To my friends and family for all of their support
ACKNOWLEDGMENTS

I thank the chair and members of my committee for their discussion and constructive feedback, the participants for their time and participation, and the National Parkinson’s Foundation for its generous support.
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MUTED ENHANCEMENT OF EMOTIONAL MEMORY IN PARKINSON DISEASE

By

Lizabeth L. Jordan

August 2010

Chair: Dawn Bowers
Major: Psychology

It is widely known that emotional events are better remembered than neutral contents. This emotional enhancement is presumably due to increased arousal at the time of encoding that facilitates consolidation into memory. Converging evidence from imaging, lesion, and behavioral studies point to the role of the amygdala in modulating this emotional enhancement effect via its influence on the hippocampus. Based on observations of amygdalar dysfunction in Parkinson’s Disease (PD), we hypothesized that PD patients would not exhibit emotional enhancement in recall of novel information. To test this hypothesis, 23 non-demented idiopathic PD patients, 23 age-matched controls, and 25 young adults were shown 44 pictures on a computer monitor and asked to recall them 30’ later. Stimuli included high-arousing (negative, positive) and low-arousing (neutral) images from the International Affective Picture Set. Subjective ratings of valence and arousal were made following completion of the memory task. All PD patients were tested on dopaminergic medications, and all subjects were screened for dementia and mood (Beck Depression Inventory-II, Apathy Scale, and State-Trait Anxiety Inventory). In line with previous literature, older and younger controls recalled significantly more emotional (negative & positive) than neutral pictures (p <0.01). In
contrast, the memory recall of the PD group was not improved with emotional stimuli. Importantly, recall of neutral pictures was comparable across the three groups. Similarly, the three groups did not differ in their valence or arousal ratings of the stimuli. Regression analyses revealed no relationship between emotional memory and scores on any mood measures. Taken together, the non-demented PD patients failed to show an emotional enhancement effect in their recall of “new” memories. This was not due to general memory failure, visuoperceptual disturbance, or lack of appreciation of emotional value of the pictures. These memory finding with PD patients may reflect diminished arousal by the emotional stimuli and/or a disconnect between arousal and amygdala-hippocampal systems. Implications will be discussed in terms of contemporary views of emotional deficits in PD.
CHAPTER 1
INTRODUCTION

Emotional Enhancement Effect in Memory

Definition

Most individuals can remember in detail where they were when they heard the news of 9/11 or when they had their first kiss. In contrast, most individuals cannot remember what they were doing the day before 9/11 or their first kiss because those events were more neutral in comparison. The better recall of emotional events such as these is known as the “emotional enhancement effect” in memory and has generally been attributed to heightened levels of moderate arousal. Not only does emotion increase the likelihood for remembering an event, it also enhances memory for accurate details (Bradley et al., 1992; Kensinger et al., 2007; as reviewed in Labar & Cabeza, 2006). Over the years, increased memory for emotional events has been documented across multiple studies and laboratories, reported in both young and older adults, and more reliably observed in free recall than recognition formats (Jacques et al., 2009; as reviewed in Kensinger, 2004; Kensinger, 2008).

Modulation Hypothesis: arousal effects and amygdala activity

One of the most widely known models for the emotional enhancement effect is McGaugh’s “modulation hypothesis” (McGaugh, 2004). According to this model, the amygdala plays a critical role in modulating the hippocampus, a neural region important for memory consolidation. It does so by mediating the effects of stress hormones and other neurotransmitters that occur with emotionally arousing stimuli. The modulation hypothesis is based on 1) research demonstrating the role of the amygdala in the exhibition of emotional enhancement in memory (Cahill et al., 1996; Hamaan et al., 2002).
Findings from both the human and animal literature have documented that selective amygdale lesions can influence emotional memory. For example, selective amygdale lesions can affect the ability of animals to associate stimuli with reward value (Aggleton, 1992). Lesion studies in humans indicate the importance of a healthy amygdala in emotional arousal effects on memory. Hamann and colleagues (1997) reported that a mixed group of amnesiac patients with presumably intact amygdalae had better memory for emotionally arousing stimuli compared to neutral stimuli. This emotional enhancement pattern was highly similar to a healthy control group and occurred regardless of valence. In contrast to Hamann et al.’s findings, Adolphs et al. (2001) reported that patients with unilateral amygdalar damage and controls with no amygdalar damage similarly demonstrated better gist memory (i.e., memory for the general information but not necessarily details) for emotional stimuli compared to neutral stimuli. However, a patient with bilateral amygdala damage did not demonstrate the emotional enhancement pattern. In addition, several psychophysics studies in healthy individuals have also found a correlation between emotional arousal, amygdala activity (as measured by skin conductance response), and memory enhancement (Anderson et al., 2008; Buchanan et al., 2006).

Finally, functional imaging studies have also linked amygdale activity to enhanced episodic memory for emotional stimuli. This was first documented in a PET imaging study…
study by Cahill and colleagues (1996) who presented unpleasant and neutral stimuli for subjects to remember. Notably, increased amygdale activation and arousal at the time of learning correlated with subsequent recall many weeks later. Hamaan et al. (1999) extended this work to include both pleasant and unpleasant stimuli (pictures). More recent studies using fMRI paradigms have reported similar findings. Specifically, successful retrieval of emotionally arousing stimuli is predicted by greater amygdala (and hippocampal) activity at the time of encoding (Dolcos et al., 2004; Kensinger & Corkin, 2004). In summary, lesion, psychophysiology, and neuroimaging studies identify amygdala activity as an important correlate for emotional enhancement in memory across the lifespan.

The modulation hypothesis is further supported by known connections between the amygdala and the hippocampus, where memory consolidation occurs. Research indicates that one physiological pathway between the amygdala and hippocampus occurs via the hypothalamus and stress hormones and neurotransmitters. Evidence for this pathway includes observation of increased activity in the basolateral nucleus of the amygdala, which connects to the hypothalamus, during successful encoding of emotional memories (Dolcos et al., 2004; as reviewed in Izquierdo & McGaugh 2000). This basolateral amygdala activity is believed to cause release of adrenal stress hormones and neurotransmitters (i.e., epinephrine, norepinephrine, and glucocorticoids) by the hypothalamus to other areas including the hippocampus. Increased amounts of stress hormones and neurotransmitters during encoding and consolidation of memories are shown in both mouse models and human research to enhance subsequent memory for emotional pictures (Buchanan & Lovallo, 2001; McGaugh 2004). In summary, the
modulation hypothesis is supported by evidence that increased amygdala activity during encoding of emotional information stimulates hypothalamic release of stress hormones that are known to improve memory consolidation at the hippocampus.

The second proposed pathway in the modulation hypothesis occurs between the amygdala, entorhinal cortex, and hippocampus. The lateral nucleus of the amygdala has projections to entorhinal cortex, which directly connects to the anterior portion of the hippocampus. Functional imaging studies demonstrate that during the successful encoding of emotional memories, the most increased activity within the medio-temporal lobe is in the entorhinal and anterior hippocampal areas (Dolcos et al., 2004). Overall, functional imaging studies provide support for a modulation hypothesis that explains how the amygdala enhances emotional memory consolidation via both hypothalamic and entorhinal connections.

**Emotional Memory in Aging**

Emotional information can be characterized along two dimensions: valence (i.e., negative or positive) and arousal (i.e., calming or exciting). Memory studies examining these two dimensions suggest that there may be age-related differences in how valence and arousal influence memory. In terms of arousal, both young and older healthy adults demonstrate a similar emotional enhancement effect for high-arousing compared to low-arousing memories (Bradley et al., 1992; Kensinger 2008). For valence, however, age appears to be important. Specifically, the memory performance of young adults benefits equally from negative and positive materials relative to neutral information (Bradley et al., 1992). For older adults, findings are mixed. Some studies find that older adults remember relatively more positive than negative materials (Leigland et al., 2004, Mather & Carstensen, 2005). This has been referred to as the “positivity effect.” Regardless,
evidence from a large body of research indicates that increased arousal and valence improve memory for emotional information in healthy young and older adults.

Importantly, the modulation hypothesis for emotional enhancement in memory is supported in healthy aging research. That is, neuroimaging studies have demonstrated that the role of the amygdala, although reduced relative to young adults, remains important in the emotional enhancement effect in memory of healthy older adults (Jacques et al., 2009b; Mather et al., 2004). Specifically, these studies use functional magnetic resonance imaging to demonstrate that amygdala activity predicted enhanced memory for negative and positive stimuli compared to neutral stimuli in both healthy young and older adults.

