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Memory for Emotional Words Following Unilateral Temporal Lobectomy

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We recently reported that patients who had received unilateral temporal lobectomy, including the amygdala and hippocampus, show impaired acquisition in a fear conditioning task (LaBar, LeDoux, Spencer, & Phelps, 1995), indicating a deficit in emotional memory. In the present paper, we examined performance of these patients on two verbal, emotional memory tasks in an effort to determine the extent of this deficit. In Experiment 1, subjects were asked to recall emotional and non-emotional words. In Experiment 2, subjects were asked to recall neutral words which were embedded in emotional and non-emotional sentence contexts. Both temporal lobectomy subjects and normal controls showed enhanced recall for emotional words (Experiment 1) and enhanced recall for neutral words embedded in emotional sentence contexts (Experiment 2). These results suggest that the deficit seen in emotional memory following unilateral temporal lobectomy is not a global deficit and may be limited to specific circumstances where emotion influences memory performance. Several hypotheses concerning the discrepancy between the present studies and the fear conditioning results (LaBar et al., 1995) are discussed.

Since the first reports of the famous amnesic patient, H.M., it has been widely accepted that the temporal lobe plays an important role in human memory (Scoville, 1968). The temporal lobe structure most often cited as critical in mnemonic processing is the hippocampus (e.g., O'Keefe & Nadel, 1978; Squire, 1987). Bilateral damage to the hippocampus in humans leads to a severe memory impairment characterized by a deficit in declarative or explicit memory (e.g., Schacter, 1988; Squire, 1986). Recent research with

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non-human animals has suggested another temporal lobe structure, the amygdala, may play a separate role in memory. The animal literature suggests that the role of the amygdala in memory may be limited to situations where emotion influences memory performance (see LeDoux, 1992, for a review). Whether or not the amygdala has a similar function in humans has been difficult to assess due to the scarcity of patients with bilateral amygdala damage who do not concurrently have hippocampal damage and the deficit in declarative or explicit knowledge that follows.

In the present paper, we examine emotional memory in patients with unilateral damage to the medial temporal lobe. These patients have unilateral damage to both the hippocampus and amygdala. It has been well documented that they often demonstrate mild forms of the type of memory deficit typical of bilateral damage to the hippocampus (e.g., Milner, 1970). We have recently reported (LaBar, LeDoux, Spencer, & Phelps, 1995) that they also demonstrate a deficit in fear conditioning, the type of emotional memory deficit that might be consistent with damage to the amygdala. Whether or not this deficit in emotional memory following unilateral temporal lobectomy extends beyond fear conditioning is not known. Given this, examining emotional memory in these patients may provide clues to the unique effects of unilateral amygdala damage and the role of the amygdala in human memory.

BACKGROUND: THE AMYGDALA AND EMOTION

Temporal lobe structures were first implicated in emotional processing in the 1930s. Kluver and Bucy (1937) reported an emotional deficit called ''psychic blindness'' following bilateral removal of the medial temporal lobes. Monkeys who received these lesions were described as no longer exhibiting fear responses to previously threatening stimuli, demonstrating a tendency to investigate the environment orally, and exhibiting hypersexuality. At about the same time, Papez (1937) proposed a circuit underlying emotional processing that included the hippocampus as one of the critical structures.

These early reports, while implicating the importance of the temporal lobe in emotional processing, did not suggest a particular role for the amygdala. Later studies, however, identified the amygdala as a critical component of an emotion system. In a later reformulation of the circuit first proposed by Papez (1937), MacLean (1949) included the amygdala as one of the structures involved. In a further examination of Kluver–Bucy syndrome in monkeys, Weiskrantz (1956) was able to isolate the amygdala as the temporal lobe structure whose damage leads to this emotional deficit. Thus, by the late 1950s it was generally acknowledged that the amygdala was somehow involved in the processing of emotional stimuli.

The nature of the involvement of the amygdala in emotional processing was further specified by several researchers who reported a deficit in fear

conditioning following lesions to the amygdala (e.g., Kapp, Pascoe, & Bixler, 1984; LeDoux, 1990; Davis, 1992). Amygdala lesions have been found to lead to deficits in conditioned bradycardia (Kapp et al., 1984), conditioned blood pressure increases (LeDoux, Farb, & Ruggerio, 1990), conditioned freezing (LeDoux et al., 1990), and potentiation of startle (Davis, 1992). These studies suggest that the amygdala is necessary for assigning emotional valence to a neutral event. In a typical study, rats with and without amygdala lesions are exposed to a tone paired with a footshock (Phillips & LeDoux, 1992). All the rats exhibit a ''freezing'' response when given a footshock. After a few pairings, rats without amygdala lesions exhibit the freezing response in the presence of the tone alone. Rats with amygdala lesions never learn to fear the tone itself, indicating an impairment in learning the emotional significance of the tone. Phillips and LeDoux (1992) have also examined other temporal lobe structures and have shown that while the amygdala may be involved in the acquisition of a fear response, the hippocampus plays a separate role and is necessary for conditioned responses to the learning context.

