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Neuropsychologia 42 (2004) 791–800

NEUROPSYCHOLOGIA

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Effects of Alzheimer disease on memory for verbal emotional information

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Received 19 March 2003; accepted 7 November 2003

Abstract

In healthy young and older adults, emotional information is often better remembered than neutral information. It is an open question, however, whether emotional memory enhancement is blunted or preserved in Alzheimer disease (AD). Prior studies of emotional memory in AD have included small samples of patients. In addition, studies that failed to find an enhancement effect in AD used stimuli lacking semantic coherence (e.g. lists of unrelated words, some that were emotional and others that were neutral). To circumvent these limitations, the present study examined a large number of AD patients ($N = 80$) and investigated whether AD patients would show better memory for a verbal description of an emotional event as compared to a neutral one. AD patients were equivalent to young and older control participants in rating the emotional descriptions for valence and arousal. Unlike the control groups, however, memory in AD patients did not benefit from the emotional narratives. We conclude that AD disrupts memory enhancement for at least some types of verbal emotional information.

Keywords: Alzheimer disease (AD); Emotional memory

1. Introduction

The panoply of sensory input that we experience in daily life is often infused with emotional relevance. This emotional information is usually better remembered than neutral information. An extreme example of this enhancement is the formation of a "flashbulb memory," in which individuals retain a particularly vivid memory of an exceptionally emotional event (Brown & Kulik, 1977). This enhancement, however, can also occur in the laboratory, using more controlled stimuli and encoding conditions: Emotional memory enhancement has been demonstrated using a range of stimuli, including words, sentences, pictures, and narrated slide shows (see Hamann, 2001; Buchanan & Adolphs, 2003, for reviews).

Numerous cognitive and neural processes likely contribute to emotional memory enhancement. At encoding, individuals may elaborate on emotional information more than neutral information. They may be prone to associate emotional items with additional semantic information, or with autobiographical experiences. These encoding strategies could lead to a richer representation of emotional information as compared to neutral information, and these differences in the richness, or distinctiveness, of the memory could underlie the enhancement effect (see Doerksen & Shimamura, 2001; Ochsner, 2000; Kensinger & Corkin, in press; Kensinger & Corkin, 2003a, for evidence that emotional information is more vividly remembered than neutral information). Individuals also rehearse emotional information more than neutral information; this increased rumination on emotional events could mediate the enhancement effect (Christianson & Engelberg, 1999). Emotion may also exert effects at retrieval: emotion may serve as a retrieval cue; for example, a person may initially remember how they felt about an event, and that cue may then allow them to generate additional features about the event. Thus, there may be additional support present for the retrieval of emotional as compared to neutral information.

Another critical factor underlying the emotional memory enhancement effect concerns the modulation of consolidation processes: emotional information may be more likely to be consolidated than neutral information, thus increasing the likelihood that emotional information will be retained over a delay. This modulation of consolidation may be mediated by the amygdala (see McGaugh, 2000, for review). Numerous lines of evidence converge on the conclusion that the amygdala is critical for the emotional memory

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doi:10.1016/j.neuropsychologia.2003.11.011

enhancement effect. Patients with amygdalar lesions, often as a result of a congenital disease (*Urbach Wiethe* disease), show either no enhancement effect, or a significantly reduced effect (Adolphs, Cahill, Schul, & Babinsky, 1997; Adolphs, Tranel, & Denburg, 2000; Buchanan, Denburg, Tranel, & Adolphs, 2001; Cahill, Babinsky, Markowitsch, & McGaugh, 1995). Additional evidence implicating the amygdala in the emotional memory enhancement effect comes from neuroimaging studies: The amygdala has frequently been found to be active during encoding of emotional stimuli (Hamann, 2001; Dolan, Lane, Chua, & Fletcher, 2000; Canli, Desmond, Zhao, & Gabrieli, 2002; Maddock, Garrett, & Buonocore, 2003), and the magnitude of amygdala activation during encoding correlates with the likelihood of retrieving emotional information (Cahill et al., 1996; Canli, Zhao, Brewer, Gabrieli, & Cahill, 2000; Hamann, Ely, Grafton, & Kilts, 1999). Arousal-related hormone release is proposed to modulate amygdalar function which, in turn, interacts with other medial temporal-lobe (MTL) structures to modulate consolidation processes (see McGaugh, 2000, for review).

Although the amygdala likely exerts its influence via modulation of other MTL structures, even individuals with severe MTL damage (e.g. amnesic patients) show a memory benefit for emotional as compared to neutral stimuli (Hamann, Cahill, & Squire, 1997; Hamann, Cahill, McGaugh, & Squire, 1997). Thus, the critical MTL region for the enhancement effect appears to be the amygdala (although the declarative memory enhancement for emotional information may also rely on the orbitofrontal cortex and cingulate gyrus; Cahill et al., 1995; Phelps, LaBar, & Spencer, 1997; Maddock et al., 2003).