The basis for the positivity effect in older adults is unknown. However, some investigators have hypothesized that the positivity effect occurs because older adults intentionally deflect their attention away from negative information and attempt to regulate their emotional experiences (Kensinger & Corkin, 2003; Leigland et al., 2004; Mather & Carstensen, 2005). Presumably this intentional emotional regulation requires processes that are mediated by prefrontal cortex, which inhibits amygdalar activation and modulation of negative memory (Jacques et al., 2009a,b; Kensinger & Corkin, 2003; Mather & Carstensen, 2005.) In summary, the amygdala continues to play an important role in the emotional enhancement of older adults’ memory.

**Summary of Emotional Enhancement in Memory**

In summary, emotional content is better remembered than neutral content. The modulation hypothesis proposes that the amygdala has a necessary role in modulating the emotion effects on memory consolidation of memories in the hippocampus.

Evidence for the modulation hypothesis is outlined in lesion, physiological, and imaging
research demonstrating the importance of the amygdala and its connections to the hippocampus. The emotional enhancement effect in memory and the modulation hypothesis is also supported in healthy aging literature.

**Parkinson’s Disease**

**Introduction**

Parkinson’s disease (PD) is a progressive dopamine depletion disorder that is characterized by tremor, rigidity, and bradykinesia (slowness of movement). Historically, it was viewed as purely a movement disorder. However, contemporary research has begun to identify behavioral changes in memory, mood, and emotional processing in PD. However, little is known about the interactions between these behavioral changes and Parkinson’s disease. Research on emotional memory in PD aims to better understand the relationship between emotional and memory deficits. The following sections review literature on memory and emotional processing in PD.

**Memory Difficulties in PD**

Although patients with Parkinson’s disease are rarely reported as being “amnesic,” neuropsychological studies do describe specific types of memory impairments. In particular, individuals with PD exhibit greater deficits in free recall memory than recognition, which can rely on more automatic, less effortful processes (Whittington et al.,2006). Additionally, non-demented individuals with PD are able to improve their recall memory when provided with cues (Appollonio et al.,1994). Similarly, on recognition tasks, PD patients rely more on familiarity (i.e., gist memory, or a feeling of knowing) rather than more effortful, true recollection (i.e., exact memory including details and source) compared to healthy controls (Edelstyn et al.,2007). When stimuli are presented in a format where information is already semantically organized
(e.g., stories), PD patients are able to recall more information than from a less structured set of stimuli (e.g., randomly-ordered list of words). (Zahodne et al., 2009). Taken together, this memory profile is often viewed as reflecting “retrieval” rather than consolidation difficulties. Moreover, these retrieval difficulties are thought to stem from frontal-striatal dysfunction resulting in poor organization and poor strategy use for effectively remembering information (Higginson et al., 2003; Bohlhalter et al., 2009).

As described previously, the acquisition of new memories by older adults appears to benefit from emotional arousal, similar to that of younger adults (Kensinger, 2008). One question that arises is whether memory encoding by individuals with Parkinson disease might also benefit from emotional stimuli. To the extent that arousal is “automatically” triggered by emotional stimuli and does not rely on the integrity of fronto-striatal circuitry, then one might expect PD patients to show emotional enhancement effects in memory. However, based on the McGaugh model, this enhancement effect would appear to be predicated on relatively intact amygdalar functioning. The following sections review the literature on known amygdala-related changes in PD.

**Pathologic Changes in PD Amygdalae**

The major neuropathological feature in PD is widespread dopaminergic depletion arising from degeneration of the substantia nigra pars compacta. Not only are the classic fronto-basal ganglia-thalamic circuits affected, but there is also widespread dopaminergic loss in mesolimbic and mesocortical projections. The latter are particularly important as these dopaminergic pathways directly influence the function of the amygdala (Salgado-Pineda et al., 2005). A number of studies have described pathologic changes in the amygdale of Parkinson patients. For example, Ouchi et al.
(1999) reported that PD patients exhibit a 30-45% reduction in dopamine agonist binding in the amygdala. Additionally, functional magnetic resonance imaging studies show that amygdalar activity is modulated by dopamine such that amygdala activation in PD is increased with dopaminergic medication (Tessitore et al., 2002).

In addition to decreased dopamine input, structural changes in the amygdala also occur in late stages of Parkinson’s disease. Reductions in total amygdala volume are estimated between 20% to 40% (Harding et al., 2002; Ouchi et al., 1999). Post-mortem studies have identified some Lewy body pathology in PD that results in significant damage to the amygdala (and hippocampus and anteromedial temporal cortex) during stages 5 - 6 of the disease process (Braak et al., 2003). The nuclei of the amygdala most affected by Lewy bodies are the accessory cortical and central nuclei, which project to areas responsible by autonomic dysfunctions (Braak et al., 1994). In comparison, other areas of the amygdala, including the basal and lateral nuclei, which project to the entorhinal cortex and hippocampus, remain relatively intact in non-demented PD cases (Braak et al., 1994; Harding et al., 2002). In summary, evidence suggests neuropathological changes in the amygdale associated with PD.

**Behavioral Changes Associated with Emotion**

A variety of mood and behavioral changes are observed in Parkinson disease, including depression, apathy, anxiety and decreased anticipation of reward/punishment contingencies (Miller et al., 2007; Kirsch-Darrow et al., 2006, Mattox et al., 2006). The basis for these changes are complex and multi-faceted, though it is tempting to attribute these behaviors to dysfunction in limbic circuitry, including the amygdala and ventral striatum. However, few studies have directly “probed” amygdala functioning in Parkinson disease. Tessitore and colleagues conducted an fMRI study in patients with
PD who were asked to discriminate emotional faces. Results revealed decreased amygdalar activation in PD relative to controls. This occurred even though the PD patients performed completely normally on the facial emotion task. Such findings imply that the reduced amygdalar activation, as indexed by fMRI, was not sufficient to influence behavioral outcome. Other studies have made “inferences” regarding limbic dysfunction based on patterns of behavior that are known to have specific neural correlates. For example, one of the neural correlates of fear potentiated or emotion modulated startle is the amygdala, based on lesion studies in the rat by Davis and colleagues (Davis et al., 1993). Bowers et al. (2006) reported that PD patients exhibited blunted reactivity to aversive stimuli in comparison to control participants. More recently, Kawamura and Kobayakawa (2009) computationally modeled the source of event-related potentials of PD patients and controls during face expression and olfactory tasks, both of which have been associated with amygdalar functioning in normal controls (Buchanan et al., 2003; Derntl et al., 2009). Results showed dipole modeling did not converge on the amygdale in the PD patients for either the facial expression or olfactory task. Taken together, such studies provide some “indirect” evidence of possible amygdale-related functional changes in patients with Parkinson disease.

**Emotional Memory in PD**

To date, only one previous study has evaluated the emotional enhancement effect in patients with Parkinson disease. Schneider et al. (2003) examined the effects of deep brain stimulation on recall memory for neutral and emotional stories in PD. The researchers reported no significant interaction between stimulation and valence conditions but did not describe main effects of stimulation or valence. That is, they did
not explain if or how the PDs delayed memory improved with emotional valence and/or deep brain stimulation. Additionally, Schneider and colleagues did not compare their PD results to a healthy control group. As a result, there is a gap in current literature about how the emotional enhancement effect is manifested in the delayed recall memory of PD patients.
CHAPTER 2
STATEMENT OF THE PROBLEM

Although primarily viewed as a movement disorder, research has begun to reveal behavioral changes associated with Parkinson’s disease. Behavioral changes in PD include emotion-related and memory impairments. The overall focus of this current study is to examine the emotional enhancement effect in memory to better understand emotion-related and memory changes in Parkinson’s disease.

Previous research has well-described the emotional enhancement effect of memory in healthy young and older adults. That is, information that is emotionally arousing is better remembered than neutral content. McGaugh’s modulation hypothesis proposes that the amygdala plays an important role in the emotional enhancement of memory. Lesion, psychophysiology, and imaging studies indicate that the amygdala modulates memory consolidation in the hippocampus via stress hormones and entorhinal connections. The current literature reports that the emotional enhancement effect in memory is preserved in healthy aging, but only one published study has investigated its pattern in Parkinson’s disease.

Parkinson’s disease, which is commonly viewed as a movement disorder caused by dopamine depletion, is associated with a variety of behavioral changes, including decreased recall memory and increased rates of depression and apathy. Specifically, the recall deficits in PD are associated with fronto-striatal-based retrieval impairments. It is possible that emotional arousal during encoding could improve PD recall memory. However, research indicates that PD is associated with pathological changes in the amygdala, including decreased dopaminergic binding and decreased volume. The basis for these changes is unknown, but likely relate to neurotransmitter changes
(especially dopamine) and pathologic entities such as Lewy bodies. To date, only one previous study has examined the emotional enhancement effect in PD delayed memory. However, the study did not publish its findings on the main effects of valence in PD memory nor a control group comparison. Consequently, it remains unknown whether memory of PD patients might display an emotional enhancement effect.

