). Mayeux, et al., (1984) found that the CSF content of the major metabolite of serotonin, 5-hydroxyindoleacetic acid (5HIAA), was lower in depressed than in nondepressed PD patients. They suggest that their findings parallel the known loss of serotonin 5HIAA in postmortem brain in PD ((Fahn, Libsch, & Cutler, 1971).

Mayeux and colleagues suggested that a reduction in CSF 5HIAA might be anticipated in most patients with PD because of one of two reasons: (1) impaired synthesis of serotonin in the median raphe where dopa decarboxylase is decreased in PD, or (2) because of "down regulation" to compensate for the loss of dopamine from nigrostriatal neurons--possibly
due the ability of serotonin to inhibit motor activity (in animals) by limiting the effect of dopamine in the substantia nigra. However, they found a further reduction of CSF 5HIAA than normally anticipated in depressed PD patients.

Van Praag (1982b) has proposed a "serotonin hypothesis" of depression based on the following findings: serotonin content is reduced in postmortem brain from suicide and depressives dying from natural causes (Birkmayer & Riederer, 1975; Lloyd, Farley, Deck, & Hornykiewicz, 1974) the CSF content of 5HIAA is reduced in depressed patients (Asberg, Thoren, Traskman, Bertilsson, & Ringberger, 1976; Asberg & Traskman, 1981); and the amino acid precursor of serotonin, plasma tryptophan, shows a decreased ratio to concentrations of competing amino acids (Moller, Kirk, & Fremming, 1976; Moller, Kirk, & Honore, 1980; van Praag, 1982a).

It is unclear whether levodopa therapy enhances or diminishes depressive symptomatology in PD. Some researchers claim that levodopa therapy does not create depressive symptoms but may in fact diminish the symptoms (Celesia & Wanamaker, 1972; Marsh & Markham, 1973). In contrast, Mindham, Marsden, and Parkes (1976) showed levodopa therapy to cause an increase in psychiatric symptoms including depression in their PD population. Still others found no effect on depression with the use of
levodopa (Marsh & Markham, 1973), and others found antidepressant use (Bowen, Hoehn, & Yahr 1972; Mayeux, Williams, Stern, & Cote, 1984) does not induce depression and may even improve depressive symptoms that were present before apparent onset of the disease (Mindham, 1970).

**Perception of Emotion and Consequent Displayed Affect in PD**

Recently, Raskin et al. (1990) noted the suggestions of frontal lobe dysfunction in PD and asked researchers to examine whether PD patients exhibit deficits in the expression of emotion but not in the perception of emotion as has been predicted (e.g., Ross, 1985); such proposals suggest that the perception of emotion is localized to right-posterior structures and expression of emotion is localized to right-anterior structures.

Buck and Duffy (1980) found that although PD patients verbally express pleasure at seeing familiar people and distress at the sight of unpleasant slides, their emotional expressions are less apparent when the sound was turned off. However, these results should be interpreted with caution due to the small number of subjects (N = 9), the unspecified determination of medical diagnosis of PD, the lack of a description as to how the population was screened for dementia or depression, and the omission of an operational definition of how "affect" was assessed.

Borod and colleagues (Borod, Alpert, et al., 1989)
administered measures of expression and perception in both facial and vocal emotional channels to schizophrenics with flat affect, right brain-damaged subjects, PD patients, and normal controls. The PD population consisted of 6 nondementing patients with a mean age of 63.8 (SD 10.4 years). The subjects were asked to deliberately produce a range of facial emotional expressions, including both positive and negative ones. For the vocal expression, subjects were audiotaped while intoning sentences with the same seven emotions as used in the facial expression task. Intensity of each expression was rated on a 7 point Likert scale and accuracy was rated by indicating the emotion produced from a multiple-choice format, using all seven emotions (happiness, surprise, interest/excitement, sadness, anger, fear, and disgust). Analysis revealed that the schizophrenic group and the PD group show less intense facial expressions than right brain damaged or normal controls. It is important to note, however, that neither the schizophrenic group nor the PD group were screened for depressive symptomatology; therefore, the cause for their relatively increased flat affect over the other groups is unclear.

**Facial Expression in PD**

Masked facies is a hallmark symptom of PD. However, to this author's knowledge there has never been a study to
document degree of depression relative to degree of masked facies in PD. Additionally, few studies have investigated the perception and expression of emotion in PD patients. Given that there is a subgroup of PD patients who do not evidence depressive symptomatology, the question arises whether or not a subgroup exists among PD patients who have masked facies but who are not depressed.

PD patients are thought to display facial expressions accurately but with less intensity than normal controls (Borod, Alpert, et al., 1989). However, Brozgold (1988) found 2 of 3 PD patients to be more expressive in posed voluntary conditions than in spontaneous conditions whereas normal controls were found to be equally expressive in both conditions. Similarly, other researchers noted impaired facial expression in spontaneous conditions requiring that PD patients respond to affectively laden slides (Buck & Duffy, 1980; Katsikitis & Pilowsky, 1988).

Katsikitis and Pilowsky (1988) used a microcomputer-based method of studying facial expression in PD and normal control samples in response to viewing cartoons. Twelve measures (end lip raise, mouth width, mouth opening, mid top lip raise; mid low lip raise; top lip thickness; lower lip thickness; eye opening; top eyelid/iris intersect; lower eyelid/iris intersect; inner eyebrow separation; and mid eyebrow raise) were standardized by being divided by one or
two reference measures: the distance between the outer canthi of the eyes for "horizontal" measures and the length of the nose for "vertical" measures. Results found that, of differences among measures most pertinent to the smiling expression, only mouth opening reached significance; PD patients did not open their mouth to the same degree during a smile as the control group. Also, the control group was found to smile on the average significantly more than the PD group. Yet, the control group rated only one cartoon as funnier than the PD group, suggesting a disparity between PD patients' perception of emotion and expression of emotion. However, these results must be interpreted with great caution for the following reasons: (1) one severely disabled PD patient with Shy Drager's syndrome disease was included along with 8 milder idiopathic PD patients to form the PD group, (2) all PD patients were on medications (8 on L-Dopa, 7 on bromocriptine, amantadine, benztropine, or benzhexol), (3) as a group, the PD subjects had higher depression scores ("moderate" range on the Levine-Pilowsky Depression Questionnaire) than the NC group, and (4) no specifics were given as to how the video taped expressions were judged for expressiveness.

Another microcomputer-based study of facial expression, designed by Katsikitis and Pilowsky (1991), measured and compared smiling behavior in PD patients, patients with
Major Depression, and controls of comparable age. The same 12 measures as used in the previously mentioned study were derived. Again cartoons were rated as being amusing or not. Two judges with no previous training in the rating of facial expressions viewed the videotaped recordings of all sessions and selected the most animated smile for each participant for microcomputer analysis. Results revealed significant differences among groups for end-lip measure, mid-top lip measure, and mid-eyebrow measure. Overall, the severely depressed group had higher measures on all three indices than the PD or NC group. Although the PD and severely depressed groups were found to smile less frequently than the NC group, no significant difference was found among groups in the rating of "funniness" each subject gave each cartoon. When each group was considered separately no significant correlation between depression scores and face measures within the PD or NC populations were found, but significant negative association emerged between depression scores and mid-eyebrow measures in the severely depressed group.

Katsikitis and Pilowsky (1988, 1991) suggested that these findings support the hypothesis that the difference in smiling intensity between the Parkinsonian and control group is due to the unresponsiveness of the muscles around the mouth, specifically the levator labii superioris and
zygomaticus minor, which are involved in raising the upper lip. They further contended that these studies support and extend the findings to include the elevators of the outer corners of the mouth—the zygomaticus major, caninus, and upper buccinator muscles (Hjortsjo, 1969).

Again these results need to be interpreted with caution because all PD patients were on medications (all were on levodopa, 20% were on antidepressants, 30% were on bromocriptine, 10% were on amantadine); the length of illness among the PD group varied widely from 1 to 20 years; and the PD group fell into the moderately depressed range on the Levine-Pilowsky Depression Questionnaire whereas the severely depressed group fell in the severely depressed range and the NCs fell in the minimally depressed range. Also, the severely depressed subjects to which the PD were compared were all taking antidepressants and antipsychotic agents (60%) and sedatives (25%) and the length of current episode of depression for 18 individuals in the severely depressed group varied from 2 to 32 weeks (mean 11.1; SD 8.6) whereas one had major depression for one year. Thus, it is not clear whether the results attained for the PD group may be attributed to effects of masked facies as a result of the Parkinson's disease (as suggested by Katsikitis and Pilowsky), antidepressant medication, depression, or severity of PD illness. Similarly, within
the Major Depressed group, it is unclear whether the effects evidenced are due to antidepressant or antipsychotic medication, length of current depressive episode, or the depressive illness in general. In addition, a nondepressed rather than a mildly depressed NC group would have been the preferable comparison group, considering that the purpose of the study was to compare smiling behavior.

**Oral/Facial Impairment in PD**

Although the clinical neurological literature suggests two separate neuroanatomical systems for voluntary (cortical) versus spontaneous (subcortical) facial movements, the brain-behavior relationships of each of these systems have not to date been clearly delineated. However, PD patients have shown deficits in facial expression in both voluntary and spontaneous conditions as would be expected given that some cortical involvement along with subcortical involvement is known to have occurred in many PD individuals.

In an effort to clarify this issue, Brozgold (1988) compared parkinsonian patients with cortical and subcortical lesions in their ability to produce facial expressions under posed and spontaneous conditions. He found mixed results to provide support for the dissociation between posed and spontaneous expressions for cortical and subcortical lesioned groups. However, two of three of the parkinsonian
patients in the study, who were thought to have subcortical lesions, produced less expression in the spontaneous than the posed conditions.

Abbs, Hartmann, and Vishwanat (1987) implemented transducers providing isometric contraction force signals for tongue, lower lip, and mandibular elevation muscles on 6 patients with idiopathic PD. Results revealed all PD patients manifested greater instability in the lip, jaw, and tongue than seen with matched controls (Abbs, Hartmann, & Vishwanat, 1987). However, broad conclusions cannot be made from these results since the subject population was poorly described; there was no mention of screening criteria for dementia or depression; and the subjects' ages, stages of PD, and medication intake was not revealed.

Abbs et al. (1987) suggested that these results permit the hypothesis that certain PD motor symptoms are due to alterations in muscle afferent function. They suggested that tongue muscles, devoid of stretch reflexes, are most impaired, while jaw-closing muscles, with numerous spindles and a monosynaptic stretch reflex are least impaired. Thus, Abbs et al. purported that PD patients' motor impairments are independent of fusimotor or muscle afferent dysfunctions and not uniform across orofacial muscle groups. They contended that these data support the position that the movement impairments associated with PD are related to
aberrant basal ganglia inputs to the motor or somatic sensory cortices. However, they pointed out that the lip muscles, devoid of muscle spindles, manifest PD rigidity similar to that observed in the extremities. Other interpretations of this disproportionate tongue impairment include descending pathways from the basal ganglia directly to the brainstem. Several studies have concurred with this hypothesis due to indications that basal ganglia dysfunctions are consistent with PD difficulties in swallowing and upper airway respiratory problems (Kimura, 1973). Abbs et al. (1987) suggested that since orofacial motor impairments in PD patients are likely to be different from those manifest in the extremities, such orofacial movement disorders in PD patients may not respond to drug manipulation in the same way as the muscle groups of the extremities.