2. Emotional memory in Alzheimer disease

The hallmark feature of Alzheimer disease is the dramatic memory deficit that occurs early in the disease. This deficit is presumed to result from neuropathological changes (intracellular neurofibrillary tangles and extracellular amyloid plaques) in MTL regions that are critical for declarative memory. Although this deficit in declarative memory has been recognized as a critical feature of Alzheimer disease for nearly a century, only recently have investigators begun to question whether AD affects the modulation of memory by emotion. Specifically, do AD patients demonstrate a normal emotional memory enhancement effect?

The handful of studies that have investigated this question have found mixed results. On the one hand, a few studies have demonstrated a marked impairment in the enhancement effect for negative pictures (Abrisqueta-Gomez, Bueno, Oliveira, & Bertolucci, 2002; Hamann, Monarch, & Goldstein, 2000; Kensinger, Brierley, Medford, Growdon, & Corkin, 2002), positive pictures (Kensinger et al., 2002; Abrisqueta-Gomez et al., 2002), negative and positive words, and negative sentences (Kensinger et al., 2002). In addition to these changes in emotionally influenced declarative memory, AD patients have also demonstrated impairments in emotionally mediated implicit memory (fear conditioning; Hamann, Monarch, & Goldstein, 2002; affective priming for positive stimuli, Padovan, Versace, Thomas-Anterion, & Laurent, 2002). In contrast, other researchers have demonstrated a relatively intact enhancement effect for positive pictures (Hamann et al., 2000), negative stories or film clips (Boller et al., 2002; Kazui et al., 2000; Moayeri, Cahill, Jin, & Potkin, 2000), and a real-life event (the Kobe earthquake; Ikeda et al., 1998; Mori et al., 1999).

A number of explanations could reconcile these contradictory findings. At a broad level, there are two possibilities. One is that the differences across studies stem from differences in the patient populations. All prior studies had relatively small samples of AD patients; thus, differences between studies could have been a result of sample differences (e.g. ratio of men to women, or disease severity). For example, it is plausible to propose that the magnitude of the enhancement effect in AD relates to the extent of amygdalar atrophy (see Mori et al., 1999, for data supporting this view). AD results in significant atrophy to the amygdala (Chan et al., 2001; Galton et al., 2001; Jack et al., 1999; Scott, DeKosky, & Scheff, 1991; Smith et al., 1999), as well as in an accumulation of neuritic plaques and neurofibrillary tangles in this structure (Arriagada, Growdon, Hedley-Whyte, & Hyman, 1992; Unger, Lapham, McNeill, Eskin, & Hamill, 1991; Vogt, Human, Van Hoesen, & Damasio, 1990). Patients with mild AD, if they have less amygdalar atrophy, may show a greater enhancement effect than patients who have progressed to more moderate stages of the disease, accompanied by greater amygdalar atrophy (see Kazui et al., 2000, for additional discussion). To address issues due to differences in sample, we tested a large group of AD patients ($N = 80$). This large sample allowed us to assess differences due to participant characteristics, such as age, sex, and disease severity. This large sample also could help to address the possibility that, in prior studies with small sample sizes, low statistical power may have prevented the detection of small enhancement effects in the AD patients. A larger sample size could ameliorate this problem: With 80 AD patients, even small enhancement effects could result in statistically significant differences between retention of emotional versus neutral information.

The second possible explanation is that differences in the stimuli have led to different findings. Numerous stimulus dimensions could affect the results: valence, arousal, encoding instructions (e.g. incidental versus intentional), viewing time, or semantic coherency. In the present investigation, we addressed the possible contributions of the latter two variables. We were interested in these factors because the prior studies that found blunted emotional memory enhancement in AD used sets of briefly presented stimuli that lacked semantic coherence (e.g. strings of unrelated words; a series of unrelated pictures each presented for a few seconds). In contrast, the majority of studies that found an enhancement

effect in AD used stimuli with semantic coherence (e.g. a real-life event; a narrated slide show) that were processed for longer periods of time. Thus, this type of coherency of the stimuli, or the amount of time for which the stimuli were viewed, could affect whether AD patients demonstrate an enhancement effect. The present study used short verbal descriptions of events (either emotionally negative or neutral), with internal semantic coherence, viewed for approximately 30 s (much longer than the typical presentation time for single words or pictures). The critical question was whether this group of AD patients would show an emotional memory enhancement effect, and whether the magnitude of the enhancement effect would be related to group characteristics.

3. Methods

3.1. Participants

The participants comprised 37 young adults, 51 older adults, and 80 AD patients (Table 1). Young adults were MIT or Harvard undergraduate or graduate students, or young adults living in the greater Boston area. Older adults were recruited from the Harvard Cooperative on Aging, the MIT Alumni Association, or via fliers posted throughout the greater Boston area. No participants were depressed. All scores on the Geriatric Depression Scale, Short Form (Sheikh & Yesavage, 1986) were less than five (scores of less than seven are considered normal). AD patients were tested as part of their neuropsychological assessment at the Memory Disorders Unit of the Massachusetts General Hospital. The clinical diagnosis of AD was established in accordance with the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) and the National Institute of Neurological and Communicative Disorders and Stroke, and the Alzheimer Disease and Related Disorders Association (McKhann et al., 1984) criteria. The Mini Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) scores of the AD patients were comparable to those in a number of prior studies of emotional memory (Kazui, Mori, Hashimoto, & Hirono, 2003: mean of 23.3; Kazui et al., 2000: mean of 22.6; Kensinger et al., 2002: mean of 24.5, unpublished data), and were somewhat higher than in a few other studies (Mori et al., 1999: mean of 17.1; Boller et al., 2002: mean of 19.6).