The research with non-human animals leads to the conclusion that the amygdala may play a unique role in the acquisition and expression of emotional responses, or at least fear, to neutral stimuli. The role of the amygdala in humans is less clear. The famous patient H.M. has a lesion very similar to the one originally described as leading to psychic blindness in monkeys. However, reports of H.M.'s emotional responses indicate that while he may appear somewhat flat in emotional expressiveness, he certainly does not exhibit the lack of fear to threatening stimuli, oral tendencies, and hypersexuality that is typical of Kluver–Bucy syndrome (Scoville, 1968). There are a few reports of patients with amygdala damage who demonstrate symptoms similar to that of psychic blindness, but these patients always have additional cortical damage (see Aggleton, 1992, for a review). This suggests that there may be some differences in the behavioral consequences of amygdala damage between humans and monkeys.

Other studies with humans, however, seem to confirm the role of the amygdala as part of an emotional processing system. There have been reports of recording and stimulating the amygdala in human epileptic patients prior to surgery. Gloor, Oliver, Quensey, Andermann, and Horowitz (1982) found that stimulating the amygdala leads to a sensation of fear or anxiety and physiological changes associated with fear. Halgren (1992) recorded from the amygdala during recall of emotional events and found an increase in firing in a subset of neurons. Both of these studies suggest that in humans the amygdala plays a role in emotional responding.

There are several reports of patients who have received partial or complete lesion of the amygdala, most often in an attempt to control behavioral problems (e.g., aggression, schizophrenia; see Aggleton, 1992, for a review). These patients seem to demonstrate some emotional changes following

amygdala lesions, but these are often difficult to quantify due to the pre-existing behavioral problems. In a case study, Lee et al. (1988) reported that prior to amygdalectomy an overly aggressive patient showed abnormally high Skin Conductance Responses (SCRs) to novel stimuli and did not show SCR habituation to repeated stimuli. Postoperatively, this patient showed normal SCR levels to novel stimuli and normal habituation. Although the aggressive behaviors in these studies are not well defined, these data seem to suggest that the amygdala in humans may be partially involved in emotional, or at least arousal, responses to stimuli.

There are a few reported cases of patients with a congenital disorder called Urbach–Wiethe syndrome that leads to bilateral amygdala damage (Tranel & Hyman, 1990; Babinsky et al., 1993). While these patients may not be ideal case studies since it is likely their brains developed without functioning amygdalae, they still may provide useful clues to the function of the amygdala in humans. Performance on memory tasks with emotional components has been examined in a few of these patients. Bechara et al. (1995) examined patient SM-046 in a fear conditioning paradigm and reported that this patient does not exhibit normal fear conditioning to neutral visual and auditory stimuli. This deficit in fear conditioning is evident despite explicit knowledge of the conditioning parameters. Memory for arousing vs. neutral portions of a story was examined in a second patient, B.P. Cahill, Babinsky, Markowitsch, and McGaugh (1995) presented B.P. and normal controls a story (pictures and narrative) in which the middle portion contained emotional events (an accident and injuries). For normal subjects, recognition of the middle portion of the story was enhanced relative to early and late portions. B.P. failed to show enhanced recall for the arousing, middle portion, but also showed impaired recall of the neutral, late portion indicating a strong primacy effect relative to controls. B.P.'s rating of the emotional content of the story was similar to controls.

It is also reported that one of these patients (SM-046), while not exhibiting any severe deficits in emotional expression, has a specific deficit in recognizing fearful facial expressions (Adolphs, Tranel, Damasio, & Damasio, 1994). Young, Hellawell, Van De Wal, and Johnson (1996) report another patient, with bilateral amygdala damage due to epilepsy and surgery, who also shows a deficit in facial affect processing. In contrast, Hamann et al. (1996) did not find a deficit in recognition of facial expressions in two encephalitic patients with bilateral temporal lobe damage, including the amygdala. They suggest that the deficit may be limited to patients whose the amygdalae are damaged early in development. Although some of the data is problematic, the evidence from patients with Urbach–Wiethe syndrome indicates that the role of the amygdala may be specific to responding to a subset of emotional stimuli and/or emotional memory.