Weismer (1990) contested Abbs et al.'s (1987) results restating that Parkinson's patients are tremendously heterogeneous in the manifestations of their movement problems. For example, when studying 15 subjects with PD of unknown cause, Schneider and colleagues found jaw proprioception to be a good discriminator of PD's orofacial sensorimotor system but found that tongue sensation was not a good discriminator (Schneider, Shirley, Diamond, & Markham, 1986). It is important to note, however, that the
subjects' stage of PD varied from stage 1 to 3, all were receiving anticholinergic drugs during the experimental session, the age ranged from 47 to 83 (mean age 65.8; SD 9.5), and age and gender matched controls were not used. However, Schneider et al. (1986) pointed out that their finding that tongue sensation was not a good discriminator of PD may be due to the scoring system of the test; that is, the use of $0 = \text{good response}, 2 = \text{fair}, \text{and} 4 = \text{poor}$ may not have been sensitive or detailed enough to show differences which were subjectively more apparent.

During tactile stimulation, Schneider et al. (1986) found two-point discrimination thresholds in the midline and perioral (around the mouth) regions of the face to be significantly larger (greater distance between the two points) in PD subjects than in controls. However, there appeared to be no significant differences between PD and normal control groups in two-point discrimination thresholds on the cheek. These results, according to Schneider et al., suggested that as in lower-order animals, the human basal ganglia may be more sensitive to events occurring near the midline or perioral region of the face than to those more lateral of the face.

Striking results were also apparent on Schneider et al.'s (1986) tests of head targeting and head tracking. PD subjects had great difficulty using perioral somatosensory
information to make controlled head movements although they made normal spontaneous, visually guided, or verbally commanded head movements. Schneider et al. suggested these findings lead to the hypothesis that PD do not have a cervical motor impairment, per se, but rather a breakdown in the link between the facial somatosensory inputs and cervical motor output. They stated that in PD the gating process may be shutting down so that the appropriate sensory signals do not gain access to effector regions. That is, in PD the loss of nigrostriatal dopamine may enhance the inhibitory sensory gating function of the basal ganglia so that there is a greater than normal inhibition of the access of sensory information to relevant motor areas, resulting in a net motor hypoexcitability. Dopamine, then, may aid in the processing of such sensory information via the basal ganglia and regulate the access of this information to the relevant motor areas. With decreased dopaminergic function, sensory information may not be readily modulated by the internal gating and filtering system of the basal ganglia. In contrast, increased dopaminergic activity may cause sensory information to pass through the system too readily and may indiscriminately lower thresholds for movement possibly resulting in abnormal dyskinetic movements.
Facial Electromyography (EMG) in PD

In Relation to Speech Movements

Rigidity in PD has been inferred from EMG studies but often observed movement patterns have not been quantified. Because the labial muscles have special characteristics and a role in speech movements, EMG studies for these muscles may provide insight into the hypothesized causal relationship between hypokinesia and muscle rigidity. Hunker, et al. (1982) set out to obtain a quantitative index of labial muscle rigidity and to determine whether these rigidity values related to associated labial speech movement aberrations. A small sample (N = 4) of parkinsonian subjects (unspecified) with limb rigidity and hypokinesia served as subjects. Results revealed all 4 parkinsonian subjects had significantly greater lower lip stiffness than normal controls and 2 of 4 parkinsonian subjects had increased upper lip stiffness relative to controls. When there were abnormal levels in the stiffness of labial muscles, there was also increased EMG activity at rest. This background EMG activity increased when the lip was passively displaced in a direction antagonistic to the muscle contraction.

A similar response was observed in EMG during speech movements, which Hunker et al. (1982) suggested was due to antagonistic muscles being reflexively activated by the
generated speech movement. They suggested that these findings provide compelling evidence of a cause-effect relation between rigidity and some aspects of hypokinesia—especially reduced range of movement. They pinpoint a threefold reasoning why this may be so: (1) the facial muscles, at least in the area of the lips, do not have muscle spindles, tendon organs, or any known receptor mediating a monosynaptic reflex, (2) the existence of classical inhibitory motoneuron collaterals in the brainstem is questionable, and (3) reciprocal inhibition between antagonistic muscles does not operate in the brainstem as it does in the spinal cord (i.e., in the orofacial system, muscle pairs that work together for symmetric midline movements are antagonistic for lateral-medial movements). Thus, Hunker et al. contended that rigidity and hypokinesia in the facial muscles must result from mechanisms other than those previously hypothesized for the extremities (i.e., inappropriate fusimotor activation or loss of recurrent inhibition).

In Relation to Speech & Levodopa Therapy

Leanderson, et al. (1971, 1972) demonstrated similar effects on the articulatory function of the facial muscles as a result of levodopa medication. The most prominent effect of levodopa was the substantial reduction of the intense resting and background EMG activity, allowing for
the proper articulatory activity to be more readily discernible. The abnormally early onset and long duration of the activity were replaced by almost normal timing. Of the 5 tested before and after medication, the production of [p] was associated with a more normalized pattern following levodopa intake, displaying an obvious reciprocal activation of antagonistic muscles. Thus, these results suggest that there are characteristics which are common to the impairment of motor function of parkinsonian limb and labial musculature. It seems that in spite of important differences in functional anatomy (i.e., limb muscles are attached to the skeleton, perform joint movements, and contain many muscle spindles whereas labial muscles move soft tissues such as muscles and skin without necessarily having an attachment to bone and contain few or no muscle spindles) the type of disturbance in motor control seems to be similar in both kinds of muscle (Leanderson et al., 1972).

Leanderson et al., (1971) recorded EMG activity using concentric needle electrodes from labial muscles simultaneously with the acoustic signal of different utterances. The muscles chosen for study were the rounding/closing muscles (orbiculares oris superior inferior) and the lip-opening spreading muscles (lev. and depressor labii). These muscles normally constitute two
functionally antagonistic muscle groups. Seven parkinsonian patients with dysarthria were examined; two had undergone bilateral thalamotomy, three unilateral thalamotomy, and two had not been surgically treated. Those who had received unilateral thalamotomy were examined on the side ipsilateral to the surgery. All subjects were examined before and after levodopa treatment. Results revealed that dysarthria in parkinsonism may be correlated with a constant muscular hyperactivity, seriously interfering with the articulatory activity. Also, as seen by Leanderson et al. (1972), a disturbance in reciprocal muscular activation, manifested in a simultaneous contraction of opposing articulatory muscles was evident, confirming the hypothesis by Hunker et al. (1982) that a deficient reciprocal activation is the prime cause for misarticulation. Leanderson et al. (1971) noted that during levodopa treatment the poor facial expression became "lively" (no specifics) and the slow and stiff articulatory lip movements faster and smoother. The slurred pronunciation, especially of the plosive consonants, improved. These changes corresponded to a considerable decrease in EMG background and more normalized articulatory activity. Thus, treatment with levodopa resulted in the re-establishment of reciprocal muscular activation.

Leanderson, et al. (1972) compared the articulatory EMG activity of lip muscles in 12 subjects with parkinsonism and
dysarthria to that in normal speakers. Only 5 of the 12, however, were studied before and after levodopa treatment, and 6 of the 12 had undergone stereotaxic surgery on one side. They recorded the articulatory activity of the labial musculature polygraphically with thin concentric needle electrodes simultaneously with the speech signal. Six muscles in one-half of the face were investigated (M. lev. lab. sup., M. orb. oris sup., M. orb. oris inf., M. dep. lab. oris, M. dep. ang. oris, and M. Mentalis). It is important to note that they investigated the non-operated side of the 6 who received stereotaxic surgery, and the side of the face ipsilateral to the "most rigid and hypokinetic half of the body" (Leanderson et al., 1972, p. 74) in the 6 who did not undergo surgery.

Results revealed that although the audible articulatory performance was comparatively good in the test situation, EMG records exhibited marked and consistent abnormalities in all 12 subjects. Specifically, they suggested as did Hunker et al. (1982) that the prime factor for the misarticulation of labial stop consonants was a deficient reciprocal activation--the production of which demands rapidly alternating closing and opening speech gesture components. In this regard, clinical improvement in the motor function of limb muscles following stereotaxic surgery in parkinsonism has been shown to be electromyographically
correlated to a reestablishment of reciprocal activation as well as a reduction of hypertonicity (Ohye, Tsukahara, & Narabayashi, 1965).

Similar results were found by Nakano, Zubick, and Tyler (1973) in a double blind study that evaluated whether speech intelligibility and labial movement improved with levodopa therapy and whether this improvement indeed proved significantly better than that with other forms of medication. The population consisted of 18 PD patients ages 42 to 74 years, free of levodopa therapy, acting as their own control. Concentric Grass E-2 needle electrodes were inserted at six points about the mouth (facial muscle groups affected: levator labii superioris, zygomaticus minor, zygomaticus major, levator labii, superioris alaeque nasi, levator anguli oris, risorius, depressor anguli oris, depressor labii inferioris, and mentalis). Patients performed oral exercises including forming an entire smile, smiling on the left side of the mouth and then the right side, labial eversion, counting, and phoneme and diphthong repetition. Results found that although all 18 subjects preferred levodopa, two had better speech on placebo, one improved on both placebo and levodopa, one did not improve on either medication, and 14 benefitted from levodopa only. Their results indicated that significant improvement occurred in labial eversion, counting, phoneme and diphthong
repetition, and tonic base-line activity during both procyclidine and levodopa therapy when compared to placebo. Also, significant benefit in symmetry, amplitude, and frequency of labial movement occurred only with levodopa use.

**In Relation to Facial Expression**

Although failure of control of facial expression is one of the characteristic symptoms in patients with PD, the details of the pathophysiological abnormalities of the face in PD are still unclear. Electrical stimulation of the trigeminal nerve can elicit suppression of the voluntary contraction of the normal jaw-closing muscles. This suppression is usually called exteroceptive suppression, and consists of 2 suppressive components--early and late. Recently, Nakashima and colleagues studied exteroceptive suppression during voluntary contraction of the masseter and temporalis muscles, produced by electrical stimulation of the mental nerve, in 23 idiopathic PD subjects (mean age was 57.1, SD 2.1 years; PD stage 2 to 4) (Nakashima, Takahashi, Azumi, & Ishida, 1989). All subjects were receiving medication. It is important to note that the "most affected side was studied" in those with PD; yet, specifics as to how the most affected side was determined was not given. The background EMG activity prior to stimulation and the latency of suppression revealed no difference between PD patients
and normal age-matched controls. However, the duration and degree of suppression were lower in the PD patients than in the normal controls, suggesting that the reduction of exteroceptive suppression might, at least in part, play a role in the failure of motor control of the face in PD. These results further support the hypothesis that this reduction may result from the disturbed function of the descending volley from the basal ganglia (Nakashima et al., 1989).