Table 1
Participant characteristics

Group	*N* (M/F)	Age (mean, S.D.)	MMSE* (mean, S.D.)
Young	37 (19/18)	20.4 (3.2)	Not measured
Older (10 min delay)	33 (17/16)	71.3 (8.3)	29.4 (0.8)
Older (24 h delay)	18 (8/10)	68.4 (6.8)	29.1 (0.94)
AD	80(33/47)	71.9 (9.4)	23.2 (4.9)

* Mini Mental State Exam; Folstein et al., 1975.

3.2. Task design and administration

3.2.1. Verbal event descriptions

The NYU stories (Randt, Brown, & Osborne, 1981) are standardized tests of declarative memory. There are six verbal descriptions, each telling of an event that occurred on a particular day of the week. The typical procedure for administration is as follows: participants read a description aloud, and are then immediately asked to recall orally everything they can about the event's occurrence. One point is allotted for each word that they correctly recall from the event description, for a total of 20 possible points[1]. They then take a 7-item written recognition test about the event description. After a 10 min delay, they are again prompted to recall orally as much as possible. Following this delayed recall, another 7-item written recognition task is given (with different questions from those given in the immediate recognition test), in which participants are asked to choose the correct answer from among three distractors (e.g. "On what day of the week did the event occur? (a) Tuesday (b) Wednesday (c) Friday (d) Saturday") Participants circle their response. For each of the six NYU stories, we designed a neutral version by replacing all of the emotional words with neutral words matched in word length, word frequency, and familiarity (Kuchera & Francis, 1967) (Appendix A).

Participants (37 young adults, 33 older adults, and 80 AD patients) were tested on a description of one emotional and one neutral event (the event read first, and the particular events tested, were pseudorandomized across participants). During the 10 min delay period, participants performed non-verbal tasks (e.g. drawing of figures, administered as part of a neuropsychological series). The description of the second event was read to the participant approximately 45 min after completion of the memory tasks associated with the description of the first event.

In an attempt to equate the overall memory performance of the older adults with that of the AD patients, we introduced a 24 h delayed recall and recognition task for a new group of 18 older adults. Thus, these adults performed the immediate recall and recognition task and a delayed recall and recognition task after a 24 h delay. The goal was to create a condition in which the overall memory performance of the older adults (at the 24 h delay) would be equated with the memory performance of the AD patients at the 10 min delay.

3.2.2. Emotional ratings

A subset of participants (30 young adults, 23 older adults, 29 AD patients) rated the stimuli for their valence (how pos-

[1] We scored the data in two ways: A "verbatim" scoring method, whereby the identical words used in the description were required to receive a point, and a "gist"-based scoring method, where points were given if the correct thematic information was provided. The results did not differ qualitatively for the two scoring systems, and we report here the data from the "verbatim" scoring method only.

itive or negative) and their arousal (how calming–soothing or exciting–agitating). These ratings were collected at the end of the experiment, after all the memory tasks had been completed. The MMSE scores for this subset of older adults (mean = 29.7, S.D. = 56) and AD patients (mean = 23.2, S.D. = 3.96) were similar to those of the larger group for whom memory scores are reported.

3.2.3. Control tasks

In addition to the administration of the NYU stories, older adults and AD patients were also given a number of tasks to assess their overall cognitive function. These tests included:

3.2.3.1. Mini-Mental State Exam (Folstein et al., 1975). Participants were given a series of questions to assess their orientation in space and time (10 points), their ability to repeat names of objects (3 points) and to later recall those object names (3 points), to spell a word backwards, as a demonstration of attention and calculation skills (5 points), and to demonstrate language ability (9 points). Thus, scores on the MMSE can range from 0 to 30 points. Scores below 24 are indicative of dementia; scores between 24 and 26 are often considered borderline; and scores above 26 are considered normal.

3.2.3.2. Boston Naming Test (Huff, Collins, Corkin, & Rosen, 1986; Kaplan, Goodglass, & Weintraub, 1978). Participants viewed 42 black-and-white line drawings of objects and were asked to produce the name of the object orally. One point was given for each item correctly named.

3.2.3.3. Category fluency (Martin & Fedio, 1983; Newcombe, 1969). Participants generated as many words as possible in 60 s that were exemplars of a particular category (animals or clothing). Categories were given one at a time. Participants were instructed not to repeat any exemplars. The score for each category was the total number of unique exemplars generated (i.e. excluding perseverations). We then took the mean of the two categories to produce the final fluency score.

3.2.3.4. Letter fluency (Benton & Hamsher, 1976). Task administration and scoring were identical to category fluency, with the exception that participants orally generated as many words as possible in 60 s that began with a particular letter of the alphabet (F or S).