Finally, there is some pharmacological evidence that the human amygdala might mediate enhanced memory during arousal. In a series of studies,

McGaugh and colleagues (see McGaugh, Introini-Collison, Cahill, Kim, & Liang, 1992 for a review) have shown that in rats, activation of β-adrenergic receptors in the amygdala leads to enhanced retention of an inhibitory avoidance response, and blocking the same receptors leads to impaired retention. Learning this inhibitory avoidance response is also impaired by lesions to the amygdala. In a test of this neuromodulatory system in humans (Cahill, Prins, Weber, & McGaugh, 1994), subjects were given propranolol, a β-adrenergic receptor blocker, and asked to remember two stories (narratives with pictures); one that was emotional and arousing, and one that was neutral. Subjects who received a placebo showed enhanced retention for the arousing portions of the emotional story. Subjects who received propranolol did not show this enhanced retention. This evidence is indirect, but nevertheless implies that the amygdala in humans is involved in memory for emotional events.

Although the research on amygdala lesions in humans is mostly limited to a few patients, the results are somewhat consistent with the animal studies in that they suggest the amygdala plays a role in emotional processing and, perhaps, may be primarily involved in emotional memory. The indirect pharmacological evidence (Cahill et al., 1994) further supports this interpretation. The questions that remain are: (1) to what extent can these findings be extended to a larger population of subjects, and (2) does the function of the amygdala extend to other situations where emotion and memory interact in humans? In the present paper we attempt to address these questions by studying patients who have received unilateral temporal lobectomies in an effort to control epilepsy. These patients provide an imperfect model in that they only have unilateral damage to the amygdala and they also have unilateral damage to other temporal lobe structures, including the hippocampus. However, research conducted with non-human animals suggests that the types of memory deficits found following damage to the hippocampus (declarative/relational memory) should differ from the memory deficits found following amygdala lesions (fear conditioning—Phillips and LeDoux, 1992; see also Zola-Morgan, Squire, & Amaral, 1989). There has been extensive research characterizing the type of memory deficit seen following hippocampal damage in humans (e.g., Squire, 1986), which does not appear to extend to emotional tasks (Hamann, Stefanacci, & Squire, 1996; Johnson, Kim, & Risse, 1985). Given our understanding of the type of deficit seen with hippocampal damage in humans, examining emotional memory in these patients might allow us to differentiate the mnemonic role of the amygdala.

MEMORY PERFORMANCE FOLLOWING UNILATERAL TEMPORAL LOBECTOMY

Research on memory following unilateral temporal lobectomy has found that some of these patients may have a mild deficit in explicit/declarative

memory, although not nearly as severe as the deficit observed following bilateral temporal lobe damage. It was first reported by Milner (1962, 1965) that patients with unilateral temporal lobe damage show subtle, material-specific memory deficits depending on the side of the lesion. Specifically, those with right temporal lobe damage (nonspeech dominant) were mildly impaired on tests of visual memory; while those with left temporal lobe damage (speech dominant) were mildly impaired on tests of verbal memory. Since these early reports, there have been several studies of memory performance in patients with unilateral temporal lobectomy and these generally confirm the results of Milner (e.g., Novelly et al., 1984; Jones-Gotman, 1986; Sass et al., 1992). Most of these studies use standard neuropsychological tests or similar tests that measure explicit memory performance. These are the types of tests on which one would expect to see deficits following bilateral hippocampal damage.

There have been a few reports of implicit memory performance in temporal lobectomy patients. Blaxton (1992) examined data-driven (i.e., word-fragment completion) and conceptual (i.e., general knowledge) priming in patients with unilateral temporal lobe damage who demonstrated an explicit memory impairment. She found that these patients were intact on data-driven priming tests as well as data-driven explicit tests (i.e., cued recall), but impaired on conceptual priming tasks. A later study by Cermak, Verfaellie, and Chase (1995) suggested that the results obtained by Blaxton (1992) are due to the same deficit observed following bilateral hippocampal damage or other lesions leading to human amnesia.