Sandyk (1981) measured the reaction time (period of latency) and the successive reaction time (between two quick repetitive voluntary movements) of the facial eye (orbicularis oris) and masseter muscles following electrical stimulation from the periphery (thenar muscles) in 15 PD patients on medication and 5 NCs. They registered voluntary movements of the facial muscles bilaterally with superficial needle electrodes from the orbicularis oris muscles and recorded eye movements oculographically. Sandyk suggested that this method allows for the measurement of the potential difference between retina and cornea changes following an eye movement and can be recorded electromyographically. Results revealed that the voluntary motor responses following peripheral stimulation were delayed in the PD group, suggesting that cortical motor areas are involved in PD; that is, following peripheral stimulation, cortical
activity is inhibited for a relatively long time. This phenomenon was most often noted following two brisk repetitive voluntary muscle movements.

**Medication Effects on Facial Expression**

A significant improvement at the 5% level for akinesia, gait, speech disorder, and facial expression occurred in 15 PD subjects (14 idiopathic, 1 post-encephalic) when piribedil was added to levodopa in a double-blind, crossover study (Callagan, Fitzpatrick, & O'Mahony, 1975). Additionally, a more highly significant improvement at the 1% level for akinesia, facial expression, and finger dexterity occurred with piribedil and amantadine in this same patient population.

Kimura and Tsukue (1971) similarly found levodopa therapy to improve voluntary facial movements in 9 schizophrenic patients with drug-induced parkinsonism. Seven points on the face were chosen for study. Results indicated that in comparison with the control group, the mobility ratios of the male parkinsonian group before levodopa treatment were significantly impaired ($p<.05$) in their ability to wrinkle the forehead and knit the brows, and the mobility ratio on closing the eyes was poorly significant ($p<.05$). However, after 4 weeks of levodopa therapy the mobility ratios on the facial movements of the patients were normalized. Kimura and Tsukue suggested that
the mask-like expression of parkinsonism might be due to lack of the contraction power of the facial muscles.

To compare subjects' responses to bromocriptine with their response to previous optimal drug treatment including levodopa, Kartzinel, Shoulson, and Calne (1976) performed a double-blind crossover study using 12 patients with idiopathic PD (ages: 47-65, M 56.4; duration of illness: 5-16 years, M 9.1 years). A 26% improvement was noted with bromocriptine; rigidity, tremor, and facial expression showed the greatest improvement. Unfortunately, however, no specifics were given as to how improvement in facial expression was ascertained.

Focus of the Present Research

Definition of Relevant Terms

idiopathic Parkinson's disease: A common degenerative disorder of the brain in which dopamine—a neurotransmitter substance of the striatum—is depleted, presumably due to a loss of neurons in the substantia nigra and degeneration of the nigrostriatal tract to which they give rise. The term "idiopathic" implies that the cause of the disease is unknown. Symptoms include resting or static tremor, muscular rigidity, micrographia, akinesia, involuntary movements, disorders of posture, disorders of righting, absence of arm swinging, bradykinesia, and difficulty starting and stopping to walk.
bradykinesia: Abnormal slowness of movement.
dyskinesia: Impairment of the capacity for voluntary movements or the occurrence of involuntary movements.
rigidity: Muscle resistance to passive motion from simultaneous agonist and antagonist muscle group contractions.
masked facies: Rigidity of facial muscles causing the appearance of loss of emotional expression in the face.
subjective experience of emotion: An individual's personally perceived emotion; often determined along valence and arousal dimensions.
valence: "Valence refers to the organism's disposition to assume either an appetitive or defensive behavioral set" (Lang, Bradley, & Cuthbert, 1990, pg. 380). Also thought of as how happy or unhappy one feels when confronted with an emotional stimulus.
arousal: "Arousal refers to the organism's disposition to react with varying degrees of energy or force.... Arousal is considered to be an intensity factor" (Lang, et al., 1990, pg. 380). Also thought of as how excited or calm one feels when confronted with an emotional stimulus.
surface electromyography: Analysis of the electrical activity of muscles via electrodes placed directly on top of the skin over the muscle region of interest.
**EMG change score**: A score derived by subtracting the EMG rectified voltage previous to the presentation of a stimulus from the total rectified voltage recorded during the presentation of a stimulus. Also called change from rest score.

**zygomatic muscle region**: The muscle region of the face that is involved in pulling the outer corners of the lips upward as if in smiling.

**corrugator muscle region**: The muscle region of the face that is involved in pulling the eyebrows together as if in frowning.

**Hypotheses**

1. The subjective experience of felt emotion, defined by valence and arousal dimensions, in the PD group will be reduced relative to that in NCs.
   a. The pattern of valence ratings will be the same as that of NCs: Pleasurable emotion will be reported after viewing positively affect-laden slides and unpleasurable emotion will be reported after viewing negatively affect-laden slides.
   b. The pattern of arousal ratings will be the same as that of NCs: More intense (arousing) emotions will be reported after viewing positively and negatively affect-laden slides than after viewing neutral affect-laden slides.
2. The PD group's level of facial EMG activity in the zygomatic and corrugator muscle regions will be significantly attenuated (reduced) relative to that of NCs when viewing pleasant and unpleasant affect-laden slides.

   a. The zygomatic EMG pattern for the PD group will be the same as that of NCs: EMG activity in the zygomatic major muscle region will be significantly higher when viewing positively affect-laden slides than when viewing negatively affect-laden slides.

   b. The corrugator EMG pattern for the PD group will be the same as that of NCs: EMG activity in the corrugator muscle region will be significantly higher when viewing negatively affect-laden slides than when viewing positively affect-laden slides.

Secondary Hypothesis.

1. Facial EMG activity across all sites in the PD group will significantly increase in magnitude during "on drug" as opposed to "off drug" cycles.
CHAPTER 3

METHOD

An explanation of how the subjects were selected and screened for entrance into the study will be provided, followed by a thorough discussion of selected stimulus materials. Subsequently, an overview of the screening instruments and experimental assessment instruments will be given. Then the electronic apparatus and physiological response measurement used in the study will be explained.

Subjects

Nineteen subjects with idiopathic Parkinson's disease and 19 healthy control subjects participated in the study. An additional six PD subjects and six normal control subjects (NCs) were excluded from participation during or after a testing session: PD subjects--one became incontinent during the EMG portion of a session, one had poor eyesight, one was too fatigued "off" medication to attend to the tasks, and three obtained scores in the "severely depressed" range on the BDI; NC subjects--two became ill after testing session one and could not complete session two, one had poor eyesight, one reported significant neurological history not previously related, one had significant medical history not previously related, and one experienced computer failure. Each subject provided informed consent to participate in the study.
The groups were demographically matched on the basis of gender, age, and education level. All subjects were Caucasian. All NCs reported being right-handed whereas 14 PD patients reported being right-handed, 2 left-handed, and 3 ambidextrous. The length of time since initial PD diagnosis ranged from 2-28 years with a mean of 7.79 years.

The PD patients were primarily recruited from ongoing longitudinal studies conducted by the University of Arizona Department of Speech and Language, although a few were referred from local neurologists or recruited from advertisements. Most PD subjects (N = 14) had received a diagnosis of idiopathic PD after a thorough neurological examination by a staff neurologist at the University of Arizona Medical Center, medical history, and neuropsychological evaluation; a few PD patients (N = 5) retained for the study did not have longitudinal data but had received a diagnosis of idiopathic PD from either the movement specialist/neurologist at the University of Arizona Medical Center or their neurologist in private practice.

For the majority of PD subjects (N = 14) for whom longitudinal data were available, further screening took place in the areas of cognitive decline, depressive symptomatology, degree of masked facies, stage of PD, and level of independence. Specific assessment data analyzed included longitudinal scores (from a 1 to 3 year time
period) on the Mini-Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975); the Global Deterioration Scale (Reisberg, Ferris, & de Leon, 1982), a clinician-rated instrument based on clinician-subject interview; the Hamilton Psychiatric Rating Scale for Depression (Hamilton, 1960), a clinician-rated instrument resulting from clinician-subject interview; the Wisconsin Card Sorting Test (WCST) (Grant & Berg, 1948; Heaton, 1981), a test of set-shifting ability and conceptual ability; and the Unified Parkinson's Disease Rating Scale (UPDRS) (Fahn, Elton, et al., 1987). For inclusion into the study the PD subjects had to receive a stage 2 (bilateral disease, without impairment of balance), stage 2.5 (mild bilateral disease, with recovery on pull test), or stage 3 (mild PD to moderate bilateral disease, some postural instability, physically independent) rating on the Hoehn and Yahr Clinical Disability Scale (Hoehn & Yahr, 1967). Additionally, PD subjects had to show evidence of masked facies on at least one UPDRS since the start of the longitudinal study (as indicated by a score of 1 ("slight, could be normal poker face") or greater on item #19 on the UPDRS and be free of signs of progressive cognitive decline and depressive symptoms. All potential subjects that met the screening criteria were contacted for possible participation in the present study.
For the few PD patients (N = 5) for whom longitudinal data were not available, a determination was made at the time of a phone screen as to whether or not they were to be included for further on site screening. All potential subjects were screened by phone in an effort to address relevant medical/psychiatric changes that may have occurred in the past year. Only those with no confounding neurological or psychiatric disorders and with adequate vision and hearing were chosen for further screening.

Once subjects agreed to participate, the MMSE was administered to screen for cognitive decline; only those who received a score of 26 or above were included in the study. It was important to rule out significant cognitive decline given that some PD patients may have a superimposed dementia as seen in Alzheimer's disease (i.e., cortical involvement) (see Lieberman and colleagues, 1973) or a dementing illness that involves subcortical as well as cortical neurodegenerative changes (Alvord, Forno, Kusske, Kauffman, Rhodes, & Goetowski, 1974; Boller, Mizutani, Roessman, & Gambetti, 1980; Hakim & Mathieson, 1979; Whitehouse, et al., 1982).

The Beck Depression Inventory (BDI) (Beck, 1987; Beck & Beck, 1972) and the UPDRS were administered at each testing session to screen for depression and motoric changes since the subject's last neurological evaluation (obtained via
their participation in the longitudinal study or with their private practitioner). Only subjects in PD stage 2, 3, or 4 who received 13 or less on one of two BDI administrations were retained for participation in the current study; all PD patients scored 9 or less on one of the BDI administrations except for 4 PD patients who scored 11-13 on one of the BDI administrations.

Demographically (i.e., age, sex, and education) matched NCs were recruited from tennis centers, senior centers, residential communities, and senior events in Tucson, Arizona, and the surrounding community. The screening process and cutoff scores for inclusion into the study (i.e., historical phone interview, MMSE, and BDI) paralleled that for the PD subjects.

Additional exclusionary criteria for PD and NC subject selection follows: (1) the presence of significant facial paralysis on voluntary facial movement tasks, (2) the presence of visual and/or auditory deficits so severe as to compromise task performance (as indicated by impaired performance on the Sloan Letters (Sloan, 1961) and/or the Repetition Test of the Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1972)) and (3) a primary language other than English. For the Sloan vision screen, subjects were asked to read lines of letters (depending on their accuracy on each line) using first the right eye and then the left
eye from a distance of 16 inches. Only those with at least 20/50 estimated vision were retained for the study.