3.3. Data analysis

For the emotional rating scores, ANOVAs and *t*-tests were conducted separately for each group. ANOVAs included sex (male, female) as a between-subject factor, and emotion (negative, neutral) and rating (valence, arousal) as within-subject factors. Additional ANOVAs and *t*-tests were conducted to compare the young and older adults, and the older adults and AD patients. These ANOVAs were conducted as described above, but with group as an additional between-subjects factor.

We also conducted ANOVAs and *t*-tests on the memory scores for each group with sex (male, female) as a between-subject factor, and emotion (negative, neutral) and delay (immediate, delayed) as within-subject factors. Recall and recognition scores were analyzed separately. Additional ANOVAs and *t*-tests compared the young and older adults, and the older adults and AD patients. These ANOVAs were as described above, but with group as an additional between-subjects factor. All reported *P*-values are two-tailed.

4. Results

4.1. Emotional ratings

4.1.1. Young and older adults

An ANOVA conducted on the emotional ratings indicated a main effect of event description type (negative, neutral; $F(1, 51) = 21.8$, $P < 0.0001$), whether the rating was on the dimension of arousal or valence ($F(1, 51) = 288.4$, $P < 0.0001$), and an interaction between description type and emotional rating ($F(1, 51) = 327.2$, $P < 0.0001$). There was no main effect of age, nor any interactions with age. Subsequent *t*-tests confirmed that, in general, arousal ratings were more positive than valence ratings, and that arousal was higher for the negative narrative than the neutral narrative, whereas valence was lower for the negative narrative than the neutral one (Table 2).

4.1.2. Older adults and AD patients

An ANOVA conducted on the emotional ratings indicated a main effect of emotion ($F(1, 50) = 22.4$, $P < 0.0001$), an effect of whether the rating was on the dimension of valence or arousal ($F(1, 50) = 219.6$, $P < 0.0001$), and an

Table 2
Emotional ratings (mean, S.E.) as a function of group

Group	Negative event description		Negative event description	
	Arousal[b]	Valence[c]	Arousal	Valence
Young adults ($N = 30$)	4.37 (0.37)	−5.53 (0.23)	0.00 (0.49)	1.87 (0.35)
Older adults[a] ($N = 23$)	4.43 (0.34)	−5.65 (0.38)	0.09 (0.59)	1.91 (0.49)
AD patients ($N = 29$)	4.41 (0.45)	−5.24 (0.36)	−0.24 (0.37)	2.10 (0.50)

[a] Because there were no significant differences in ratings given in the two groups of older adults, we combined the data from older adults tested at the 10 min and 24 h delay intervals.

[b] Arousal ratings ranged from –7 (extremely calming or soothing) to 7 (extremely exciting or agitating).

[c] Valence ratings ranged from –7 (extremely negative) to 7 (extremely positive).

interaction between emotion and rating ($F(1, 50) = 243.9$, $P < 0.0001$). Critically, there was no significant main effect of group, nor any significant interactions (all $P > 0.2$). These results indicate that the AD patients' ratings of emotional content were similar to those of the young and older adults: the AD patients were not impaired in their ability to perceive the emotional information as being low in valence, and high in arousal (Table 2).

4.2. Memory performance

4.2.1. Young adults

4.2.1.1. Recall. An ANOVA conducted on the recall scores indicated a main effect of emotion ($F(1, 36) = 7.15$, $P < 0.01$) and delay ($F(1, 36) = 52.91$, $P < 0.0001$), and no interactions. Subsequent *t*-tests indicated that young adults showed better recall for the negative event description than the neutral event description, both with immediate recall ($t(36) = 2.29$, $P < 0.05$) and with delayed recall ($t(36) = 3.23$, $P < 0.01$). Recall scores for the negative event ($t(36) = 5.07$, $P < 0.0001$) and the neutral event ($t(36) = 7.36$, $P < 0.0001$) were lower after the delay than when tested immediately (Table 3).

4.2.1.2. Recognition. An ANOVA conducted on the recognition scores indicated a marginal main effect of emotion ($F(1, 36) = 3.59$, $P < 0.07$) and a significant main effect of delay ($F(1, 36) = 51.55$, $P < 0.0001$). No interactions were significant. Subsequent *t*-tests indicated that young adults showed marginally better recognition for the negative description than the neutral description when tested after a delay ($t(36) = 1.72$, $P < 0.10$), but no difference when recognition was assessed immediately after presentation of the event descriptions. Recognition scores for the negative description ($t(36) = 5.15$, $P < 0.0001$) and the neutral description ($t(36) = 7.89$, $P < 0.0001$) were lower after the delay than when participants were tested immediately.

4.2.2. Older adults (immediate and 10 min delay)

4.2.2.1. Recall. An ANOVA conducted on the recall scores indicated main effects of emotion ($F(1, 32) = 7.23$, $P < 0.01$) and delay ($F(1, 32) = 30.17$, $P < 0.0001$). No other main effects or interactions were significant. Subsequent *t*-tests indicated that older adults showed better recall for the negative event description than the neutral event description, with immediate recall ($t(32) = 2.24$, $P < 0.05$) and delayed recall ($t(32) = 2.24$, $P < 0.05$). Recall scores for the negative stimuli ($t(32) = 6.41$, $P < 0.0001$) and the neutral stimuli ($t(32) = 5.76$, $P < 0.0001$) were higher when assessed immediately than after a delay (Table 3).