This current study aims to close the gap in published literature by fully describing the emotional enhancement effect in PD memory. A full description of this topic will provide a better understanding of both emotional processing and memory and their interaction in PD. If individuals with PD do benefit from the emotional enhancement effect in memory, then emphasis on emotional content may be a useful encoding strategy to improve their memory. If individuals with PD do not benefit from emotional enhancement effects, then deficits in emotional memory may be contributing to the increased rates of apathy and other mood changes characteristic of PD. Overall, this study will investigate the emotional enhancement effect in memory of PD so that we can better understand how changes in emotional processing affect individuals with PD.

**Specific Aim 1**

Four specific aims were addressed in this study. To determine whether healthy older adults demonstrate an emotional enhancement effect comparable to young adults. A comparison between a healthy young and older adult group will provide a model for normal changes in healthy aging, which we can later compare to results from the PD group. Previous research demonstrates that both young and healthy older adults have better memory for emotionally arousing stimuli compared to neutral ones. The modulation hypothesis proposes that the emotional enhancement effect on memory remains intact with healthy amygdala-hippocampal connections. The young and
healthy older adults in this study presumably have intact amygdala and hippocampal function. Therefore, it was predicted that the healthy young and older adults will have similar patterns of emotional enhancement in memory, such that emotionally arousing content (both negative and positive) will be better remembered than neutral content.

Specific Aim 2

To determine whether patients with Parkinson’s disease demonstrate an emotional enhancement effect comparable to that of age matched controls. Based on the McGaugh model of emotional memory and coupled with previous reports of pathological changes of the amygdala in PD, it was hypothesized that PD patients would have better memory for emotionally arousing pictures than low-arousing neutral pictures, but the emotional enhancement would be blunted in comparison.

Specific Aim 3

To determine whether anticipated blunting of the emotional enhancement in memory might be due to decreased ability of the PD patients to subjectively evaluate and report the emotional content of the memory stimuli. Based on previous observations that Parkinson’s patients rate emotional stimuli similarly to healthy controls (Miller et al., 2009; Simons et al., 2004; Smith et al., 1996), it was expected that the PD patients would subjectively evaluate the emotional content of stimuli similarly to the healthy control groups.

Specific Aim 4

To determine whether variations in emotional memory is better explained by disease status or other factors (i.e., mood, baseline memory) on emotional memory in PD patients relative to controls. For example, deficits in baseline memory may affect their exhibition of the emotional enhancement effect. That is, if the Parkinson’s patients
are recalling a very low amount of information, then the emotional enhancement effect may not be significant due to floor effects. Mood may be another possible confounding factor because Parkinson’s disease status is highly correlated with depression, apathy, and anxiety. Therefore, it is important to examine the effect of baseline memory and mood on emotional memory in order to control for possible disease-related confounding factors. Based on memory research in PD, it was predicted that the PD patients would recall less information than the healthy control groups. Additionally, based on the research describing amygdalar dysfunction caused by PD independent of disease-related mood changes, it was expected that disease status would be more predictive of emotional memory than mood reports.
CHAPTER 3
METHODS

An overview of the study design is shown in Figure 3-1. The following sections will describe each of these study elements in more detail.

Figure 3-1. Diagram of Research Design

**Participant Recruitment**

Participants included three groups: 1) individuals with idiopathic Parkinson Disease (N = 23), 2) healthy older adults (OA, N = 23), and 3) younger adults (YA, N = 25). The Parkinson’s patients were recruited from the University of Florida’s Movement Disorders Center. All had undergone extensive neurologic screening in order to establish a definitive diagnosis of PD based on the UK Brain Bank criteria (Hughes et al., 1992). All of the Parkinson’s patients, except one, were candidates for deep brain stimulation surgery. Healthy young and older adults were recruited from the Gainesville
community and reimbursed $10 for their travelling expenses. Major exclusion criteria for all participant groups were dementia (MMSE <25), current uncontrolled psychiatric disorder (e.g., substance abuse), or past history of neurological illness or surgery other than Parkinson’s disease (e.g., stroke, traumatic brain injury).

Characteristics of the study sample are depicted in Table 3-1. As shown, the PD group was well-educated, in their 60’s, primarily Caucasian, and had more men than women (i.e. 13/10 ratio). The PD group was slightly less educated than the older adult control group (PD mean = 15.3 yrs, OA = 16.9 yrs, p < .05), but did not differ on other characteristics. Other than age, the YA control group was similar to the other groups with the exception of years of education, which differed from the OA controls (YA mean = 15.4 yrs, OA mean = 16.9 yrs, p < .05).

Table 3-1. Participant Demographic & Disease Characteristics, mean (sd)

<table>
<thead>
<tr>
<th></th>
<th>YA (N=25)</th>
<th>OA (N=23)</th>
<th>PD (N=23)</th>
<th>significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>21.16 (1.41)</td>
<td>66.26 (9.54)</td>
<td>63.22 (10.50)</td>
<td>.001**</td>
</tr>
<tr>
<td>Educ</td>
<td>15.44 (1.96)</td>
<td>16.98 (2.36)</td>
<td>15.20 (2.20)</td>
<td>.05*</td>
</tr>
<tr>
<td>Sex(M/F)</td>
<td>15/10</td>
<td>13/10</td>
<td>13/10</td>
<td></td>
</tr>
<tr>
<td>Hand (R/L)</td>
<td>24/1</td>
<td>20/3</td>
<td>23/2</td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>20 Cauc</td>
<td>22 Cauc</td>
<td>19 Cauc</td>
<td></td>
</tr>
<tr>
<td>Hoehn-Yahr (On/Off)</td>
<td>2.94 (.76) on</td>
<td>2.42 (.24) off</td>
<td></td>
<td></td>
</tr>
<tr>
<td>UPDRS III^ (On/Off)</td>
<td>27.76 (10.1) on</td>
<td>46.57 (11.9) off</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LED+</td>
<td>861.6 (712.9)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1Significantly different from YA; 2.Significantly different from OA; 3. Significantly different from PD group; **Significant Kruskall-Wallis H [χ^2(2) =68.65, p <.001, eta^2=.69] with post-hoc ranks tests; *Significant one-way ANOVA [F(2,68) = 4.59, p <.05] with Bonferonni –corrected post-hoc tests; ^UPDRS III = Unified Parkinson Disease Rating Scale- motor; +LED = levodopa equivalent dose.

Disease characteristics of the PD group are also depicted in Table 3-1. As shown, the PD patients ranged from moderate to severe disease severity according to standard
measures for staging disease progression (Hoehn and Yahr, 1967) and motor symptom severity (Unified Parkinson Disease Scale, UPDRS; Fahn et al., 1987). The Hoehn-Yahr and UPDRS scores were obtained during routine neurology clinic visits within six months of the experimental protocol.

**Screening and Baseline Measures**

Evaluation took place in the Cognitive Neuroscience Laboratory at the McKnight Brain Institute. The PD patients completed testing while “on” their dopaminergic medications. Informed consent was obtained according to University and Federal guidelines.

All participants underwent brief cognitive screening with the Mini Mental Status Examination (MMSE; Folstein et al., 1975) in order to rule out dementia. Individuals with MMSE scores equal or less than twenty-five were excluded from further study participation and analyses. A total of 25 Parkinson’s disease patients were recruited; 2 patients were excluded due to low MMSE scores resulting in final N of 23 PD participants. Participants who met the inclusion criteria completed selected measures of attention, recent memory and mood. Attention and memory measures included Digit Span and Logical Memory subtests from the Weschler Memory Scale – III (WMS-III; Weschler, 1945/1997). These were given in order to characterize participants’ attention and recent memory abilities using neutral, standardized measures. Mood measures included Beck Depression Inventory – II (BDI-II; Beck et al., 1996), Apathy Scale (Starkstein et al., 1992), and State Trait Anxiety Inventory (STAI; Spielberger et al., 1970). These were given in order to assess the participants’ current mood status. Between group differences in attention, recent memory and mood were examined.
Emotional Memory Task

Participants received an emotional memory task that consisted of three phases: a picture study phase, a delayed free recall task and a phase involving subjective ratings of valence and arousal.

Picture Stimuli

The stimuli were forty-six grayscaled versions of pictures from the International Affective Picture system (IAPs), a standardized set of emotion pictures with normative ratings for valence and arousal (Lang & Bradley, 2001). Pictures were not presented in their original color versions because the same stimulus set was being used in a companion pupillometry study which required color and luminance to be standardized across stimuli. Importantly, previous work by Bradley and colleagues (2001) found no difference in valence and arousal ratings between color and grey-scale versions of the IAPS pictures.