**Materials**

**Stimulus Materials: International Affective Picture System**

This picture system (Lang & Greenwald, 1988; Lang, Greenwald, & Bradley, 1988, 1990; Lange Ohman, & Vaitl, 1988) was developed to ascertain how pleasant/unpleasant (valence), calm/excited (arousal), and submissive/dominated (dominance) each slide made an individual feel (i.e., subjective experience of emotion). In the current study, the dominance factor was not included because it has been shown to be relatively insignificant in factor analytic studies. Lang and colleagues kept several considerations in mind when designing the stimuli: (1) slides were included similar to those used in conditioning studies of fear and preparedness, (2) an attempt was made to "expand the affective space" by carefully choosing slide contents which might be expected to fall in more remote areas of the valence-arousal space, (3) a relatively even proportion of human, animal, and inanimate object stimuli were used, (4) several exemplars of varying stimulus complexity, color, and composition were used within a particular content class, (5) a broad representation of a range of affective materials using the slide medium was attempted, (6) all slides are in color, and (7) all slides, whether of human, animal, or
object content, always contain a single example of the stimulus which is placed in the foreground with few competing stimuli.

The overall aim of the stimulus composition was to increase contrast between figure and background, thus easing perceptual recognition. There are however, minor physical variations in complexity, luminance, and image size.

A tripartite classification of stimulus situations and behavior resulted: (1) Animals: predatory/ferocious--innocuous--immature/domestic, (2) Humans: dangerous--innocuous--pleasant/erotic (subclassified into faces: mutilated, angry, neutral, happy; bodies: mutilated, healthy, nudes), and (3) Non-communicative: human artifacts (weapons, useful technology) and environmental stimuli (urban and rural scenery, pollution).

In initial evaluation of the Affective Picture System, Lang and colleagues had subjects report their subjective experience of emotion by moving a handle to denote which of 5 manikins (Self-Assessment Manikin, Lang, 1980) on a computerized screen depicted their felt emotion, or by marking a paper-and-pencil adaptation of the Self-Assessment Manikin designed to minimize language-mediated evaluative differences across countries. Each of the two dimensions was ordinally scaled with 5 figures, with the possibility of also placing an "X" between figures (see Figure 1). Thus,
Figure 1. Paper and pencil adaptation of the Self-Assessment Manikin (Lang, 1980) used to rate valence and arousal dimensions.
each scale ranges from 1-9.

For the current study, an electronic box (11" x 17") was devised in which the SAM figures were enlarged, and electronic buttons were positioned underneath and between the SAM figures to allow for the same scale of 1-9. The box was designed to illuminate during the rating period following each slide viewing period. Order of the rating dimensions was not randomized across trials as was done for the norming sample; rather the subjects were instructed to rate the valence dimension and then the arousal dimension. This order remained constant for all slides in Set A and Set B. This was a decided compromise, given that many of the subjects are elderly and may become confused by the trial by trial switch in order.

For the current study, two sets of slide images, 27 each (Set A and Set B), were chosen from the complete IAPS set (240) based on significant valence and arousal ratings obtained from prior validation studies. Eighteen of the most valent and most arousing slides (9 pleasant, 9 unpleasant) for both males and females were chosen with an attempt to match eighteen additional slides by valence, arousal, and slide content. Eighteen slides with normatively-determined neutral valence and arousal were chosen as well. However, because the original IAPS was normed on college students, an attempt was made to include
several slides likely relevant to older adults (e.g., surgery). Thus, Set A and Set B contained 27 slides each--9 pleasant, 9 neutral, and 9 unpleasant.

Figures 2 and 3 illustrate the similar distribution of the 27 slides for Set A and Set B, respectively, relative to valence/arousal quadrants across positive neutral and negative slide content. Figures 4 and 5 show the similar distribution of slide stimuli for Set A and Set B, respectively, relative to sex across positive, neutral, and negative slide content. One-half of the PD subjects and one-half of the NCs were randomly chosen to view Set A during their first testing session and similarly, the other half of each respective group were randomly selected to view Set B. Then each subject viewed the other stimulus set at his/her second testing. The slides within each set were ordered in randomly derived sets of three (e.g., one pleasant, one neutral, one unpleasant; one neutral, one pleasant, one unpleasant, etc.).

Neuropsychological Testing Instruments

Screening Instruments.

1. Mini-Mental State Examination (MMSE). Each subject was administered the MMSE once--at the end of their "on" drug testing session. The MMSE was developed as a bedside test that could be used to detect cognitive impairment. Decade norms for 194 healthy individuals ages 40-89, developed by
Figure 2. Overall coordinates for valence and arousal dimensions for slide set A.

Figure 3. Overall coordinates for valence and arousal dimensions for slide set B.
Figure 4. Male and female coordinates for valence and arousal dimensions for slide set A.

Figure 5. Male and female coordinates for valence and arousal dimensions for slide set B.
Bleecker, Bolla-Wilson, Kawas, and Agnew (1988), were used for screening purposes in the current study.

Bleecker and colleagues found MMSE scores to range from 25-30 for age groups 50-59 and 60-69 and scores to range from 26-30 for the 70-79 age group. Therefore, a cutoff score of 26 was determined for inclusion into the present study.

2. Long Form of the Beck Depression Inventory (BDI). The purpose of the BDI is to screen for depression by self-report statements. The subject checks 21 four-choice statements. Statements refer to areas of sadness, pessimism/discouragement, sense of failure, dissatisfaction, guilt, self-dislike, suicidal ideation, social withdrawal, indecisiveness, unattractiveness, work inhibition, fatigability, and loss of appetite (among others). A score of 0-9 indicates no depressive symptomatology (Beck, 1987). The BDI was used here for two reasons: (1) the BDI has been shown to have acceptable validity and reliability in the elderly (Gallagher, Breckenridge, Steinmetz, & Thompson, 1983; Gallagher, Nies, & Thompson, 1982), and (2) the BDI has been used extensively in studies using facial electromyography in depressed and nondepressed individuals, allowing for greater comparisons with data resulting from the present study.

3. SLOAN LETTERS. This is a test of visual acuity to be
administered at a distance of 16 inches. Subjects were instructed to read successive lines of letters from the alphabet that decreased in size in an effort to determine their left and right visual acuity. For inclusion into the study, subjects had to evidence at least an estimated 20/50 visual acuity.

4. **Repetition Test of the BDAE.** Originally designed to detect speech/language deficits, this test requires the subject to repeat high and low probability phrases. The phrases are initially stated by an examiner who is positioned in front of the subject, allowing for full view of the examiner's lips. For purposes of the current study, only high frequency phrases were given, to gain a global index of the subjects' hearing ability.

5. **Voluntary Facial Commands.** To assure that subjects were free of significant facial paralysis, all PD subjects' and NCs' voluntary facial movement was assessed at both testing sessions using a modified version of Nelson's (1974) neurological examination. This involved asking each subject to wrinkle the forehead, pull the eyebrows together, close the eyelids tightly, wink, open the mouth, pull back the mouth, raise the outer corners of the lips, pull down the corners of the lips, blow out the cheeks, pucker the lips, wrinkle the nose, and whistle.
Testing Instruments. The following instruments were administered to both subject groups (PD and NC) at each of two testing sessions:

1. **Profile of Mood States (POMS).** The POMS (McNair, Lorr, & Droppleman, 1992) is an instrument consisting of 65 five-point adjective rating scales that the subject rates so that his/her current mood state may be assessed. The POMS covers six mood states, including depression, and is reported to be sensitive to drug treatment in the elderly (McNair, 1979). This instrument has been used as a measure of mood states in psychiatric outpatients and as a method for assessing changes in such patients.

2. **Unified Parkinson's Disease Rating Scale (UPDRS).** The UPDRS (Fahn, et al., 1987) had been administered to most of the subjects once a year by a neurologist over a one to three year period and was given by the investigator of the current study at both testing situations. The UPDRS is a clinician-rated instrument, resulting from an interview with the subject. There are four main components: (1) mentation, behavior, and mood, (2) activities of daily living, in which "best" and "worst" scores are given based on "on/off" PD medication effects; (3) motor examination, with emphasis on lateral differences, if any, and (4) complications of therapy in the past week. Stage of PD disease (Modified Hoehn and Yahr Staging) and degree of independence (Modified
Schwab and England Activities of Daily Living) is then determined from the UPDRS scores.

**Apparatus and Physiological Response Measurement**

Valence and arousal ratings were obtained using a computerized adaptation of the paper-and-pencil version of the Self-Assessment Manikin (SAM; Lang, 1980). That is, the 5 figures representing valence were positioned above 5 figures for arousal, and mounted on an 11" x 17" platform box which was illuminated during rating periods. There were 9 buttons (one under each of the 5 figures and one between each of the figures), designating a rating from 9 to 1 (happy to unhappy) for the valence dimension, and 9 buttons with a rating from 9 to 1 (excited to calm) for the arousal dimension. The device was located 18 inches in front of the subject at a 45 degree angle, and the slides were viewed from a distance of 9 feet.

Slide duration was controlled by a computerized script that automatically advanced the slide after 6 seconds of exposure. Upon slide advancement a blank screen was viewed during the rating period. Tape recorded and digitized instructions, under computer control, accompanied the slide showing. EMG physiological signals were sampled at 10 Hz for 5s before slide onset, for 6s during picture presentation, and for 5s after slide offset. Surface facial EMG activity was recorded bilaterally over the corrugator
and zygomatic major muscle regions, using Beckman silver/silver chloride sensors (diameter of inner circumference = 1 cm), through J & J M-501 bioamplifiers to an IBM PS/2 286 computer. Although surface electrodes have a wide pick-up area (Basmajian, 1974, p.29), it was not felt that the present study justified the greater discomfort and risk produced by needle electrodes. EMG recording was referenced to a common electrode placed adjacent to two EMG electrodes at each site (see Figure 6); inter-electrode distance was 1 cm. Standard skin and electrode preparation (as outlined by the J & J manual) were followed; skin was cleaned and abraded, and electrodes were attached with adhesive collars, using highly conductive ECG Electrolyte cream (Sigma Creme).

Signals were narrow bandpass filtered from 100 Hz to 200 Hz and rectified with a range of from 0 to 100 microvolts. Change scores were calculated separately for EMG activity at each facial muscle site by subtracting the activity during the 4th second (of 5 seconds) preceding picture onset (baseline) from the average response during the 6s picture viewing interval. The 5th second before picture onset was not used as baseline because the noise caused by the slide projector during advancement of the next slide during this second decidedly confounded baseline measures (e.g., eye blink or clearing of throat seemed to
Figure 6. Location of bilateral surface EMG electrode placement.
occur often).