4.2.2.2. Recognition. An ANOVA conducted on the recognition scores indicated main effects of emotion ($F(1, 31) = 15.78$, $P < 0.0001$) and delay ($F(1, 32) = 50.76$, $P < 0.0001$), and a significant interaction between the two ($F(1, 32) = 7.99$, $P < 0.01$). Subsequent *t*-tests indicated that older adults had higher recognition scores for the negative event description than the neutral event description after a delay ($t(32) = 3.88$, $P < 0.0001$), but had similar recognition scores for the two events immediately after event presentation.

4.2.3. Older adults (immediate and 24 h delay)

4.2.3.1. Recall. An ANOVA conducted on the recall scores indicated a main effect of event type (negative, neutral; $F(1, 17) = 55.63$, $P < 0.0001$) and of delay ($F(1, 17) = 117.78$, $P < 0.0001$), and no interaction between event type and delay ($F < 2$, $P > 0.15$). Subsequent *t*-tests confirmed that this older adult group showed higher immediate ($t(17) = 5.82$, $P < 0.0001$) and delayed ($t(17) = 5.16$, $P < 0.0001$) recall scores for the details of the negative event than for the neutral event (Table 3). Thus, the 24 h delay did not appear to attenuate the emotional memory enhancement effect.

4.2.3.2. Recognition. An ANOVA conducted on the recognition scores indicated an effect of event type ($F(1, 17) = 4.48$, $P < 0.05$), an effect of delay ($F(1, 17) = 119.03$, $P < 0.0001$), and an interaction between event description and delay ($F(1, 17) = 6.08$, $P < 0.05$). Subsequent *t*-tests confirmed that this older adult group showed significantly better delayed recognition memory for the description of the negative event as compared to the neutral one ($t(17) = 3.7$, $P < 0.01$), whereas the immediate recognition scores were similar for the descriptions of the negative and neutral events ($t(17) < 1$, $P > 0.7$).

Table 3
Mean immediate (immed) and delay recall and recognition (recog) scores (SE) as a function of event description (negative or neutral) and participant group

Group	Immed recall		Delayed recall		Immed recog		Delayed recog	
	Negative	Neutral	Negative	Neutral	Negative	Neutral	Negative	Neutral
Young	11.65 (0.73)	10.19 (0.53)	9.51 (0.64)	7.68 (0.43)	6.76 (0.07)	6.59 (0.09)	5.68 (0.22)	5.16 (0.22)
Older (10 min delay)	9.48 (0.43)	7.97 (0.57)	7.82 (0.46)	6.48 (0.53)	6.76 (0.08)	6.61 (0.10)	5.48 (0.25)	4.67 (0.22)
Older (24 h delay)	10.17 (0.60)	7.33 (0.53)	3.94 (0.37)	1.94 (0.22)	5.94 (0.22)	6.06 (0.21)	3.89 (0.25)	2.83 (0.22)
AD	4.46 (0.45)	4.31 (0.42)	2.64 (0.43)	2.85 (0.44)	3.89 (0.20)	3.72 (0.20)	3.31 (0.23)	3.49 (0.23)

4.2.4. AD patients

4.2.4.1. Recall. An ANOVA conducted on the recall scores indicated a main effect of delay ($F(1, 79) = 53.43$, $P < 0.0001$). We found no main effect or interaction with emotion. Subsequent *t*-tests indicated that AD patients had poorer recall scores for the negative event ($t(79) = 5.99$, $P < 0.0001$) and neutral event ($t(79) = 6.08$, $P < 0.0001$) following a delay as compared to immediately after event presentation (Table 3).

4.2.4.2. Recognition. An ANOVA conducted on the recognition scores indicated no main effects, along with a marginal interaction between emotion and delay ($F(1, 79) = 3.52$, $P < 0.07$). This interaction arose because the AD patients recognized non-significantly more items from the negative event than the neutral event when assessed immediately ($t(79) = 0.87$, $P > 0.3$), but recognized non-significantly more items from the neutral event than the negative event following a delay ($t(79) = 0.88$, $P > 0.3$).

4.2.5. Comparison of young and older adults

4.2.5.1. Recall. An ANOVA comparing the recall performance of young and older adults indicated significant main effects of emotion ($F(1, 68) = 14.45$, $P < 0.0001$) and delay ($F(1, 68) = 79.94$, $P < 0.0001$), and a significant interaction between group and delay ($F(1, 68) = 6.28$, $P < 0.05$). Subsequent *t*-tests indicated that the interaction between group and delay resulted because the young and older adults had more similar recall scores following the delay ($t(69) = 2.01$, $P < 0.07$) than when assessed immediately after event presentation ($t(69) = 2.65$, $P < 0.01$).