The target stimulus set for the current study included: a) 14 neutral pictures (low arousal); b) 14 positive pictures (moderate to high arousal), and c) 14 negative pictures (moderate to high arousal). Four additional neutral pictures (low arousal) were used as “filler stimuli.” Detailed Information about the content of the target pictures is presented in Table 3-2. The negative and positive pictures significantly differed from the neutral pictures in terms of arousal, (negative v. neutral mean difference = 3.43, t(39) = 12.50, p <.001; positive v. neutral mean difference = 2.82, t(39) =10.27 , p <.001) and the negative and positive valenced stimuli did not statistically differ in terms of arousal.

Study Phase

During the “encoding” phase, participants watched a series of 46 IAPS pictures (4 fillers, 42 targets), one at a time, on a 20-inch flat screen computer monitor that was
situated on a table in front of them. Participants were instructed to view the pictures as if they were watching a movie. They were not warned about a later memory test. Each picture was presented for 6-sec, with a 2-sec inter-stimulus interval that consisted of a gray screen with a white, centered fixation cross. Throughout the study phase, participants sat comfortably in a chair, approximately 24 inches from the monitor. The total duration of the study phase was 368 seconds.

Presentation of the picture set always began and ended with two neutral filler pictures. These initial and final filler pictures were included to control for recency and
primacy effects in memory recall and were excluded in subsequent analyses for memory. The 42 target pictures were randomized, but balanced in terms of frequency of occurrence of positive, negative, and neutral pictures across the first and last half of the set. All participants viewed the picture in the same order.

Recall Test

Recall was tested approximately 30 minutes following presentation of the last target item. During the delay, participants completed mood and other questionnaires and took a bathroom break, if necessary. Recollection of the pictures was evaluated in a free recall format. During recall testing, participants were asked to describe in detail as many of the pictures they had previously seen as possible. Participants were instructed to provide enough detail about a picture so that it could be easily identified by another person as belonging to the target set. Participants were prompted to provide more details if vague descriptions were provided. The recall test was un-timed and discontinued when the participant reported that he/she could not describe more pictures. All responses were recorded verbatim by the experimenter for later scoring by blinded raters.

Subjective Ratings of Valence and Arousal

After the free recall memory test, subjective ratings of valence and arousal were made for each target picture. Participants were shown a booklet containing the forty-six target pictures along with separate valence and arousal scales using the Self-Assessment Manikin (SAM, Figure 3-3; Bradley & Lang, 1994). For both scales, the ratings ranged from 1 to 9. For valence scale, a score of 1 represented unpleasant (feelings like unhappiness, unsatisfaction, upset, and despair) and a score of 9 represented pleasant (feelings of happiness, pleasantness, satisfaction, and hope). For
the arousal scale, a score of 1 represented calmness (feelings of relaxation, sluggishness, tiredness) and a score of 9 represented excitement (feelings of stimulation, frenziedness, wide-awake). On both scales, a score of 5 represented no change from baseline emotion.

Methods for Reducing Free Recall Data

The criterion for correct recall was that a response had to provide sufficient (and accurate) detail that corresponded to only one picture in the encoding set. Responses that were ambiguous and/or described more than one picture from the encoding set were not categorized as a correct recall response. These criteria were used by three blinded raters for identifying correctly recalled items. Recall ratings were made by three undergraduate research assistants who were familiar with the stimulus set but did not know the identity of the respondents. Correctly recalled pictures were categorized according to their IAPs standardized ratings into a valence type (negative, neutral, positive) and an arousal type (low, moderate, arousing). The breakdown of the entire stimulus set into their picture type is presented in Table 3-3. As shown, there was equal distribution of negative, positive, and neutral stimuli in the set (i.e., 14 each). However, this was not the case for “arousal.” Because of unequal distribution, the final outcome measure for each participant was the proportion of pictures correctly recalled in each picture valence and arousal type by dividing the number recalled by the total number of pictures presented.

Statistical Analyses

To determine whether the emotional enhancement effect differed among the PD, older, and younger control groups (Aims 1 and 2), two 3 X 3 repeated measures analyses of variance (ANOVA) were conducted. In one, the dependent variable
Figure 3-3. Self-Assessment Manikin Scale used to rate the valence (pleasantness) and arousal (intensity) of the stimuli.

Table 3-3. Breakdown of Target Stimuli by Valence and Arousal Type

<table>
<thead>
<tr>
<th></th>
<th>Negative</th>
<th>Neutral</th>
<th>Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>14</td>
<td>14</td>
<td>14</td>
</tr>
<tr>
<td>Low-arousing</td>
<td>0</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>Moderately-arousing</td>
<td>9</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>High-arousing</td>
<td>5</td>
<td>0</td>
<td>4</td>
</tr>
</tbody>
</table>

(dv) was the proportion of pictures correctly recalled according to valence categories (pleasant, unpleasant, neutral). In the second, the dv was the proportion of pictures recalled according to arousal categories (low, medium, high). For both analyses, the between subject factor was group (YA: young adults, OA: healthy older adults, PD: Parkinson’s disease patients), and the within-subjects factor was picture type (either valence: negative, neutral, positive or arousal: low, moderate, high). Bonferonni post-hoc comparisons were conducted, as appropriate.

To determine whether the PD patients differed in their evaluation of affective pictures relative to the control groups (Aim 3), subjective ratings of valence and arousal
were analyzed using non-parametric statistics. First, two omnibus Kruskal-Wallis H tests were conducted to examine between group (young, healthy older, and PD) differences. One Kruskal-Wallis H test examined between group differences across valence ratings (dv’s = negative, neutral, and positive ratings). The second examined between group differences across arousal ratings (dv’s = low, moderate and high ratings). Next, an omnibus Friedman’s Chi-squared test and Nemenyi’s post hoc tests were conducted to examine valence differences across an average of all participant ratings (dv’s = averaged across-groups negative, neutral, and positive ratings). A second omnibus Friedman's Chi-squared test and further Nemenyi’s post hoc tests were conducted to examine arousal differences across an average of all participant ratings (dv’s = averaged across groups’ low, moderate and high arousing ratings). Finally, similar Friedman’s Chi-squared test and Nemenyi’s post hoc tests were conducted to examine valence and arousal differences within each participant group.

Specific aim 4 was to compare the effect of disease status to other characteristics (i.e., baseline memory and mood) on emotional memory in Parkinson’s disease. In order to investigate baseline memory as a potential confounding factor affecting the emotional enhancement in PD memory, memory for neutral items were compared between the PD and healthy older adult group. Additionally, scores on the WMS-III Logical Memory delayed recall subtest were statistically compared between groups using an independent t-test. In order to investigate mood status as a possible confounding factor between groups, multiple regression models were used to compare the effects of disease status, depression (as measured by the BDI II), apathy, and state anxiety (STAI-state) scores on memory for emotional pictures. Participant group and
mood scores were entered as possible predictors into a stepwise regression using a $p<.05$ criterion. Four separate regressions were conducted, one for each of the following dependent variables: memory for negative, positive, moderate-arousing, and high-arousing pictures.
CHAPTER 4
RESULTS

Total mean recall of items from the 42 item picture set was as follows: Parkinson group = 7.36 (SD 4.17), Older adults = 10.83 (SD 3.7), and Younger Adults = 13.2 (3.73). Results of a one-way ANOVA and follow-up post-hoc comparisons (Bonferroni-adjusted) indicated that the 3 groups all significantly differed from each other (F(2,67) = 13.41, p< .001; PD vs. OA: t = -3.00, p < .01; OA vs. YA: t = -2.13, p < .05; PD vs. YA: t = -5.17, p < .01 ). Thus, the PD group recalled significantly fewer items than the older controls who recalled fewer items than the younger adults.

In order to examine the influence of emotional valence and arousal on memory recall, it was necessary to convert the raw recall scores to proportionate scores. Although there were equal numbers of negative, positive and neutral pictures in the stimulus set (i.e., 14 each), this was not the case for “arousal.” For arousal, there was unequal distribution of pictures across the low (n=12), moderate (n=21) and high arousing (n = 9) categories. (See Table 3-4). Because of this unequal distribution, proportionate recall scores were computed for each arousal type by dividing the number of recalled items by the total number of pictures in that category. A similar procedure was followed for valence in order to allow for easier comparison with arousal findings, by putting them on the same measurement metric.

Specific Aims 1 and 2: Group Differences in Free Recall

Valence Analyses

The proportionate recall scores of the PD, OA, and YA are shown in the table below (Table 4-1).