Another implicit memory task, eyeblink conditioning, has been examined following unilateral temporal lobectomy. Animal models of eyeblink conditioning have found that temporal lobe structures are not necessary in simple eyeblink conditioning (see Lavond, Kim, & Thompson, 1993, for a review), but that the hippocampus seems to be necessary in more complex eyeblink conditioning paradigms (e.g., Berger & Orr, 1983; Solomon, Vander Schaff, Thompson, Weiss, 1986). Gabrielli et al. (1995) have reported that eyeblink conditioning is intact following bilateral hippocampal damage in humans. Consistent with these findings, Daum Channon, and Gray (1992) found that temporal lobectomy patients were intact using a simple discrimination eyeblink paradigm. The patients acquired the conditioned response at the same rate as normal controls. However, when given a conditional discrimination paradigm in which the reinforcement status of the conditioned stimulus (CS) is only apparent when two cues (i.e., a light and tone) are combined, these patients were impaired (Daum, Channon, Polkey, & Gray, 1991). The temporal lobe patients respond to the single CS, the tone, but do not inhibit their response when the tone is preceded by a light indicating that the tone will not be reinforced. This pattern of responding to a single cue, but not complex cues, is consistent with the type of deficit seen following hippocampal lesions in animals (e.g., Cohen & Eichenbaum, 1993; Sutherland & Rudy, 1989).

The studies on memory performance following unilateral temporal lobectomy seem to show subtle deficits in some patients, which is similar in type but not severity to the deficit seen in human amnesia following bilateral hippocampal damage. The finding of intact eyeblink conditioning suggests that temporal lobectomy patients are not impaired on all conditioning tasks, but only the more complex paradigms. In contrast to nonemotional eyeblink conditioning, one might expect a different pattern of results to emerge when an emotional or fear conditioning paradigm is used. As outlined earlier, the animal studies demonstrate that the amygdala may be a critical structure in these fear conditioning tasks. To test this hypothesis, we conducted a series of studies to examine fear conditioning in temporal lobectomy patients with unilateral amygdala damage.

FEAR CONDITIONING FOLLOWING UNILATERAL TEMPORAL LOBECTOMY

In a series of studies, LaBar et al. (1995) examined fear conditioning following unilateral temporal lobectomy. In these studies, the unconditioned stimulus (US) was a series of short, loud white noise bursts and SCR was measured as the unconditioned response (UR). In a simple discrimination paradigm, the reinforced conditioned stimulus (CS+) was a tone and the unreinforced stimulus (CS-) was a second tone. The study was conducted on 22 temporal lobectomy subjects (11 left, 11 right), 22 normal controls, and 3 epileptic patients who had lesions in other brain areas following surgery to control epilepsy.

There was no difference in performance between patients who had received right or left temporal lobectomy so these subjects were combined. There was also no difference in level of SCR and habituation to the unconditioned stimulus, suggesting that the temporal lobectomy subjects show normal responses to arousing stimuli. As can be seen in Fig. 1, the epileptic and non-epileptic control groups showed an increase in SCR to the CS+, relative to the CS−, during acquisition. The temporal lobectomy subjects showed no increase in responding to the CS+ and demonstrated fairly flat responses. In spite of the lack of a learned conditioned response, all but two of the temporal lobe subjects were able to explicitly report that the CS+ was followed by the US, while the CS− was unreinforced.

A second study was conducted using a conditional discrimination paradigm similar to one used by Daum et al. (1991) in their eyeblink conditioning studies. The CS was a single tone. If the tone was preceded by one colored light (e.g., red) it was reinforced (the CS+). If the tone was preceded by the color-opponent light (e.g., green), it was not reinforced (CS−). With this paradigm we found, like Daum et al., that the temporal lobectomy subjects were impaired, in spite of the fact that they could explicitly report the correct stimulus relationships. However, our results differed from Daum et al.'s in

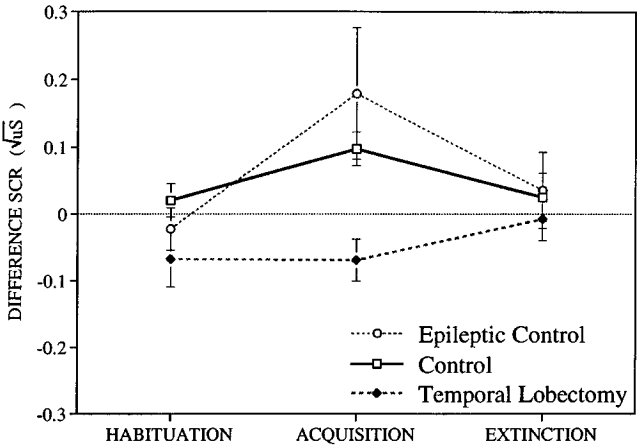

FIG. 1. Simple discrimination conditioning in temporal lobectomy patients, nonepileptic control subjects, and epileptic control subjects with other brain excisions. The data are difference scores obtained by subtracting SCR on CS− (unreinforced) trials from SCR on CS+ (reinforced) trials. On all figures, error bars represent standard error.