4.2.5.2. Recognition. An ANOVA comparing the recognition performance of young and older adults indicated a significant effect of emotion ($F(1, 68) = 14.30$, $P < 0.0001$) and delay ($F(1, 68) = 102.99$, $P < 0.0001$), and a significant interaction between emotion and delay ($F(1, 68) = 7.10$, $P < 0.01$). The interaction between emotion and delay resulted because immediate recognition scores were similar for the negative and neutral event descriptions, whereas delayed recognition scores were higher for the negative than for the neutral event description. Importantly, we found no main effects or interactions with group.

4.2.6. Comparison of older adults and AD patients

4.2.6.1. Recall. An ANOVA comparing the recall performance of older adults and AD patients indicated a significant effect of group ($F(1, 111) = 61.54$, $P < 0.0001$), delay ($F(1, 111) = 58.14$, $P < 0.0001$), and emotion ($F(1, 111) = 5.56$, $P < 0.05$). An ANOVA revealed a significant interaction between group and emotion ($F(1,111)=9.57$, $P < 0.01$). Subsequent *t*-tests indicated that this interaction stemmed from the fact that older adults showed an effect of emotion on recall scores ($t(32) = 2.24$, $P < 0.05$), whereas AD patients did not.

4.2.6.2. Recognition. An ANOVA comparing the recognition performance of older adults and AD patients indicated a main effect of group ($F(1, 111) = 55.51$, $P < 0.0001$), and delay ($F(1, 111) = 84.05$, $P < 0.0001$), and a marginal effect of emotion ($F(1, 111) = 3.26$, $P < 0.08$). An ANOVA also indicated a marginal interaction between group and emotion ($F(1, 111) = 3.87$, $P < 0.06$), along with a significant interaction between group and delay ($F(1, 111) = 28.85$, $P < 0.0001$), and a three-way interaction between group, emotion, and delay ($F(1, 111) = 9.98$, $P < 0.01$). The marginal interaction between group and emotion reflected the fact that older adults, but not AD patients, benefited from the emotional content of the narrative. The interaction between group and delay resulted because older adults showed poorer memory following a delay than when tested immediately, whereas the AD patients did not show this effect of delay. The three-way interaction between group, emotion, and delay resulted because older adults showed similar recognition scores for negative and neutral stimuli when memory was assessed immediately, but better recognition scores for the negative than the neutral event when assessed after a delay. In contrast, the AD patients did not show a difference in their recognition scores as a function of event type, either when tested immediately or following a delay.

4.2.7. Comparison of AD patients with older adults at 24 h delay

4.2.7.1. Recall. A potential concern was that the higher performance of the older adults, as compared to the AD patients, could create difficulties when measuring the enhancement effect. In particular, perhaps the inability to detect an emotional enhancement effect in the AD group resulted from their overall low level of memory performance. To address this possibility, a group of older adults ($n = 18$) completed a recall and recognition test after a 24 h delay, as well as immediately after reading the event descriptions.

An ANOVA conducted on the immediate and delayed recall scores indicated a main effect of group ($F(1, 96) = 7.40$, $P < 0.01$), event description type ($F(1, 96) = 13.86$, $P < 0.0001$), and delay ($F(1, 96) = 190.61$, $P < 0.0001$), along with significant interactions between group and event type ($F(1, 96) = 18.71$, $P < 0.0001$), and group and delay ($F(1, 96) = 59.5$, $P < 0.0001$). Critically, when an ANOVA was conducted only on the delayed recall scores, there was no longer a significant effect of group ($F < 1$, $P > 0.8$), while there was still a significant effect of event type ($F(1, 96) = 10.4$, $P < 0.05$) and an interaction between group and event type ($F(1, 96) = 15.93$,

$P < 0.0001$). Thus, even under conditions where the overall memory performance of the older adults and AD patients were equated, the older adults showed a memory benefit for the verbal description of the negative event as compared to the neutral one, whereas the AD patients did not (Table 3).

4.2.7.2. Recognition. An ANOVA conducted on the immediate and delayed recognition scores indicated a significant effect of group ($F(1, 96) = 7.30$, $P < 0.01$), a significant effect of delay ($F(1, 96) = 155.81$, $P < 0.0001$), and a significant interaction of group and delay ($F(1, 96) = 83.76$, $P < 0.0001$). There was no effect of event description ($F < 2$, $P > 0.2$), likely because the immediate recognition scores did not differ based on the event content, for either the older adults nor the AD patients. While the AD group did not show an effect of event content on their delayed recognition scores, the older adults did. Importantly, when an ANOVA was conducted on only the delayed recognition scores, there was no effect of group ($F < 1$, $P > 0.9$), an effect of event type ($F(1, 96) = 4.01$, $P < 0.05$), and an interaction between group and event type ($F(1, 96) = 7.84$, $P < 0.01$). Thus, as with the recall scores, the delay manipulation succeed in matching the overall memory performance of the older adults and the AD patients. Nevertheless, the older adults showed significantly better recognition performance for the descriptions of the negative event than for the neutral one ($t(17) = 3.7$, $P < 0.01$), whereas the AD patients did not ($t(79) < 1$, $P > 0.3$).