Procedure

The PD subjects participated in two testing sessions: one "on" parkinsonism medication--about one hour after medication intake--and one "off" PD medication--at least 12 hours after abstention. "On"/"off" drug trials were deemed a necessary component of the study design for several reasons: (1) the variable ability of PD individuals to voluntarily move facial muscles and speak relative to levodopa therapy in PD (cited previously), (2) the "on-off" phenomenon or akinesia paradox that may occur during prolonged L-Dopa therapy (Claveria, Calne, & Allen, 1973), and (3) the possible laterality of EMG effects due to levodopa therapy (see De Vito, Riklan, & Misiak, 1972 who noted right surface EMG ratings from the sternocleidomastoid muscles of the neck were significantly lower for post levodopa than for pre levodopa groups but found no significant difference between pre- and post levodopa groups with respect to left EMG).

Because it was necessary to have all PD "off" drug sessions first thing in the morning, the on/off drug trial for each PD patient was counterbalanced: one-half of the PD patients had testing session one "on" drug, and one-half of the group had testing session one "off" drug. Thus, time of day and drug on/off condition were counterbalanced for the PD group. Matched normal controls were exposed to two
counterbalanced testing sessions, as well, even though no drug was administered. However, some of the normal controls underwent both testing sessions in the afternoon due to their work schedules. The two testing sessions were separated by a one day waiting period in most instances. Within each group there was also counterbalancing relative to which slide set (A or B) was viewed at the first testing session.

Subjects sat in a recliner chair in a small (8 x 10 feet), shielded, sound-attenuated, temperature-controlled, dimly lit room during the attachment of the electrodes by this experimenter and during the EMG experimental manipulation. The experimenter sat out of the subject's peripheral vision (behind and to the subject's right) during the spontaneous EMG manipulation. The slide image (4 feet x 3 feet) was projected 9 feet from the subject's eyes.

Prior to electrode attachment subjects were administered the POMS to ascertain their current mood state and to allow time for familiarity with the surroundings. After attachment of sensors, the subject listened to tape recorded and digitized instructions; he/she was told that pictures differing in emotional content would be displayed, and that each picture should be attended to the entire time it is exposed on the screen. After picture offset, the taped and digitized instructions asked the subject to rate
each picture on both SAM dimensions (valence and arousal) and then to relax between pictures. Subjects were instructed to select a valence and arousal rating of 1-9 for each slide by pushing one of 9 buttons corresponding to 5 illuminated figures as well as 4 "between figures" spaces for the valence dimension and for the arousal dimension. Also, PD subjects were instructed to occasionally blink between trials in an effort to lubricate their eyes and eliminate any blurring of their vision that may result from lack of blinking.

Each slide was presented for 6s (controlled by computer program and automatic slide advance) and a variable interval of 20s-35s occurred between picture presentations to allow individuals the time needed to rate their self-reported emotion and have EMG activity return to baseline. A 2 minute resting baseline was initiated before the picture series to facilitate laboratory adaptation, and two neutral pictures served as practice stimuli before either the 27 picture set A or set B began.

The 27 slides for set A and the 27 slides for set B were grouped into nine blocks of 3 slides each, consisting of 1 unpleasant (negative), 1 neutral, and 1 pleasant (positive) slide randomly arranged. The order of blocks in set A and set B were arranged in three varied orders; thus, subjects viewed one of three orders for slide set A and for
slide set B. The order of slides in sets A and B were counterbalanced across subjects and testing sessions.

Following the 27 picture trials for testing session one and testing session two ("on drug" or "off drug"), the PD subjects were administered the movement portion of the Unified Parkinson's Disease Rating Scale to validate PD symptoms. Normal control subjects were administered the UPDRS, as well, to allow for comparison.

In an effort to screen for facial paralysis the following voluntary facial/eye commands were given, and performance was subjectively rated by the experimenter: wrinkle forehead, pull the eyebrows together, close eyelids tightly, wink, open the mouth, pull back the mouth, raise the outer corners of the lips, pull down the corners of the lips, blow out the cheeks, pucker the lips, wrinkle the nose, whistle, and make repetitive eye blinks. Each movement was rated according to the following scale which parallels that used in the UPDRS: 0 = normal; 1 = minimal reduction in movement but could be normal; 2 = slight but definitely abnormal diminution of movement; 3 = moderate reduction in movement; 4 = no movement. Normal controls followed this course, as well, for consistency of method across populations.

At the end of testing session one and two the PD subjects were administered the Beck Depression Inventory
(BDI) to screen for degree of self-reported depressive symptoms relative to levodopa medication manipulation. Normal control subjects were administered the BDI at the end of each testing session, as well, for group comparison purposes. The decision to screen subjects for depression at the end of the experimental session was made so that their mood state at the beginning of the session would not be unwantingly modified by having them focus on responses to questions relating to depressive symptomatology.

At the end of the "on" drug testing session for PD subjects and the end of the "mock" "on" drug testing session for NCs, the Mini-Mental State Examination was administered to screen for cognitive decline. Data from those subjects who did not meet criteria for depression and/or cognitive decline were not analyzed for the current study.
CHAPTER 4
RESULTS

Descriptive characteristics for the Parkinson's (PD) and normal control (NC) groups will be provided, followed by an explanation of how each group's mental state and mood state was analyzed. Subsequently, validation of PD motor symptoms among the PD group will be explained. Then results from testing the primary and secondary hypotheses will be related.

Descriptive Statistics

The means and standard deviations for age and education of the PD and NC groups are presented in Table 1. A two-way analysis of variance (ANOVA) with group and sex as between subjects factors was computed for the dependent variables of both age and education. As expected, given the pairwise matching procedure employed for selection of control subjects, no significant main effects for age or for education was found. Also, no significant interaction effect between group and sex for age or for education were found.

Mental State vs. Mood State

The means and standard deviations for the Mini-Mental State Examination (MMSE) of the PD and NC groups are also presented in Table 1. Analysis, using a two-way ANOVA (group x sex) revealed a small but significant main effect
Table 1. Means and Standard Deviations for Descriptive Characteristics of the Parkinson's (PD) Group and Normal Control (NC) Groups by Sex.

<table>
<thead>
<tr>
<th></th>
<th>PD</th>
<th>SD</th>
<th>NC</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>71.37</td>
<td>7.12</td>
<td>70.79</td>
<td>6.53</td>
</tr>
<tr>
<td>Ed</td>
<td>15.29</td>
<td>2.62</td>
<td>15.08</td>
<td>2.97</td>
</tr>
<tr>
<td>MMSE</td>
<td>28.58</td>
<td>1.56</td>
<td>29.42</td>
<td>1.00</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>68.52</td>
<td>8.50</td>
<td>66.08</td>
<td>9.29</td>
</tr>
<tr>
<td>Ed</td>
<td>14.93</td>
<td>2.17</td>
<td>16.57</td>
<td>2.30</td>
</tr>
<tr>
<td>MMSE</td>
<td>29.00</td>
<td>1.15</td>
<td>29.71</td>
<td>0.49</td>
</tr>
</tbody>
</table>

**Note.** Ed = education in years; MMSE = Mini-Mental State Examination.
for group, $F(1,36) = 3.81, p < .0592$, with the PD group scoring lower than the NC group. No significant main effect for sex or interaction effect between group and sex was found.

To ascertain levodopa medication effects on self-reported depressive symptomatology, a two-way ANOVA (group x on/off drug condition) was computed, revealing a significant main effect for group, $F(1,36) = 7.83, p < .0082$. Note that the NC group underwent a second testing session to correspond with the PD's "on" drug session. No significant main effect for on/off drug condition or interaction effect between group and on/off drug condition was found.

The means, standard deviations, and significance levels for the Beck Depression Inventory (BDI) scores, comparing subject groups within each drug condition are presented in Table 2. Separate one-way ANOVAs with group as the between subjects factor were computed for the "on" BDI scores and for the "off" BDI scores. A significant main effect for group relative to each drug condition was found: "on" drug: $F(1,36) = 8.40, p < .0064$; "off" drug: $F(1,36) = 6.55, p < .0148$. Inspection of group means reveals the PD group scored higher on the BDI than the NCs on both the "on" and "off" drug condition.

To ascertain whether differences in mood state existed between the groups relative to drug condition, a two-way
Table 2. Means, Standard Deviations, and Significance Levels for the Beck Depression Inventory (BDI) for the Parkinson's (PD) and Normal Control (NC) Groups Relative to Drug Condition.

<table>
<thead>
<tr>
<th></th>
<th>PD</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td>DF</td>
<td>F</td>
</tr>
<tr>
<td>BDI-ON</td>
<td>8.11</td>
<td>3.77</td>
<td>4.58</td>
<td>3.73</td>
<td>36</td>
<td>8.40</td>
</tr>
<tr>
<td>BDI-OFF</td>
<td>8.95</td>
<td>5.79</td>
<td>4.84</td>
<td>3.92</td>
<td>36</td>
<td>6.55</td>
</tr>
</tbody>
</table>

Note. NC subjects underwent a second testing session for comparison of the PD "on" drug testing session.

* = Significant Difference p < .05
ANOVA (group x on/off drug condition) was computed, indicating a significant main effect for group for the following scales: Tension/Anxiety, $F(1,36) = 21.70$, $p < .0001$; Fatigue, $F(1,36) = 4.90$, $p < .0333$; Vigor, $F(1,36) = 5.94$, $p < .0199$; Confusion-Bewilderment, $F(1,36) = 7.01$, $p < .0120$; and Total Mood Disturbance, $F(1,36) = 5.69$, $p < .0225$. Of these scales, no significant main effect of on/off drug condition was noted, although significance was approached for the Confusion-Bewilderment scale, $F(1,36) = 3.75$, $p < .0607$. A significant interaction effect between group and on/off drug condition was found for the Tension/Anxiety scale, $F(1,36) = 5.29$, $p < .0274$, and the Vigor scale, $F(1,36) = 4.45$, $p < .0420$. In contrast, the Depression-Dejection and Anger-Hostility scales showed no significant main effects for group or for on/off drug condition or significant interaction effects between group and on/off drug condition.

Means and standard deviations for individual Profile of Mood State (POMS) scale scores for each group relative to drug condition are presented in Appendix A. Separate one-way ANOVAs with group as the between subjects factor were computed on each of the individual POMS' scales for the "on" drug testing session and for the "off" drug testing session. Results suggest that although the PD group felt significantly more tense and confused than the NC group--irrespective of drug condition--the PD group did not feel
significantly more depressed or angry than the NC group. Also, the results suggest that the PD group had less energy and felt more fatigued when "off" their levodopa medication than when "on" medication.

**Validation of PD Motor Symptoms on the UPDRS**

Analysis of the UPDRS items confirm the symptoms of PD in the PD group, including masked facies. Separate three-way ANOVAs (group x on/off drug condition x right/left side of body) were computed for each UPDRS item. The following items showed expected significant main effects for group: tremor at rest (for right and left arm), rigidity (for right and left arm and leg), finger taps (for right and left hand), hand movements (for right and left hand), alternating hand movements (for right and left hand), leg agility (for right and left leg), and body bradykinesia (for right and left side of body) (see Appendix B for significance levels). Review of within group means suggests that the PD group had more difficulty than the NCs performing each task. The body bradykinesia task showed a significant main effect for on/off drug condition, $F(1,36) = 4.62, p<.0387$, significant interaction effects between group and on/off drug condition, $F(1,36) = 11.08, p<.0021$, and significant interaction effects between group, right/left side of body, and on/off drug condition, $F(1,36), p<.0184$. No other significant main effects or interaction effects were found relative to the
other movement tasks.