Results of a 3 (Group) X 3 (Valence: negative, neutral, positive) ANOVA are
Table 4-1. Mean Proportionate Recall of Negative, Neutral, and Positive Items (SE)

<table>
<thead>
<tr>
<th></th>
<th>YA</th>
<th>OA</th>
<th>PD</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>negative</td>
<td>.41 (.03)</td>
<td>.33 (.03)</td>
<td>.22 (.03)</td>
<td>.32 (.02)</td>
</tr>
<tr>
<td>neutral</td>
<td>.24 (.03)</td>
<td>.15 (.03)</td>
<td>.15 (.03)</td>
<td>.18 (.02)</td>
</tr>
<tr>
<td>positive</td>
<td>.32 (.02)</td>
<td>.30 (.03)</td>
<td>.19 (.03)</td>
<td>.27 (.01)</td>
</tr>
<tr>
<td>Total</td>
<td>.32 (.02)</td>
<td>.26 (.02)</td>
<td>.19 (.02)</td>
<td></td>
</tr>
</tbody>
</table>

shown in Table 4.2. There was no violation of homogeneity of variance (i.e., shown in Table 4.2. There was neither violation of homogeneity of variance (i.e., nonsignificant Levene’s test) nor violation of the sphericity assumption (i.e., nonsignificant Mauchley’s test). As shown, there was a significant main effect for Group (F(2, 67) = 11.65 =11.65, p < .001). Post-hoc comparisons (Bonferroni-adjusted) revealed that the PD patients recalled significantly fewer items than either the older adults (t (67) = 2.6, p < .05) or the young adults (t (67) = 4.82, p < .001). The older adults recalled fewer items than the younger adults at trend level (t(67) = -2.22, p = .09). There was also a significant main effect for Valence (F (2,134 = 30.91, p < .001). Post-hoc comparisons revealed differential recall of all picture valences. Negative pictures were recalled better than positive pictures (t(134) = 2.65, p < .05), and both negative and positive pictures were recalled better than neutral pictures (negative: t(134) = 7.62, p<.001; positive: t(134) = -5.21, p<.001).

Finally, the Group X Valence interaction was also significant (F(4,134 )= 2.5, p < .04). This is graphically depicted in Figure 4.1. Post-hoc comparisons revealed that

Table 4-2. Free Recall Analyzed as a Function of Valence

<table>
<thead>
<tr>
<th>Source</th>
<th>Type III Sum of Squares</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>Sig.</th>
<th>Partial Eta Squared</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>.22</td>
<td>2</td>
<td>.11</td>
<td>11.65</td>
<td>.00</td>
<td>.26</td>
</tr>
<tr>
<td>Valence</td>
<td>.74</td>
<td>2</td>
<td>.37</td>
<td>30.91</td>
<td>.00</td>
<td>.32</td>
</tr>
<tr>
<td>Valence*Group</td>
<td>.12</td>
<td>4</td>
<td>.03</td>
<td>2.50</td>
<td>.04</td>
<td>.07</td>
</tr>
</tbody>
</table>
the memory recall of the young and older control groups significantly benefitted from emotional valence, whereas that of the PD group did not. Specifically  a) for the YA group, negative pictures were better recalled than positive pictures $t(134) = 2.65, p < .05$ and neutral pictures ($t(134) = 7.62, p < .001$ ); similarly, positive pictures were better recalled than neutral pictures ($t(134) = -5.21, p < .001$); b) for the OA group, the negative and positive pictures did not differ ($p > .10$), although both were better recalled than neutral pictures (negative vs. neutral: $t(134) = -5.68, p < .001$, positive vs. neutral: $t(134) = -4.96, p < .001$), and c) for the PD group, there were no significant differences in recall among any of the picture valences (all $p$'s > .09). Additional comparisons across groups revealed that there were no differences in the recall of neutral pictures between the PD, older, or young adult groups (PD v. OA: $t(134) = -0.01, p = 1.0$; OA v. YA: $t(134) = -2.36, p = .06$; YA v. PD: $t(134) = -2.34, p = .07$). The Parkinson’s group recalled significantly fewer negative pictures and positive pictures than both the healthy young and older adult group (negative: PD v. OA, $t(134) = -2.81, p < .05$, PD v. YA,
t(134) = -4.76, p < .001; positive: PD v. OA t(134) = -2.97, p < .05, PD v. YA t(134) = -3.53, p < .01). The young and older adult groups did not significantly differ from each other in their recall of negative or positive pictures (negative: t(134) = 1.92, p = .18, positive: t(134) = .53, p = 1.0).

Arousal Analyses

The proportionate recall scores of the PD, OA, and YA as a function of arousal are shown in the table below (Table 4.3).

<table>
<thead>
<tr>
<th></th>
<th>YA</th>
<th>OA</th>
<th>PD</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>low</td>
<td>.26 (.03)</td>
<td>.16 (.03)</td>
<td>.16 (.03)</td>
<td>.19 (.02)</td>
</tr>
<tr>
<td>mod</td>
<td>.26 (.03)</td>
<td>.20 (.03)</td>
<td>.16 (.03)</td>
<td>.21 (.02)</td>
</tr>
<tr>
<td>high</td>
<td>.37 (.02)</td>
<td>.34 (.02)</td>
<td>.20 (.02)</td>
<td>.30 (.01)</td>
</tr>
<tr>
<td>Total</td>
<td>.30 (.02)</td>
<td>.23 (.02)</td>
<td>.17 (.02)</td>
<td></td>
</tr>
</tbody>
</table>

Results of a 3 (Group) X 3 (Arousal: low, moderate, high) are shown in Table 4-4. Again, there were no violations of homogeneity of variance or the sphericity assumption (i.e., nonsignificant Levene’s test, nonsignificant Mauchley’s test). All main effects and interactions were significant. Decomposition of the significant main effect for Group (F(2, 67) = 9.99, p < .001) using Bonferroni adjusted t-tests revealed that the PD patients recalled significantly fewer items than the young adults (t (67) = 4.45, p < .001). In contrast, the older adult group did not significantly differ from either the young adult or Parkinson’s disease group (OA v. YA: t (67) = -2.34, p = .07, OA v. PD: t(67) = 2.10, p = .12. There was also a significant main effect for Arousal (F(2,134) = 21.62, p < .001). Post-hoc comparisons revealed that high arousing pictures were better recalled than either moderate (t(134) = 5.39, p < .001) or low arousing pictures (t(134) = 6.09, p < .001). There were no recall differences between moderate and low arousing pictures.
(t = .24, p =1.0).

The Group X Arousal interaction was also significant ($F(4,134) = 2.69, p < .03$) and is shown in Figure 4.2. Post-hoc comparisons revealed that both the older and younger adults recalled more high arousing than moderate or low arousing pictures (OA: High vs. moderate $t(134) = 4.59, p < .001$; High vs. low $t(134) = 5.81, p < .001$; YA: High vs. moderate $t(134) = 3.66, p < .01$; High vs. low $t(134) = 3.38, p < .01$). Low and moderate arousing pictures did not significantly differ in either the young or older adult group (YA: $t(134) = -1.18, p = 1.00$, OA: $1.28, p = .62$). In contrast, the PD group did not demonstrate any significant differences in memory recall according to arousal type (all p’s > .50). The young adults recalled significantly more low-arousing pictures than the older adults and Parkinson’s patients (YA vs. OA: $t(134) = 2.54, p < .06$; YA vs. PD: $t(134) = 2.51, p < .05$). Additionally, the young adult group recalled significantly more moderately-arousing pictures than the Parkinson’s disease (YA vs. PD: $t(134) = 2.53, p < .05$). Both the young and older adult group recalled significantly more high-arousing pictures than the Parkinson’s disease group. (YA vs. PD: $t(134) = -5.21, p < .001$, OA vs. PD: $t(134) = 4.32, p < .001$). The older and young adult groups did not significantly differ in their recall of moderate- or high-arousing pictures (moderate: $t(134) = 1.57, p = .36$, high: $t(134) = .82, p = 1.0$). The healthy older adult and Parkinson’s disease group did not significantly differ in their recall of low-arousing or moderately-arousing pictures (low-arousing: $t(134) = .01, p = 1.0$, moderate-arousing: $t(134) = .95, p = 1.0$).

A final analysis was conducted in order to learn whether the extent of emotional enhancement differed for the young and older control groups. The dependent variable was the percent change in memory for low-arousing to high-arousing pictures.
Table 4-4. Free Recall Analyzed as a Function of Arousal

<table>
<thead>
<tr>
<th>Source</th>
<th>Type III Sum of Squares</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>Sig.</th>
<th>Partial Eta Squared</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>.18</td>
<td>2</td>
<td>.09</td>
<td>9.99</td>
<td>.00</td>
<td>.23</td>
</tr>
<tr>
<td>Arousal</td>
<td>.51</td>
<td>2</td>
<td>.26</td>
<td>21.62</td>
<td>.00</td>
<td>.24</td>
</tr>
<tr>
<td>Arousal*Group</td>
<td>.13</td>
<td>4</td>
<td>.03</td>
<td>2.69</td>
<td>.03</td>
<td>.07</td>
</tr>
</tbody>
</table>

Figure 4-2. Free Recall Analyzed as a Function of Arousal

(Specifically, the calculation was dv = (high – low) / (low +1) *100.) A percent change score was used because the two groups differed in their baseline recall of low arousing pictures (i.e., older adults were significantly worse). Results of an independent t-test revealed that the older adult group had a significantly greater benefit for high-arousing pictures than the young adult group (OA mean = 17.25 (SE = 2.80), YA mean = 9.34 (SE = 2.56), t(46) = -2.09, p <.05) . This is graphically depicted in Figure 4-3.