4.2.8. Relation of enhancement effect to cognitive function in the AD group

Pearson correlations indicated no significant relation between scores on any of the neuropsychological tasks (MMSE, Boston Naming Test, category fluency, letter fluency), or age, and the extent of memory benefit for negative as compared to neutral event descriptions (all $r < 0.18$, $P > 0.2$). An ANOVA indicated that sex was also unrelated to the magnitude of the emotional enhancement effect ($P > 0.3$). These results suggest that the blunted enhancement effect occurs relatively early in the disease, and thus does not correlate with measures of disease severity (MMSE scores) or semantic memory. One important caveat to note is that only measures of verbal memory were assessed; it is possible that performance on tasks of nonverbal memory would show a correlation with the magnitude of the emotional enhancement effect (see Kazui et al., 2003).

5. Discussion

In this study of emotional memory, young adults showed significantly better memory for an emotional event description as compared to a neutral event description. This memory benefit was present on an immediate recall task, as well as on a 10 min delayed recall task[2]. Although part of the modulatory effect of emotion on memory is thought to occur via an amygdalo-hippocampal interaction that boosts consolidation of emotional information (Cahill, 1999; Cahill & McGaugh, 1998; McGaugh, 2000; McGaugh, Cahill, & Roozendaal, 1996), the enhancement effect in this study and prior studies using short retention intervals (Burke, Heuer, & Reisberg, 1992; Christianson, Loftus, Hoffman, & Loftus, 1991; Ferre, 2002; Kensinger et al., 2002; Wessel, Van der Kooy, & Mercklebach, 2000) suggests that there may be additional effects of emotion that act at the encoding stage to boost even immediate recall or recognition performance. The neural underpinnings of this enhancement have not been elucidated. Recent neuroimaging data have been mixed with regard to whether amygdalar activation correlates with short-term retention of emotional information (Kensinger & Corkin, in press; Kensinger & Corkin, 2003a; see Hamann, 2001, for further discussion), or whether regions outside of the amygdala may mediate this effect (Tabert et al., 2001).

The older adults also showed memory enhancement for the emotional event description, and the magnitude of their enhancement was comparable to that in the young adults. This study, therefore, provides evidence that the emotional memory enhancement effect is preserved with normal aging (see also Kensinger et al., 2002; Denburg, Buchanan, Tranel, & Adolphs, 2003; but see Charles, Mather, & Carstensen, 2003). It is unclear whether this spared enhancement indicates that amygdala function is preserved in healthy aging. Although the amygdala shows signs of atrophy with normal aging (Smith et al., 1999), the extent of the atrophy may not be sufficient to disrupt the emotional memory enhancement effect. It is also possible, however, that cognitive processes that do not rely on the amygdala (e.g. increased elaboration of emotional information) contribute to the enhanced memory for emotional information in older adults.

In marked contrast to the young and older adults, the AD patients showed no memory benefit for the verbal description of the emotional event. Critically, the AD patients were capable of processing the emotional information: they rated the descriptions of emotional events as low in valence (negative) and high in arousal, just as did the young and older adults. This finding is consistent with prior studies indicating intact emotional ratings in AD (Kazui et al., 2000, 2003), and suggests that the disrupted memory enhancement in AD

[2] Because of the successive memory tasks given (e.g. recall–recognition–recall–recognition), it is possible that performance on later memory tasks was affected by carryover effects from the first task. Nevertheless, the conclusions drawn from this study are supported even if we only consider the first recall task, which would not have been influenced by any carryover effects. Another possibility is that performance on the later tasks was influenced by the presence of a filled delay period; participants performed unrelated tasks during the delay interval. However, this filled delay interval could not have affected performance on the immediate recall task.

does not result from an inability to cognitively process emotional information.[3]

The lack of an enhancement effect in the AD group suggests several conclusions. First, the inability of some prior studies to demonstrate memory enhancement to negative stimuli (Abrisqueta-Gomez et al., 2002; Hamann et al., 2000; Kensinger et al., 2002), likely is not explained simply by the relatively low statistical power in those studies. In the present study, even with a large sample of AD patients, we could not detect an emotional memory enhancement effect. In the young and older adult groups, enhancement effects were detectable in samples that were smaller than the AD group. Similarly, in the older adult group tested after a 24 h delay, memory enhancement effects were detected even when overall levels of memory performance were equated with the AD group.

Second, the findings of the present study suggest that the lack of enhancement in some prior studies likely was not due to the relatively low semantic coherence of the stimuli, or to the short period of time for which each stimulus was processed (e.g. a few seconds). Even with stimuli with more internal coherence (i.e. verbal descriptions of events) viewed for about 30 s, AD patients did not demonstrate any memory benefit for emotional event descriptions.

Third, group characteristics (e.g. sex, disease severity) did not correlate significantly with the magnitude of the enhancement effect, suggesting that differences among the samples of AD patients do not explain the mixed findings with regard to whether or not AD patients demonstrate emotional memory enhancement (but see Kazui et al., 2003, for evidence that the magnitude of emotional memory enhancement correlates with scores on visual memory tasks).