To ascertain whether the groups differed in masked facies, a two-way ANOVA (group x on/off drug condition) was computed for item #19--facial expression. A highly significant main effect for group, \( F(1,36) = 35.70, p < .0001 \), was found. No significant main effect for on/off drug condition or significant interaction effect between group and on/off drug condition was found. Review of the means (PD: \( M = 1.6, SD = 0.50 \); NC: \( M = 0.60, SD = 0.60 \)) confirms that the PD group displayed less facial expression than the NCs.

**Primary and Secondary Hypotheses Tested**

A mixed model multivariate analysis of variance (MANOVA) design was used for testing the primary and secondary hypotheses. Because of more concern for the possibility of a Type II error over a Type I error for most hypotheses, all statistical analyses used the criterion of a .05 alpha level.

**Primary Hypothesis--Subjective Experience of Emotion**

**Collapsing of Slide Set A & Slide Set B Data.** For an explanation of how positive (P), neutral (O), and negative (N) slide types were derived see Appendix C. Inspection of means and standard deviations for each group's valence ratings relative to slide type for Set A and B are provided in Table 3. A three-way ANOVA (2x3x2) (group x slide type (PON) x slide set) was computed to ascertain whether
Table 3. Means and Standard Deviations for Valence Ratings by Slide Type (PON) and Slide Set (A, B) for the Parkinson's (PD) and Normal Control (NC) Groups.

<table>
<thead>
<tr>
<th></th>
<th>PD</th>
<th></th>
<th>NC</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>SET A</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>7.09</td>
<td>.66</td>
<td>7.42</td>
<td>.76</td>
</tr>
<tr>
<td>O</td>
<td>4.95</td>
<td>.57</td>
<td>5.06</td>
<td>.58</td>
</tr>
<tr>
<td>N</td>
<td>2.21</td>
<td>.58</td>
<td>2.43</td>
<td>.79</td>
</tr>
<tr>
<td>SET B</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>6.96</td>
<td>.66</td>
<td>7.03</td>
<td>1.01</td>
</tr>
<tr>
<td>O</td>
<td>4.58</td>
<td>.69</td>
<td>5.16</td>
<td>.76</td>
</tr>
<tr>
<td>N</td>
<td>2.07</td>
<td>.67</td>
<td>2.38</td>
<td>.82</td>
</tr>
</tbody>
</table>

Note. P = Positive, O = Neutral, N = Negative. Scale: 1-9 with 1 = unhappy and 9 = unhappy.
significant group slide type or slide set differences existed in perceived valence. No significant main effect for group was found. However, a significant main effect for set was found, $F(1,36) = 8.09$, $p<.0073$, with set A evoking slightly higher valence ratings than set B. Although no significant interactions were found between group and slide set, a significant three-way interaction effect between group, type of slide, and slide set was evident, $F(1,36) = 4.07$, $p<.0212$.

The similarity of mean arousal ratings relative to slide type (PON) across groups and slide sets may be ascertained by inspection of the means and standard deviations in Table 4. A three-way ANOVA (2x3x2) (group x slide type (PON) x slide set (A or B)) found no significant main effect for group or for set, and no significant interaction effect between group, slide type, and slide set. Thus, results demonstrate that the PD and NC groups had the same pattern of arousal for slide set A and for set B.

Data for slide set A and B were collapsed for all remaining analyses for the following reasons: (1) all subjects viewed both slide sets, (2) the presentation of slide sets was counterbalanced across the "on" drug and "off" drug sessions, and (3) significant group differences by slide set interactions were not found for the valence or
Table 4. Means and Standard Deviations of Arousal Ratings by Slide Type (PON) and Slide Set (A, B) for the Parkinson's (PD) and Normal Control (NC) Groups.

<table>
<thead>
<tr>
<th></th>
<th>PD SET A</th>
<th></th>
<th>PD SET B</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>PD A</td>
<td>5.28</td>
<td>1.68</td>
<td>5.09</td>
<td>1.68</td>
</tr>
<tr>
<td>O</td>
<td>3.37</td>
<td>1.13</td>
<td>3.35</td>
<td>1.40</td>
</tr>
<tr>
<td>N</td>
<td>6.33</td>
<td>1.23</td>
<td>6.19</td>
<td>1.04</td>
</tr>
<tr>
<td>PD B</td>
<td>5.52</td>
<td>1.45</td>
<td>5.10</td>
<td>1.78</td>
</tr>
<tr>
<td>O</td>
<td>4.28</td>
<td>1.23</td>
<td>3.95</td>
<td>1.37</td>
</tr>
<tr>
<td>N</td>
<td>6.50</td>
<td>1.81</td>
<td>6.06</td>
<td>1.81</td>
</tr>
</tbody>
</table>

Note. P = Positive, O = Neutral, N = Negative. Scale: 1-9 with 1 = calm and 9 = excited/aroused.
arousal dimensions. Subsequently, mean valence ratings (collapsing across slide sets A and B) for the positive, neutral, and negative slides were computed, yielding three mean valence ratings for drug "on" and three mean valence ratings for drug "off" for each individual. This same procedure was applied to mean arousal ratings yielding six mean arousal ratings for each individual.

**Valence.** Table 5 shows the means and standard deviations for each group's valence ratings for each slide type (PON). To test the hypothesis that PD patients would report significantly less valent emotions than NCs, a three-way ANOVA (2x2x3) (group x on/off drug condition x slide type (PON)) was computed. No significant main effect was found for group or for on/off drug condition although the expected significant main effect for slide type, $F(2,72) = .49, p<.0000$, was found. In addition, no significant two or three-way interaction effects were found (see the graphic representation of group mean valence ratings relative to drug condition and slide type in Appendix D).

Review of each group's mean valence ratings by slide type (PON) and inspection of Figure 7 shows the expected valence pattern for both the PD and NC groups. Both groups rated positive slides as evoking "happy" feelings (towards 9 on the valence rating scale) and negative slides as evoking "unhappy" feelings (towards 1 on the valence rating scale).
Table 5. Means and Standard Deviations of Valence Ratings by Slide Type (PON) for the Parkinson's (PD) and Normal Control (NC) Groups.

<table>
<thead>
<tr>
<th></th>
<th>PD</th>
<th></th>
<th>NC</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Positive</td>
<td>7.15</td>
<td>.51</td>
<td>7.20</td>
<td>.88</td>
</tr>
<tr>
<td>Neutral</td>
<td>4.76</td>
<td>.58</td>
<td>5.18</td>
<td>.67</td>
</tr>
<tr>
<td>Negative</td>
<td>2.13</td>
<td>.58</td>
<td>2.35</td>
<td>.81</td>
</tr>
</tbody>
</table>
Figure 7. Mean valence ratings by group and slide type (PON).
**Arousal.** Means and standard deviations for each group's arousal ratings for each slide type (PON) are presented in Table 6. To test the hypothesis that the PD patients would report significantly less arousing emotion than NCs in response to affect laden slides, a three-way ANOVA (2x2x3) (group x on/off drug condition x slide type (PON)) was computed. No significant main effect was found for group or for on/off drug condition, although the expected significant main effect for slide type was found, $F(2,72) = 53.14, p<.0000$. Also, no significant two or three-way interaction effects were found (see graphic representation of group mean arousal ratings relative to drug condition and slide type in Appendix E).

Review of each group's mean arousal ratings by slide type (PON) and inspection of Figure 8 shows the expected arousal pattern for both the PD and NC groups. Both groups rated positive and negative slides as being more arousing (towards 9 on the arousal rating scale) than neutral slides.

**Primary Hypothesis--Facial EMG Activity**

In order to test the primary hypothesis that facial EMG activity in the zygomatic and corrugator muscle regions would be reduced among the PD group relative to the NC group, a series of two-way ANOVAs (group x slide type (PON)) were computed. The dependent variable for each ANOVA was either zygomatic or corrugator facial EMG activity as
Table 6. Means and Standard Deviations of Arousal Ratings by Slide Type (PON) for the Parkinson's (PD) and Normal Control (NC) Groups.

<table>
<thead>
<tr>
<th>Type</th>
<th>PD</th>
<th>NC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Positive</td>
<td>5.39</td>
<td>1.54</td>
</tr>
<tr>
<td>Neutral</td>
<td>3.81</td>
<td>1.02</td>
</tr>
<tr>
<td>Negative</td>
<td>6.44</td>
<td>1.73</td>
</tr>
</tbody>
</table>
Figure 8. Mean arousal ratings by group and slide type (PON).
defined by the change from rest scores for each muscle group. For each subject, an average change score was computed for each EMG muscle site (right zygomatic, left zygomatic collapsed, right corrugator, left corrugator) relative to each of the three slide types (Positive, Neutral, Negative). These average change scores were used for the following EMG analyses.

**Zygomatic EMG Change Scores.** To test the hypothesis that zygomatic EMG activity would be significantly less in the PD group than in the NC group, a two-way ANOVA (2x3) (group x slide type (PON)) was computed with left and right zygomatic change scores collapsed. A significant main effect for group, $F(1,36) = 6.39$, $p < .0160$, was found. Inspection of the means--PD $M = 0.74$; NC $M = 3.17$--indicates that the PD group had significantly less zygomatic activity than the NCs. Also, an expected significant main effect for slide type, $F(2,72) = 11.18$, $p < .0001$, was found, indicating that the selected slides did indeed evoke differential muscle movement relative to positive, neutral, or negative slide content. A significant interaction effect between group and slide type, $F(2,72) = 4.10$, $p < .0206$, was also found with group differences in zygomatic activity being most marked in response to the positive slides. The graphic representation of group differences relative to group and slide type (PON) in the zygomatic muscle region is depicted
in Figure 9. It is apparent that the PD group expressed significantly less zygomatic activity than the NC group.

**Corrugator EMG Change Scores.** To test the hypothesis that corrugator EMG activity is significantly less in the PD group than in the NC group, a two-way ANOVA (2x3) (group x slide type (PON)) was computed with left and right corrugator change scores collapsed. No significant main effect for group was found. However, the expected significant main effect for slide type, \( F(2,72) = 8.58, \ p < .0005 \), was found, indicating that the selected slides did indeed evoke differential muscle movement relative to positive, neutral, and negative slide content. The interaction effect between group and slide type (PON) approached significance, \( F(2,72) = 2.89, \ p < .0618 \).

Inspection of the graphic representation of group differences relative to slide type, depicted in Figure 10, illustrates this trend toward the PD group displaying less corrugator activity in response to negative emotionally laden slides than the NC group.