In summary, both the healthy young and older adult groups demonstrated better memory for negative and positive pictures compared to neutral. Additionally, the young and older adult groups did not significantly differ in their recall of negative, neutral, or positive pictures. In contrast, the Parkinson’s group did not demonstrate better memory
Figure 4-3. Comparison of Emotional Enhancement Effect between young and older adult group as a function of percent change from memory for low to high arousing pictures.

*Asterisk indicates p <.05 significance.

for negative or positive pictures compared to neutral, and they recalled significantly fewer negative and positive pictures than both the young and older adult groups. When analyzed as a function of arousal, both the healthy young and older adults demonstrated better memory for high-arousing pictures compared to low and moderately-arousing pictures. Additionally, the older adult groups benefitted significantly more than the young adults for high compared to low-arousing pictures. In comparison, the Parkinson’s disease group did not have significantly better memory for high-arousing compared to low or moderately arousing pictures. The Parkinson’s group recalled significantly fewer high-arousing pictures than both the young and older adult groups.
Specific Aim 3: Between-Group Comparison of Subjective Ratings

Valence Analyses

The average subjective ratings of the PD, OA, and YA are shown in the table and figure below (Table 4-5, Figure 4-4).

Table 4-5. Average Subjective Ratings of Negative, Neutral, and Positive Items (se)

<table>
<thead>
<tr>
<th></th>
<th>YA</th>
<th>OA</th>
<th>PD</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative</td>
<td>2.60 (.16)</td>
<td>2.33 (.17)</td>
<td>2.38 (.23)</td>
<td>2.43 (.11)</td>
</tr>
<tr>
<td>Neutral</td>
<td>4.62 (.06)</td>
<td>4.51 (.13)</td>
<td>4.53 (.14)</td>
<td>4.55 (.07)</td>
</tr>
<tr>
<td>Positive</td>
<td>6.61 (.13)</td>
<td>6.87 (.19)</td>
<td>7.07 (.23)</td>
<td>6.84 (.11)</td>
</tr>
<tr>
<td>Total</td>
<td>4.60 (.04)</td>
<td>4.56 (.09)</td>
<td>4.65 (.11)</td>
<td></td>
</tr>
</tbody>
</table>

Figure 4-4. Average Subjective Ratings of Negative, Neutral, and Positive Items

*significantly different p < .05.

Results of an omnibus Kruskal-Wallis H test found no significant main effect of group (i.e., no differences between participant groups) on any valence ratings (negative: \( H(2) = 2.24, p = .33 \), neutral: \( H(2) = .19, p = .91 \), positive: \( H(2) = 4.57, p = .10 \)).
Consequently, no post-hoc analyses were conducted to further examine group differences. An omnibus Friedman's Chi-squared test identified a significant main effect of valence type (\(\chi^2 (2) = 134.45, p < .001, \eta^2 = .65\)). A Nemenyi's post hoc test revealed significant differences between all valence levels, \(p < .05\) (negative vs. neutral mean rank difference = -.89, negative vs. positive).

Finally, the Group X Valence interaction was not significant. That is, all participant groups demonstrated the same pattern of significant differences between negative, neutral, and positive picture ratings (all \(p\)'s < .05, YA: negative vs. neutral mean rank difference = -1.00, negative vs. positive mean rank difference = -2.00, neutral vs. positive mean rank difference = -1.00, OA: negative vs. neutral mean rank difference = -.83, negative vs. positive mean rank difference = -1.91, neutral vs. positive mean rank difference = -1.09, PD: negative vs. neutral mean rank difference = -.83, negative vs. positive mean rank difference = -1.91, neutral vs. positive mean rank difference = -1.09). This is graphically depicted in Figure 4-4.

**Arousal Analyses**

The average subjective ratings of the PD, OA, and YA are shown in the table and figure below (Table 4-6, Figure 4-5).

<table>
<thead>
<tr>
<th></th>
<th>YA</th>
<th>CON</th>
<th>PD</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>low</td>
<td>3.04 (.33)</td>
<td>3.78 (.32)</td>
<td>3.54 (.35)</td>
<td>3.45 (.19)</td>
</tr>
<tr>
<td>mod</td>
<td>4.62 (.32)</td>
<td>4.94 (.30)</td>
<td>5.06 (.36)</td>
<td>4.89 (.19)</td>
</tr>
<tr>
<td>high</td>
<td>5.81 (.35)</td>
<td>6.36 (.32)</td>
<td>6.04 (.34)</td>
<td>6.06 (.19)</td>
</tr>
<tr>
<td>Total</td>
<td>4.78 (.29)</td>
<td>5.32 (.27)</td>
<td>5.11 (.31)</td>
<td></td>
</tr>
</tbody>
</table>
Results of an omnibus Kruskal-Wallis H test found no significant main effect of group (i.e., no differences between participant groups) for arousal ratings (low: $H(2) = 3.72, p = .16$, moderate: $H(2) = .81, p = .67$, high: $H(2) = 1.45, p = .48$). Consequently,

![Figure 4-5. Average Subjective Ratings of Low, Moderate, and High Arousing Items *significantly different p < .05.](image)

no post-hoc analyses were conducted. An omnibus Friedman's Chi-squared test identified a significant main effect of arousal type ($\chi^2 (2) = 97.43, p < .001$, eta2= .47). A Nemenyi's post hoc test revealed significant differences between all arousal levels, $p < .05$ (low vs. moderate mean rank difference = -.89, low vs. high mean rank difference = -1.66, moderate vs. high mean rank difference = -.78).

Finally, the Group X Arousal interaction was not significant. That is, all participant groups demonstrated the same pattern of significant differences between low, moderate, and high-arousing picture ratings (all $p$'s < .05, YA: low vs. moderate mean rank difference = -.92, low vs. high mean rank difference = -1.65, moderate vs. high mean rank difference = -.73, OA: low vs. moderate mean rank difference = -.70, low vs. high mean rank difference = -1.65, moderate vs. high mean rank difference = -
.96, PD: low vs. moderate mean rank difference = -1.04, low vs. high mean rank difference = -1.70, moderate vs. high mean rank difference = -.65) This is graphically depicted in Figure 4-5.

In summary, there were no group differences among the subjective valence or arousal ratings of the stimuli set. Additionally, all groups appropriately rated each picture type significantly different. That is, on the valence scale, the negative pictures were rated significantly lower than the neutral pictures, and neutral pictures were rated significantly lower than the positive pictures. Similarly on the arousal scale, the low-arousing pictures were rated significantly lower than the moderately-arousing pictures, and the moderately-arousing pictures were rated significantly lower than the high-arousing pictures. No group X valence or groups X arousal interactions were revealed.

**Specific Aim 4: Contributions to Emotional Memory**

Additional analyses were conducted to learn whether cognitive, mood, and various disease-related factors might contribute to performance on the emotional memory task.

The cognitive and mood profiles of each group are shown in Table 4-7.

| Table 4-7. Participant Profiles, mean (sd) |
|------------------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
|                                          | YA                              | OA                              | PD                              | significance |
| MMSE                                     | 29.67 (.57)         2,3          | 28.81 (1.33)        1,3          | 27.61 (1.79)        1,2          | .001*          |
| Digit Span (ss)                          | 13.66 (9.90)       | 11.14 (3.23)          | 10.09 (2.71)          | ns               |
| Logical Memory Delayed Recall (SS)       | 11.28 (2.42)       | 12.82 (2.82)          | 10.82 (3.85)          | ns               |
| BDI II                                   | 6.36 (4.71)        2,3          | 2.77 (2.49)        1,3          | 10.54 (7.54)        1,2          | .001*          |
| Apathy Scale                             | 10.88 (3.59)       2            | 6.95 (4.31)        1,3          | 10.86 (6.17)        2            | .01**          |
| STAI- state anxiety (%)                  | 49.67 (28.87)      | 34.16 (26.26)         | 58.3 (28.69)          | .05*             |
| STAI- trait anxiety (%)                  | 55.00 (26.60)      | 42.76 (27.79)         | 59.05 (29.22)         | ns               |
| # on Antidepressant                      | 0                  | 1                  | 9                   | --               |

1. Significantly different from young adult group; 2. Significantly different from older adult group; 3. Significantly different from Parkinson’s disease group

*Significant Kruskall-Wallis H with post-hoc ranks tests: MMSE [H(2) =18.33, p <.001, eta2=.30]; BDI-II: [H(2) =20.48, p <.001, eta2=.30], STAI-State [H(2) =6.70, p <.05, eta2=.11]

**Significant one-way ANOVA [F(2,66) = 5.07, p <.01] with Bonferroni corrected post-hoc test
As shown, the 3 groups significantly differed on a dementia screening measure, the Mini-Mental State Exam (YA > OA > PD), though the PD group was well within the normal range. There were no differences among the groups on measures of attention span (WMS-III Digit Span) or 30 minute delayed recall of stories (WMS-III Logical Memory II). However, there were significant group differences across 3 of the 4 mood measures including the Beck Depression Scale –II, the Apathy Scale, and a measure of state anxiety (STAI). All groups differed from one another on the BDI-II (PD > YA > OA). For the Apathy Scale, the older adults endorsed fewer symptoms than either the younger adults or the PD groups, who did not differ from one another on apathy. Finally, the PD group endorsed more symptoms of current anxiety than the older adult control group. In summary, there were significant group differences on several indices of mood and the MMSE. Importantly, however, none of these differences reflected scores that were clinically significant (i.e., all within the normal range clinically).