What, then, could account for the contradictory findings? A few possibilities remain. First, perhaps differences in arousal level affect whether or not AD patients demonstrate enhancement. In the present study, although all participant groups rated the stimuli as having moderate arousal, the stimuli probably did not elicit the same level of arousal as a real-life event, or a narrated slide show, the stimuli that have most frequently been associated with a preserved enhancement effect in AD (Ikeda et al., 1998; Kazui et al., 2000, 2003; Mori et al., 1999). It is plausible that, given a sufficient level of arousal, AD patients may show an emotional memory enhancement effect (but see Ikeda et al., 1998; Mori et al., 1999 for evidence that even when memory for a real-life emotional event is assessed, the enhancement effect is not normal). Future studies, that parametrically vary the arousal level of stimuli, will be needed to examine whether stimuli with sufficient arousal allow AD patients to benefit from the emotional salience of the stimuli. Nevertheless, the results of the present study indicate that there are some emotional stimuli that are sufficiently emotional to elicit memory enhancement in young and older adults, but not in AD patients.

Second, differences in modality may be important. Real-life events, and narrated slide shows, combine multiple modalities of information (visual, auditory), and it may be that this blending of information creates a richer memory trace, thereby providing a greater demonstration of the enhancement effect. The present study cannot distinguish the contribution of sensory complexity to the enhancement effect; future studies varying stimulus modalities will be required to further our understanding of how these stimulus characteristics contribute to the emotional memory enhancement effect in AD.

Additional experiments will also be required to elucidate the neural underpinnings of the emotional enhancement effect in AD. One possibility is that the amygdalar atrophy that occurs in AD (see Chow & Cummings, 2000, for review) may be sufficient to disrupt this effect. This hypothesis is supported by a number of observations. First, in mild AD, the amygdala and neighboring MTL regions are the regions most affected. The emotional memory disruption in AD likely does not result from MTL damage outside of the amygdala, because amnesic patients, with spared amygdala but damage to other MTL regions, show preserved enhancement effects (Hamann, Cahill, & Squire, 1997; Hamann, Cahill, McGaugh, et al., 1997). Thus, amygdalar atrophy is a likely link to the diminished emotional memory enhancement. Second, although even in mild AD, some neuropathological changes are seen beyond the MTL (e.g. in temporal neocortex; Price & Morris, 1999; Arriagada et al., 1992), if deficits in neocortical areas were accountable for the blunted emotional enhancement effect, we should have seen significant correlations between the amount of emotional enhancement and scores on semantic memory tasks thought to rely on temporal neocortex (e.g. Schmolck, Kensinger, Corkin, & Squire, 2002). Third, because AD patients have shown impaired acquisition of fear conditioning (Hamann et al., 2002), the altered emotional memory deficits do not appear to be limited to cognitive or neural processes related to declarative memory. Further, a study examining structure-function correlations (Mori et al., 1999) found that amygdalar volume was the best predictor of emotional memory enhancement in AD. This prior study assessed memory for a real-life event, following a 2 month delay, however, and it is unclear whether this correlation will hold for the majority of other studies that have examined emotional memory enhancement in AD patients using a brief delay (of 10 min or less). While it is possible that the amygdalar atrophy in AD is also responsible for the blunted emotional enhancement on these tasks with short delay intervals, it may also be that atrophy in regions outside of the amygdala underlies this alteration in emotional memory.

Regardless of the neuroanatomical correlates, the results of this experiment provide evidence that there are some

[3] This finding does not eliminate the possibility that while their cognitive processing of the stimuli is normal, their physiological responses are not. Future studies, using physiological recording, will be needed to examine whether AD patients demonstrate normal biological, as well as cognitive, responses to emotional stimuli.

verbal stimuli for which AD patients do not show emotional memory enhancement but for which young and older adults do. This finding held across the AD group; there were no group characteristics (sex, age, or dementia severity) that related to the presence of the enhancement effect. The lack of significant correlations must be interpreted cautiously, however, because they could have resulted from the relatively low variance in the enhancement scores for the AD patients. Nevertheless, these results suggest that there is not an overwhelming relation between group characteristics and the magnitude of the enhancement effect. In conclusion, the results of this study provide evidence that there are some, semantically coherent, verbal stimuli that are sufficiently emotional to elicit a memory enhancement effect in healthy young and older adults, but for which AD patients do not show emotion-related memory modulation.

Acknowledgements

Supported by NIH grants AG05134 (to the Massachusetts Alzheimer Disease Research Center) and AG021525, and by a Howard Hughes Medical Institute Predoctoral Fellowship (E.A.K.). We thank Anne Krendl, Gail O'Kane, Olivier Piguet, and Sarah Steinvorth for helpful discussion and Martine Lamy for assistance testing young and older adult participants.

Appendix A

Example of a negative and a neutral event description.

Negative event description	Neutral event description
On Saturday, 2nd August, in Seattle, Washington a shattering earthquake struck the theater district on Sterling Avenue, trapping 20 performers and smothering 5 stage hands plus 12 spectators.	On Saturday, 2nd August, in Seattle, Washington, a newspaper reporter visited the theater district on Sterling Avenue observing 20 performers and interviewing 5 stage hands plus 12 spectators.

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