**Zygomatic and Corrugator EMG Patterns.** Means and standard deviations for each group's zygomatic EMG change scores relative to slide type (PON) are depicted in Table 7. To test the hypothesis that each group would show the same expected pattern of zygomatic activity relative to slide type, separate one-way ANOVAs across slide type (PON) (with
Figure 9. Mean zygomatic EMG change scores by group and slide type (PON).
Figure 10. Mean corrugator EMG change scores by group and slide type (PON).
Table 7. Means and Standard Deviations of Zygomatic and Corrugator EMG Change Scores by Slide Type (PON) for the Parkinson's (PD) and Normal Control (NC) Groups.

<table>
<thead>
<tr>
<th></th>
<th>PD</th>
<th>NC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Zygomatic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>.59</td>
<td>.82</td>
</tr>
<tr>
<td>O</td>
<td>-.08</td>
<td>.47</td>
</tr>
<tr>
<td>N</td>
<td>-.01</td>
<td>.71</td>
</tr>
<tr>
<td>Corrugator</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>-.02</td>
<td>.44</td>
</tr>
<tr>
<td>O</td>
<td>.12</td>
<td>.76</td>
</tr>
<tr>
<td>N</td>
<td>.44</td>
<td>.87</td>
</tr>
</tbody>
</table>

Note. P = Positive, O = Neutral, N = Negative.
on/off drug conditions collapsed) were computed for the PD and the NC group. For the PD group, a significant main effect of slide type (PON), $F(2,36) = 8.45$, $p<.0010$, was found. Similarly, the NC group analyses showed a significant main effect for slide type, $F(2,36) = 7.60$, $p<.0018$. Review of group means indicates that both groups showed more zygomatic EMG activity in response to positive slide content than to neutral or negative slide content.

Means and standard deviations for each group's corrugator EMG change scores relative to slide type (PON) also are depicted in Table 8. To test the hypothesis that each group would show the same expected pattern of corrugator activity relative to slide type, separate one-way ANOVAs across slide type (PON) (with on/off drug conditions collapsed) were computed for the PD group and the NC group. For the PD group, a significant main effect of slide type (PON), $F(2,36) = 6.07$, $p<.0054$, was found. Similarly, the NC group showed a significant main effect for slide type, $F(2,36) = 5.72$, $p<.0070$. Review of group means indicates that both groups showed more corrugator EMG activity in response to negative slide content than to neutral or positive slide content.

Figures 11 and 12 show the expected pattern of zygomatic and corrugator EMG activity in response to slide type for the PD group and for the NC group, respectively. It
Figure 11. Pattern of mean EMG change score responses by slide type (PON) and EMG site for the Parkinson's group.
Figure 12. Pattern of mean EMG change score responses by slide type (PON) and EMG site for the normal control group.
is evident that both groups showed elevated muscle activity in the zygomatic region in response to positive slides and decreased muscle activity in the zygomatic region in response to negative slides. Also, both groups showed elevated muscle activity in the corrugator region in response to negative slides but reduced muscle activity in the corrugator region in response to positive slides.

Secondary Hypothesis--Levodopa Medication Effects on EMG Activity

A series of three and four-way ANOVAs with repeated measures were performed between the PD patients' "on" drug and "off" drug testing sessions to determine if significant differences in facial EMG activity and subjective experience of emotion exist relative to medication effects. It was hypothesized that the PD group would show significantly greater facial EMG activity across the zygomatic and corrugator muscle regions when "on" levodopa medication than when "off" medication.

Baseline EMG Activity. A graphic representation of baseline EMG activity by group, EMG site, and on/off drug conditions is depicted in Figure 13. To ascertain if significant differences existed between groups on resting baseline EMG activity relative to levodopa levels, a four-way ANOVA (group x on/off drug condition x side of face x EMG site) was computed. No significant main effect for
Figure 13. Mean baseline EMG activity by on/off drug condition for the Parkinson's (PD) and normal control (NC) groups.
group or for on/off drug condition was found. Also, no significant four-way interaction effects between group, on/off drug condition, side of face, or EMG site was found.

Zygomatic EMG Change Scores and Slide Presentation. To test the hypothesis that zygomatic EMG activity would be reduced in the PD group when "off" levodopa medication relative to EMG activity "on" medication, a three-way ANOVA (2x2x3) (group x on/off drug condition x slide type (PON)) was computed with right/left zygomatic EMG activity collapsed. A significant main effect for group, $F(1,36) = 6.39$, $p < .0160$, and the expected significant main effect for slide type, $F(2,72) = 11.18$, $p < .0001$, was found, suggesting that each group displayed differential EMG activity in response to varied slide content. However, no significant main effect for on/off drug condition was found. Also, no significant interaction effects between group and on/off drug condition or between group, on/off drug condition, and slide type were found. Results suggest that levodopa medication levels have no significant effect on the degree of displayed zygomatic EMG activity across slide type.

Corrugator EMG Change Scores and Slide Presentation. To view a graphic representation of "on" versus "off" mean zygomatic and mean corrugator EMG change scores within the PD and/or the NC group see Appendix F and G, respectively. To test the hypothesis that the PD group would show
significantly less corrugator activity when "off" levodopa medication than when "on" medication, a three-way ANOVA (2x2x3) (group x on/off drug condition x slide type (PON)) was computed with right/left corrugator EMG activity collapsed. No significant main effect for group or for on/off drug condition was found, although the expected significant main effect for slide type (PON), $\bar{F}(2,72) = 8.58, p<.0005$, was found. Also, no significant interaction effect between group and on/off drug condition or between group, on/off drug condition, and slide type (PON) was found, suggesting that levodopa medication levels do not significantly effect corrugator EMG activity across slide type.

**Summary**

The results indicate that although the PD group reported "feelings" as intensely as the NC group on valence and arousal dimensions of emotion, the PD group expressed less facial muscle movement in the zygomatic and corrugator muscle regions than did the NCs. However, the pattern of valence and arousal ratings as well as EMG activity in response to affect laden slides were essentially the same for both groups. That is, positive affect laden slides evoked higher zygomatic EMG activity and higher valence and arousal ratings whereas negative slides evoked higher corrugator EMG activity and arousal ratings but lower
valence ratings.
CHAPTER 5
DISCUSSION

The purpose of the present study was to determine whether individuals with idiopathic Parkinson's disease report feeling emotions as intensely as those without the disorder and/or whether they express the same degree of facial muscle movement in response to emotional stimuli. Following an explanation of how this study's paradigm is unique in comparison to previous studies, the results will be briefly reviewed. Then, implications of the results as well as suggested directions for future research will be discussed.

The present study differs from prior experimental studies of facial EMG in PD in five ways: (1) a specific subtype of PD individuals (i.e., some degree of masked facies, no dementing illness, and no significant depressive symptoms) was sought for inclusion into the study, (2) demographically matched normal control subjects (NCs) were used, (3) the study was designed to allow for both within group and between group comparisons, (4) bilateral EMG sites were used in the zygomatic and corrugator muscle regions, and (5) "on" and "off" levodopa medication trials (using matched alternative slide stimuli) were employed for the PD group, in comparison to repeated assessments, with the same alternate slide stimuli, for the NCs.
The present study demonstrates that the PD and NC groups show similar levels and patterns of valence and arousal ratings relative to slide type. Both groups rated more happy emotion for positive slide exposure than during negative slide exposure, and they reported more arousal for positive and negative slide exposure than for exposure of slides with neutral content.

This study also demonstrates that individuals with idiopathic Parkinson's disease (without evidence of depression) exhibit significantly less slide-evoked muscle movement in the zygomatic region and a borderline-significant trend toward less muscle movement in the corrugator region--irrespective of levodopa medication condition--than individuals without PD. As expected, both the PD and NC group showed higher EMG activity in the zygomatic muscle region while viewing positive slides and higher EMG activity in the corrugator muscle region while viewing negative slides.

The present study also tested the hypothesis that spontaneous, slide-evoked, facial musculature movement was attenuated in PD individuals when "off" levodopa medication than when "on" medication. Results indicate that, for this sample of PD patients, levodopa condition did not significantly affect spontaneous facial muscle movement in the zygomatic or corrugator region when visual emotional
stimuli were employed. Past research (Abbs et al., 1987; Schneider et al., 1986) has shown that in PD the loss of nigrostriatal dopamine may enhance the inhibitory sensory gating function of the basal ganglia so that there is a greater than normal inhibition of the access of sensory information to relevant motor areas. This is thought to result in a net motor hypoexcitability. Dopamine has been hypothesized to aid in the processing of such sensory information via the basal ganglia. Decreased dopamine may hinder modulation of the internal gating and filtering system of the basal ganglia, and increased dopamine may cause sensory information to filter through the system at an abnormally quick rate. When sensory information passes too quickly, thresholds for movement are hypothesized to be lowered, resulting in abnormal movements.

It is interesting that in the current study, levodopa levels (manipulated via medication) did not seem to affect spontaneous facial muscle movement in response to emotional stimuli. Thus, results suggest that in the zygomatic and corrugator facial muscle regions, dopamine does not function in the manner described. Rather, as Hunker et al. (1982) and others have suggested, impaired facial muscle movement in PD individuals may be due to an, as yet, unclarified relationship between rigidity and some aspects of hypokinesia, especially reduced range of movement in the
orofacial system. In this system muscle pairs that work together for symmetric midline movements are antagonistic for lateral-medial movements.

The present results lend support to the conclusion that PD patients who exhibit masked facies actually feel emotions just as intensely as those without PD. This hypothesis has implications for the spouse's, family's, and/or caregiver's perceptions of the PD individual's emotional state. The present PD sample of persons had less vigor and felt more fatigued and confused--irrespective of levodopa levels--but did not report significant depressive symptoms. This is of interest, given that to a lay person these individuals may be perceived as depressed or lacking emotion because of their masked facies. Thus, practical implications of the present results include the advisability of informing relatives of PD patients that emotional experience is likely normal despite reduced facial expression.

The present study attempted to contrast differential predictions derived from the James-Lange versus the Cannon-Bard theories of emotion. Because less facial musculature movement in this PD group, compared to NCs, was observed, despite reported "feelings" of emotions being similar to that in NCs, the results are more consistent with the Cannon-Bard theory. This theory posits emotion to be more centrally mediated, causing bodily changes to occur almost
simultaneously with emotional experience. However, the potential contributions to emotional experience of other aspects of peripheral facial feedback cannot be ruled out by the present study. It may be that the facial musculature is not of primary or secondary importance in determining the subjective emotional experience, and that other factors such as the skin of the face (with blood flow and temperature components), and/or the sound of one's own voice, and/or breathing patterns are primary contributions to felt emotion. If that is the case, then a feedback cycle of impulses descending from the cortex to the periphery and then back to the cortex, as the James-Lange theory posits, may make an important, and perhaps determining contribution to the subjective experience of emotion.

Thus, although the results of the present study are more consistent with the Cannon-Bard theory, modified versions of the James-Lange theory cannot be addressed. That is, these results argue against James' implied hypothesis and S. S. Tomkins' originally stated hypothesis (1962, 1963) that the facial musculature plays a role in "felt" emotion. However, the results are not able to address the general theory that a feedback cycle, via some other peripheral effectors, is necessary for the experience of emotion.