Multiple regression analyses for recall of emotional pictures were conducted in order to determine if mood scores (BDI-II, Apathy, State Anxiety) explained more variance than group membership in emotional memory. Group membership, BDI-II, AS, and State Anxiety scores were simultaneously entered into a stepwise-selecting multiple regression model, using a p< .05 criterion. Three separate regression analyses examined each of the three types of emotional memory (memory for negative, positive, and high-arousing pictures) indentified by the ANOVAs to be significantly different from neutral memory (memory for neutral, low- and moderate-arousing pictures) of healthy young and older adults. The final regression models identified are presented in Table 4-8. As shown, group membership was a significant predictor for negative, positive, and
high-arousing pictures. In comparison to the young adult group, membership to the healthy older adult group predicted lower recall of negative pictures \( [\beta = -.26, t(61) = -2.11, p <0.05] \). Membership to the Parkinson's group predicted even lower recall of negative pictures compared to the young adult group, \( [\beta = -.56, t(61) = -4.45, p <0.001] \). Only membership to the Parkinson's group predicted recall of positive and high-arousing pictures \( [\text{positive pictures: } \beta = -.40, t(61)= -3.49, p <0.001, \text{ high-arousing pictures: } \beta = -.56, t(61) = -4.98, p <0.001] \). All of these regression models were significant \( \text{negative: } F(2, 60), p <.001, \text{ positive: } F(1, 61), p <.001, \text{ high-arousing: } F(1, 61), p < .001 \). None of the multiple regression models identified mood scores as significant predictors of recall of negative, positive, or high arousing pictures.

To summarize, group differences explained a significant amount of variance in recall memory for all types of emotional pictures. Mood scores did not contribute to the regression models predicting emotional memory.

### Table 4-8. Summary of Final Multiple Regression Models

<table>
<thead>
<tr>
<th>Dependent Variable (recall of --- pictures)</th>
<th>Significant predictor (( \beta )*)</th>
<th>SS Reg</th>
<th>df 1</th>
<th>df 2</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>negative</td>
<td>OA group (-.26), PD group (-.56)</td>
<td>0.37</td>
<td>2</td>
<td>60</td>
<td>9.93</td>
<td>0.00</td>
</tr>
<tr>
<td>positive</td>
<td>PD group (-.41)</td>
<td>0.19</td>
<td>1</td>
<td>61</td>
<td>12.17</td>
<td>0.00</td>
</tr>
<tr>
<td>high-arousing</td>
<td>PD group (-.54)</td>
<td>0.32</td>
<td>1</td>
<td>61</td>
<td>24.79</td>
<td>0.00</td>
</tr>
</tbody>
</table>

* Beta (\( \beta \)) weights for group membership are relative to YA group
There were three major findings in the present study. First, an emotional enhancement effect was observed in both the young and older controls. Both groups recalled significantly more emotional pictures than neutral pictures, and this effect appeared to be primarily driven by the high arousing pictures. Thus, the emotional enhancement effect was most salient for high-arousal pictures and occurred across both negative and positive valences. One important question is whether the younger and older adults benefitted equally by the emotional nature of the pictures. Did age make a difference? Our findings suggested yes. Although the older adults recalled fewer neutral memories than the younger adults, the percentage improvement in recall afforded by the “emotional” nature of the pictures was actually greater for the older adults than for the younger adults. The older adults actually benefitted more from high arousing picture emotionality. This is the first study to demonstrate this.

A secondary observation in the recall of the younger and older adults related the “positivity effect.” The positivity effect refers to the propensity for some older adults to pay attention to and recall more positive rather negative events (Leigland et al., 2004; Mather & Carstensen, 2005). This phenomenon was first described by Carstensen (as reviewed by Mather & Carstensen, 2005) who attributed it to emotional regulation on the part of older adults in an attempt to focus on the positive. In the present study, we found no evidence for the positivity effect in the older adult sample. Kensinger (2008) recently proposed that the positivity effect only occurs with less arousing stimuli and that it is overshadowed when more potent, evocative stimuli are used (as in the present study).
Taken together, this study identified emotional arousal effects in memory for healthy older and younger adults (Specific Aim 1) and replicated previous findings in the literature (Bradley et al., 1992; Kensinger 2008). Both groups recalled more emotionally arousing pictures (negative and positive) than neutral pictures, with older adults benefitting somewhat more from the arousing nature of the pictures than the younger adults.

The second major finding of the present study related to the performance of the Parkinson patients on the emotional memory task. The Parkinson group did not recall significantly more emotional pictures than neutral, low-arousing pictures. That is, the PD group showed no difference in memory for negative, neutral, or positive stimuli. Unlike the healthy control groups, their recall memory was not significantly enhanced by emotional content (i.e., “blunted” emotional enhancement effect).

The basis for the emotionally blunted memory in the PD group is unclear, though there are several possibilities. One possibility is that the PD patients failed to cognitively appraise and/or appropriately discern the emotional nature of the pictures. This was tested in the present study by examining the subjective picture ratings that were completed by the PD patients relative to the controls (Specific Aim 3). Results showed that the three groups (PD, OA, YA) did not significantly differ in their ratings of valence and arousal. The PD patients rated the IAPS pictures similarly to the older and younger controls. This finding corresponds to some, but not all previous reports in the literature regarding IAPS ratings by Parkinson patients (Miller et al., 2009; Simons et al., 2004; Smith et al., 1996; cf. Bowers et al., 2006). Thus, the third major finding was that the current PD sample appeared able to perceive and evaluate the emotional content of
the pictures they viewed in a manner similar to the controls. These data do not support inaccurate perception or appraisal by the PD group as the basis for emotional memory blunting.

Another possibility for the blunted emotional enhancement effect in the PD group is that it was secondary to global memory impairment. Two lines of evidence argue against this proposal. First, the PD patients performed in the average range on a standardized neuropsychological memory measure (i.e., delayed recall of WMS-III stories) and no differently from the older control group. Second, on the emotional memory task, the PD patients recalled the same number of neutral memories as the older adult controls. What distinguished the two groups was the failure of the PD patients to show enhanced recall for emotional memories. Therefore, baseline differences in recall memory cannot explain group differences in emotional enhancement effects. Nor can it be argued that the emotional enhancement effect in PD patients did not have enough power to be statistically significant. It is possible that an increased memory load may have revealed group differences in neutral memories between the PD and healthy older adult groups; however, these results suggest that the group differences are more likely to occur in impaired memory for emotional rather than neutral content when both were previously studied.

Another possibility for the blunted emotional enhancement in the PD patients relates to mood differences. Indeed, significant group differences were identified on measures of depression (BDI-II), apathy (AS), and state anxiety (STAI), though not always in the expected directions (i.e., PD and young adults had higher apathy scores than older adults). These group differences are in agreement with reports of greater
depression, apathy, and anxiety symptoms in Parkinson’s disease (Miller et al., 2007; Kirsch-Darrow et al., 2006). Even so, the scores across all mood scales remained in the non-clinical ranges for the PD group. We conducted post-hoc statistical analyses to examine the possibility that differences in mood status might better explain variation in emotional memory more than group differences. Multiple regression analyses determined that differences between the healthy individuals and Parkinson’s patients in emotional memory were not better explained by differences in symptoms of depression, apathy, or state anxiety.

In summary, the failure of the PD group to show an emotional enhancement effect in their memory recall of pictures could not be explained by mood status or baseline memory differences (Specific Aim 4). Nor was it due to inability to cognitively appraise the emotional meaning of the picture stimuli.