Matsumoto and Lee (1993) has asked two fundamental
questions, "Are there neural tracts leading from the facial musculature or facial skin that carry neural information back to an "integration center" after an expression has been produced?" or "Does the neural feedback occur earlier in the facial innervation process?" (p.250). Although results of the present study help to answer part of the former question, an integration of advanced research in the neuroanatomy and neurophysiological control of facial expressions as well as a better theoretical understanding of emotion is needed before these questions may be adequately addressed. Only then may these questions and others (e.g., Is a neural feedback system a necessary component of felt emotion?) be sufficiently answered.

**Implications for Future Research Directions**

Future research that assesses facial EMG activity in young adults compared to older adults is warranted to ascertain whether facial musculature movement decreases with age.

There exists some research suggesting that older adults have reduced magnitude of physiologic response to emotional stimuli. Levenson et al. (1991) found that although their normal elderly population showed lower ANS changes than the younger subjects in response to emotional stimuli, the older group had the same ANS pattern of response as the younger adults. Although EMG measures were not employed, Levenson
et al. (1991) found the magnitude of ANS activity to be smaller in old age regardless of the eliciting task. Levenson et al. (1991) suggested that although there is currently no evidence to suggest that ANS activity associated with emotion is any more diminished with age than ANS activity associated with nonemotional stimuli, emotional ANS response may be especially reduced in old age because emotion-specific ANS activity has been selected by evolution for its adaptive functions that are critical for survival, and as one ages, the need for adaptive functions may diminish. Similarly, it could be argued that as one ages, the evolutionary need to produce facial expressions for survival (e.g., anger) diminishes as well, resulting in reduced degree of facial musculature movement. However, greater display rules may be applied by older adults contributing to less facial EMG activity.

Carstensen (1987) has suggested that elderly people may appear to be less emotional because of lowered levels of social activity. This "selectivity" model suggests that in old age an affect regulation strategy is invoked by reduced social interactions; by purposefully limiting social interaction to people, events, and topics of personal enjoyment and interest, positive emotional experiences are optimized whereas negative emotional experiences are minimized. This lessening of negative experience, according
to Carstensen, could create the appearance of lowered emotionality.

The current study did show relatively small magnitude EMG change score differences within each group (although significantly different EMG change score patterns for positive, neutral, and negative slide content were evident). However, Greenwald et al. (1989) and Lang et al. (1993) found similar facial EMG change score magnitudes across positive, neutral, and negative slide content in their population of young college students. Thus, comparisons between the present results and studies of younger adults suggests that facial EMG activity in response to emotional stimuli may remain relatively constant throughout the normal aging process. There is clearly a need to compare healthy adult age groups, within a single study, in order to adequately test for age effects.

Finally, future studies are warranted in the areas of mood and normal aging, and/or mood and PD, to help support or negate the James-Lange versus the Cannon-Bard theories of emotion. Studies that may prove most useful in this endeavor are those with paradigms using Lane and Schwartz's (1987) Emotional Awareness Scale as well as measures of facial galvanic skin response and facial temperature while subjects imagine emotional thoughts or auditorily hear emotional statements or described scenarios. The Emotional
Awareness Scale is of special use in determining how developmentally aware individuals are of their emotions using a Piagetian conceptual framework. Once the level of emotional awareness is known, valence or arousal ratings ascertained from individuals following imagery or auditory trials of emotional content will be more meaningful.
### APPENDIX A

**Means, Standard Deviations, and Significance Levels of Individual Profile of Mood State Scales for the Parkinson's (PD) and Normal Control (NC) Groups Relative to Drug Condition**

<table>
<thead>
<tr>
<th></th>
<th>PD</th>
<th></th>
<th>NC</th>
<th></th>
<th>DF</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T-ON</td>
<td>36.95</td>
<td>4.49</td>
<td>33.94</td>
<td>4.10</td>
<td>36</td>
<td>4.62</td>
<td>.0384*</td>
</tr>
<tr>
<td>T-OFF</td>
<td>40.74</td>
<td>5.39</td>
<td>33.32</td>
<td>4.07</td>
<td>36</td>
<td>22.92</td>
<td>.0001*</td>
</tr>
<tr>
<td>D-ON</td>
<td>35.53</td>
<td>2.46</td>
<td>37.05</td>
<td>3.22</td>
<td>36</td>
<td>2.69</td>
<td>.1094</td>
</tr>
<tr>
<td>D-OFF</td>
<td>37.26</td>
<td>5.84</td>
<td>37.32</td>
<td>4.04</td>
<td>36</td>
<td>0.00</td>
<td>.9744</td>
</tr>
<tr>
<td>A-ON</td>
<td>38.37</td>
<td>1.46</td>
<td>39.58</td>
<td>2.91</td>
<td>36</td>
<td>2.62</td>
<td>.1140</td>
</tr>
<tr>
<td>A-OFF</td>
<td>39.21</td>
<td>4.05</td>
<td>40.32</td>
<td>4.71</td>
<td>36</td>
<td>.60</td>
<td>.4433</td>
</tr>
<tr>
<td>V-ON</td>
<td>60.58</td>
<td>9.09</td>
<td>64.05</td>
<td>10.80</td>
<td>36</td>
<td>1.15</td>
<td>.2908</td>
</tr>
<tr>
<td>V-OFF</td>
<td>56.84</td>
<td>11.00</td>
<td>67.05</td>
<td>8.71</td>
<td>36</td>
<td>10.05</td>
<td>.0031*</td>
</tr>
<tr>
<td>F-ON</td>
<td>42.63</td>
<td>5.70</td>
<td>41.32</td>
<td>5.13</td>
<td>36</td>
<td>.56</td>
<td>.4594</td>
</tr>
<tr>
<td>F-OFF</td>
<td>45.26</td>
<td>7.82</td>
<td>40.68</td>
<td>4.60</td>
<td>36</td>
<td>4.84</td>
<td>.0343*</td>
</tr>
<tr>
<td>C-ON</td>
<td>38.63</td>
<td>5.30</td>
<td>35.53</td>
<td>3.26</td>
<td>36</td>
<td>4.73</td>
<td>.0363*</td>
</tr>
<tr>
<td>C-OFF</td>
<td>42.05</td>
<td>6.96</td>
<td>36.05</td>
<td>4.11</td>
<td>36</td>
<td>10.45</td>
<td>.0026*</td>
</tr>
<tr>
<td>TMD-ON</td>
<td>4.79</td>
<td>14.87</td>
<td>-1.16</td>
<td>17.53</td>
<td>36</td>
<td>1.27</td>
<td>.2269</td>
</tr>
<tr>
<td>TMD-OFF</td>
<td>18.42</td>
<td>28.53</td>
<td>-0.53</td>
<td>19.23</td>
<td>36</td>
<td>5.76</td>
<td>.0217*</td>
</tr>
</tbody>
</table>

*Note.* Mean standard score for each scale = 50 (SD 10) except for the TMD scale (the sum of all six scales). NC subjects underwent a "mock" drug trial for comparison purposes only. KEY: T = Tension-Anxiety; D = Depression-Dejection; A = Anger-Hostility; V = Vigor; F = Fatigue; C = Confusion-Bewilderment; TMD = Total Mood Disturbance.

* = Significant Difference p < .05
APPENDIX B

Significance Levels for Individual Movement Items on the Unified Parkinson's Disease Rating Scale

Between Groups

<table>
<thead>
<tr>
<th>DEPENDENT VARIABLE</th>
<th>DF</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facial Expression</td>
<td>36</td>
<td>35.70</td>
<td>.0001*</td>
</tr>
<tr>
<td>Tremor at Rest of Arms</td>
<td>36</td>
<td>6.55</td>
<td>.0148*</td>
</tr>
<tr>
<td>Tremor at Rest of Legs</td>
<td>36</td>
<td>2.71</td>
<td>.1087</td>
</tr>
<tr>
<td>Action Tremor of Hands</td>
<td>36</td>
<td>1.95</td>
<td>.1716</td>
</tr>
<tr>
<td>Rigidity of Arms</td>
<td>36</td>
<td>19.21</td>
<td>.0001*</td>
</tr>
<tr>
<td>Rigidity of Legs</td>
<td>36</td>
<td>10.73</td>
<td>.0024*</td>
</tr>
<tr>
<td>Finger Taps</td>
<td>36</td>
<td>17.96</td>
<td>.0001*</td>
</tr>
<tr>
<td>Hand Movements</td>
<td>36</td>
<td>22.00</td>
<td>.0001*</td>
</tr>
<tr>
<td>Alternating Hand Movements</td>
<td>36</td>
<td>37.17</td>
<td>.0001*</td>
</tr>
<tr>
<td>Leg Agility</td>
<td>36</td>
<td>15.79</td>
<td>.0003*</td>
</tr>
<tr>
<td>Body Bradykinesia</td>
<td>36</td>
<td>32.42</td>
<td>.0001*</td>
</tr>
</tbody>
</table>

* = Significant Difference p < .05
APPENDIX C

Method for Deriving Positive, Neutral, and Negative Slide Types (PON)

Within each group the mean valence across on/off drug sessions was computed from the most valent to the least valent slide within each slide set (A and B). The nine slides with the lowest mean valence ratings were determined to have "Negative" content (N), the nine slides with the highest mean valence ratings were determined to have "Positive" content (P), and the nine slides with mean valence ratings between that given for the negative and positive slides were determined to have "Neutral" content (O). To assure that each group rated the same nine slides within each category (e.g., Positive, Neutral, Negative), mean valence ratings relative to specific slide content within each group was compared. Although the order of the nine slides within the Positive, Neutral, and Negative categories varied a bit for each group, the same nine slides were rated as belonging within the positive, neutral, or negative valence category. This same procedure was applied to the mean arousal ratings that corresponded to the mean valence ratings, yielding three distinct arousal categories of positive, neutral, and negative.
APPENDIX D

Mean Valence Ratings by Group. On/Off Drug Condition, and Slide Type (PON)

PARKINSON'S CONTROLS

<table>
<thead>
<tr>
<th></th>
<th>Pos</th>
<th>Neut</th>
<th>Neg</th>
<th>Pos</th>
<th>Neut</th>
<th>Neg</th>
</tr>
</thead>
<tbody>
<tr>
<td>On Drug</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Off Drug</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX E

Mean Arousal Ratings by Group.

On/Off Drug Condition, and Slide Type (PON)
APPENDIX F

Mean Zygomatic and Mean Corrugator EMG Change Scores by On/Off Drug Condition and Slide Type (PON) Within the Parkinson's Group

[Graph showing mean EMG change scores for Zygomatic and Corrugator muscles in the Parkinson's group on and off drug]
APPENDIX G

Mean Zygomatic and Mean Corrugator EMG Change Scores by On/Off Drug Condition and Slide Type (PON)

Within the Normal Control Group

![Graph showing mean EMG change scores for zygomatic and corrugator muscles by drug condition and slide type. The graph compares On Drug and Off Drug conditions for each slide type (Pos, Neut, Neg).]
References


Cannon, W.B. (1915). *Bodily changes in pain, hunger, fear, and rage* (p. 26).