**Proposed Neuropathology of Blunted Emotional Enhancement**

The best-articulated and supported model for emotional enhancement effects in memory is that of McGaugh (2004). According to this model, the amygdala directly modulates the hippocampus by mediating the effects of arousal associated with stress hormones and neurochemicals that occur with emotionally arousing stimuli. Within the framework of this model, there are at least two likely candidate deficits that could give rise to emotionally blunted memory in PD. One relates to deficient arousal and the other relates to failure of the amygdala to influence the hippocampus.

The current study does not have data that can directly address the hypoarousal hypothesis. Arousal can be induced by multiple neural systems in the brain, both cortical and limbic (Critchley, 2002). In a parallel study in our lab (Dietz et al., 2010), pupil responses were measured while different groups of PD patients and controls
viewed the same emotional and neutral pictures as used in the present study. The pupillary response is a measure of autonomic arousal that is triggered by the hypothalamus in response to cortical (prefrontal, parietal), subcortical (thalamic), and limbic (amygdale, cingulate) inputs (Critchley, 2002). Dietz et al. (2010) found that pupil dilation of PD patients was larger when they viewed emotional pictures (negative and positive) and smaller when they viewed neutral pictures. This “arousal modulation” of pupil responses in the PD group was similar to that of the control group, indicating that PD patients could be differentially aroused by emotional picture stimuli. Because a different group of PD patients were used in the Dietz study, we can only raise the possibility that perhaps our PD patients also had a normal pattern of autonomic arousal in response to the same set of emotional pictures.

If PD patients are able to become emotionally aroused at the autonomic level, the question becomes why they are unable to use this “arousal” to assist in the encoding and consolidation of memories, in line with the McGaugh model. One likely explanation relates to abnormal amygdalar function in modulating the hippocampus. A variety of studies have described pathological and functional abnormalities in the amygdala of PD patients (Harding et al., 2002; Ouchi et al., 1999; Braak et al., 2003) Using fMRI, Tessitore et al. (2002) observed no functional activity of the amygdala in PD patients who were off medication; when the PD patients were rescanned during their “on dopaminergic” state, amygdala activity significantly increased, though it continued to be reduced relative to healthy controls.

In healthy individuals, amygdala activity is typically inhibited by prefrontal cortex (Marowsky et al., 2005). When confronted with arousing stimuli, an influx of dopamine
suppresses this inhibition and results in amygdala activation (Marowsky et al., 2005), which results in downstream effects on multiple systems to which the amygdala projects, including the hippocampus. Bowers at al. (2006) applied this line of reasoning to Parkinson disease and proposed that PD patients have impaired dopaminergic gating of the amygdala, thereby resulting in diminished amygdalar activation and influence on the startle circuitry in the brainstem. A similar line of reasoning can be applied to the present study. Thus, the amydala is not sensitive to dopaminergic changes associated with arousing stimuli such as emotional pictures. Subsequent amygdalar inhibition by prefrontal cortex is not sufficiently suppressed due to impaired dopaminergic gating within the amygdala. This results in decreased downstream activation of the hippocampus. In this manner, we propose that memory of people with PD does not significantly benefit from emotional enhancement effects because abnormal dopaminergic gating in the PD brain does not significantly increase amygdala activity and modulation of memory consolidation in the hippocampus.

**Limitations of the Present Study & Future Directions**

There are several limitations to this study. First, this study lacked an independent assessment of arousal. It is not empirically known if the PD patients in this study were hypoaroused or similarly aroused as the healthy controls, such as those PD participants in Dietz’s study. In order to examine the direct relationship between psychophysiological arousal, a future paradigm should incorporate pupillometry or another central measure of arousal/activation, such as fMRI or ERP.

A second limitation is that all PD patients were tested on their dopaminergic medication. Based on the hypothesis that reduced emotional memory occurs in PD due to decreased availability of dopamine during encoding, one would predict that PD
patients tested off of their dopaminergic medication would have even greater
impairments in emotional memory compared to PD patients tested on medication.
However, in order to demonstrate this hypothesis, a future study would need to test PD
patients both on and off their dopaminergic medications.

A third limitation is that our results may not generalize to the greater PD population
because our PD sample was not entirely homogenous and had a restricted range of
disease severity. For example, our PD group included some individuals who had
previously undergone deep brain stimulation (DBS N = 2). When the DBS patients
were excluded from the analyses, the same pattern of blunted emotional memory was
observed. However, the significant group by picture type interaction was no longer
statistically significant, most likely due to the reduced sample size and power in the
analysis. Our findings may be limited to PD patients in the midstage of the disease
process because our PD sample was restricted to Hoehn and Yahr stages 2 – 3 (on
medication). The emotional enhancement effect may still be present in the earliest
stages of the PD process and it would be interesting to explore the trajectory of emotion
related changes in memory.

Finally, the nature of our stimuli (gray-scaled IAPS pictures) is another limitation of
this study. Bradley and colleagues reported that black and white IAPs pictures elicit the
same psychophysiological arousal as colored versions; however, it is possible that the
lack of colored stimuli influenced the arousing nature of the stimuli we used.
Additionally, the emotional pictures tended to be more complex visual stimuli than the
neutral pictures. Therefore, it is possible that memory enhancement occurred due to
increased interest in the complexity of the pictures rather than the emotional content.
Furthermore, this study was a measure of emotional enhancement in nonverbal information and did not assess memory for verbal stimuli. Theoretically, similar results should be demonstrated with verbal information; however, this study did not empirically test this hypothesis. In addition to verbal content, a future study should examine emotional enhancement of remote episodic memories in PD. It is possible that memory for remote information encoded previous to disease onset may be differentially affected by emotion compared to recent memories, which this present study only assessed.

**Overall Implications**

Previous studies have identified significant emotion-related changes associated with Parkinson’s disease. In particular, people with Parkinson’s disease experience increased rates of apathy, anxiety and depression (Kirsch-Darrow et al., 2006). In addition to mood changes, Parkinson disease has been associated with decreased reward sensitivity (Mattox et al., 2006), decreased emotion modulation of startle (Bowers et al., 2006), and decreased processing of affective signals such as facial expressions or prosody (Clark et al., 2008; Lloyd, AJ, 1999). However, the latter findings have not been universal. Researchers suggest that deficits in dopamine to mesocortical and mesolimbic pathways contribute to the manifestation of blunted emotional reactivity and expression in PD.

This current study adds to this literature by showing that non-demented patients in the mid-stages of Parkinson disease fail to display the typical pattern of emotional enhancement in memory that is typically observed in age-peers. The blunted emotional memory was not due to a generalized memory defect. Nor was it related to depression symptoms, medications, or failure to perceive/appreciate the meaning of the pictures to be remembered. Rather, we propose that this muting may reflect an
 amygdala-based defect in activating and modulating the hippocampus, in line with the
model of McGaugh. The basis for this defect is unclear, but one mechanism could
involve faulty dopaminergic gating of the amygdala, resulting in increased 'inhibition' of
the amygdale in the manner described by Marowsky et al. (2005).

One important question pertains to the broader implications of the emotional
memory finding, since it occurred in patients who were relatively intact in terms of
cognitive and psychiatric status. It is possible that deficits in accumulating new
emotional memories precipitate and/or contribute to emotional flattening and decreased
motivation. Specifically, Bradley and colleagues (2001) propose that high-arousing
events activate an affective motivational system such that negative high-arousing stimuli
activate the defensive motivational system and positive high-arousing stimuli activate
the appetitive motivational system. Thus, deficits in memory for high-arousing events
may contribute to sustained impairments, such as apathy, in the affective motivational
system in PD. Future research should continue to explore the relationship between
deficits in emotional processing and more complex psychiatric mood states in patients
with Parkinson disease.
LIST OF REFERENCES


BIOGRAPHICAL SKETCH

Lizabeth Jordan is a Ph.D. graduate student in the Department of Clinical and Health Psychology at the University of Florida (U.F.). She grew up in Orlando, FL and completed her undergraduate studies in the Brain and Cognitive Sciences at the Massachusetts Institute of Technology (M.I.T.) in Cambridge, MA. At M.I.T., Lizabeth began developing her interest in the brain, cognition, and effects of aging and age-related diseases, such as Parkinson’s disease (PD). Non-motor symptoms of PD are the main focus of her research in Dr. Dawn Bower’s Cognitive Neuroscience Laboratory at UF, where she is a current graduate assistant.

Her study in emotional memory was inspired by research that she was involved in with Dr. Suzanne Corkin, Ph.D. while she was an undergraduate at M.I.T. Alongside graduate students in Dr. Corkin’s behavioral neuroscience laboratory, Lizabeth helped to design and conduct research investigating aging effects on source memory of neutral and emotional words. This undergraduate research experience led her to hypothesize her own ideas about emotional memory in PD.