the pattern of responding. In the Daum et al. study, the patients were impaired because they overgeneralized and showed an increased response to the CS−. In our study the patients were impaired because they failed to respond to the CS+. This different pattern of results on the conditional discrimination study, combined with the different results in the simple discrimination study, implies that there may be different neural substrates underlying impairments seen in eyeblink and fear conditioning in unilateral temporal lobectomy patients. Specifically, we propose that the deficit seen with fear conditioning is due to the unilateral amygdala damage, whereas the deficit seen in the more complex conditional discrimination eyeblink paradigm may be mediated by the unilateral hippocampal damage.

Consistent with this interpretation is a study of fear conditioning following unilateral amygdala lesions in rats (LaBar & LeDoux, 1996). Using the same US (loud white noise) as the human study described above, LaBar and LeDoux (1996) found that unilateral damage to the amygdala, on both the right and left, leads to an impairment in a conditioned freezing response. These results provide additional support to the notion that it is the amygdala damage that leads to the deficit in fear conditioning for the unilateral temporal lobectomy subjects.

This deficit seen in fear conditioning following unilateral temporal lobectomy is not surprising given the animal research (e.g., LeDoux, 1992) and the impairment reported in the case study of patient SM-046 (Bechara et al., 1995). What is not known is whether this deficit in fear conditioning is

representative of a deficit on all or most emotional memory tasks. Emotional memory, aside from fear conditioning, has not been studied in these patients and fear conditioning is not what one would consider a typical emotional memory task in humans. If one is going to claim that the amygdala has a special role in memory tasks which are influenced by emotion, ideally these patients would show a deficit on several types of emotional memory tasks. In this paper we report two studies examining unilateral temporal lobectomy patients on verbal memory tasks where emotional factors influence memory performance. In Experiment 1, we examine memory for emotional words and in Experiment 2, we examine for memory neutral words embedded in emotional sentences. If the amygdala is involved in all types of emotional memory tasks, we might expect to see a deficit specifically in those situations where emotional factors influence memory performance.

EXPERIMENT 1

Method

Subjects. Twenty-six subjects with medically refractory complex partial seizures of medial temporal lobe origin were studied 2–6 years following unilateral anteromedial en bloc temporal lobe resection (13 left, 13 right). The surgical procedure involved an approximate 3.5 cm resection of the anterior middle and inferior temporal gyri allowing access to the temporal horn. This is followed by dissection of the occipito-temporal fasciculus and subsequent removal of 70–80% of the amygdala and all of the hippocampus, parahippocampus, and projection fibers to their posterior extent at the atrium of the lateral ventricle (Spencer, Spencer, Mattson, Williamson, & Novelly, 1984). All patients received postoperative anatomical MRI scans confirming the extent of surgical procedure. A representative scan in coronal and parasagittal sections is provided in Fig. 2. This procedure is highly successful in controlling seizure activity in the patients, with most patients reporting few, if any, seizures postoperatively. These patients typically show some selective cognitive improvement following surgery due to reduction in seizure activity (Novelly et al., 1984), and most are gradually withdrawn from anticonvulsant medication in accordance with postoperative neurological assessment. All but 4 subjects were taking anticonvulsant medication at the time of testing. A subset of 23 patients (11 right, 12 left) participated in Experiment 1.

Neuropsychological testing results, including the Wechsler Adult Intelligence Scale—Revised (WAIS-R) and the Russell adaptation of the Wechsler Memory Scale (WMS; Russell, 1975), are presented in Table 1 along with data on other clinical variables. Neuropsychological data was available for a subset of patients tested both 6 months preoperatively and 12 months postoperatively. Consistent with previous studies (e.g., Novelly et al., 1984), patients with left (speech dominant) medial temporal lobe epilepsy showed some impaired verbal memory in comparison to patients with right (nondominant) medial temporal lobe epilepsy on WMS subtest scores (pre-operative: WMS delayed verbal, $t(17) = 2.80$, $p < .05$; WMS immediate verbal, $t(17) = 2.45$, $p < .05$; postoperative: WMS delayed verbal, $t(12) = 2.82$, $p < .05$; WMS immediate verbal, $t(12) = 2.42$, $p < .05$). Right and left medial temporal lobe epilepsy patients were not significantly different on any of the WAIS-R IQ scores or other subject characteristics.

The control group consisted of 23 nonepileptic adult subjects matched for age and gender without a history of epilepsy or other neurological impairment. Control subjects were recruited

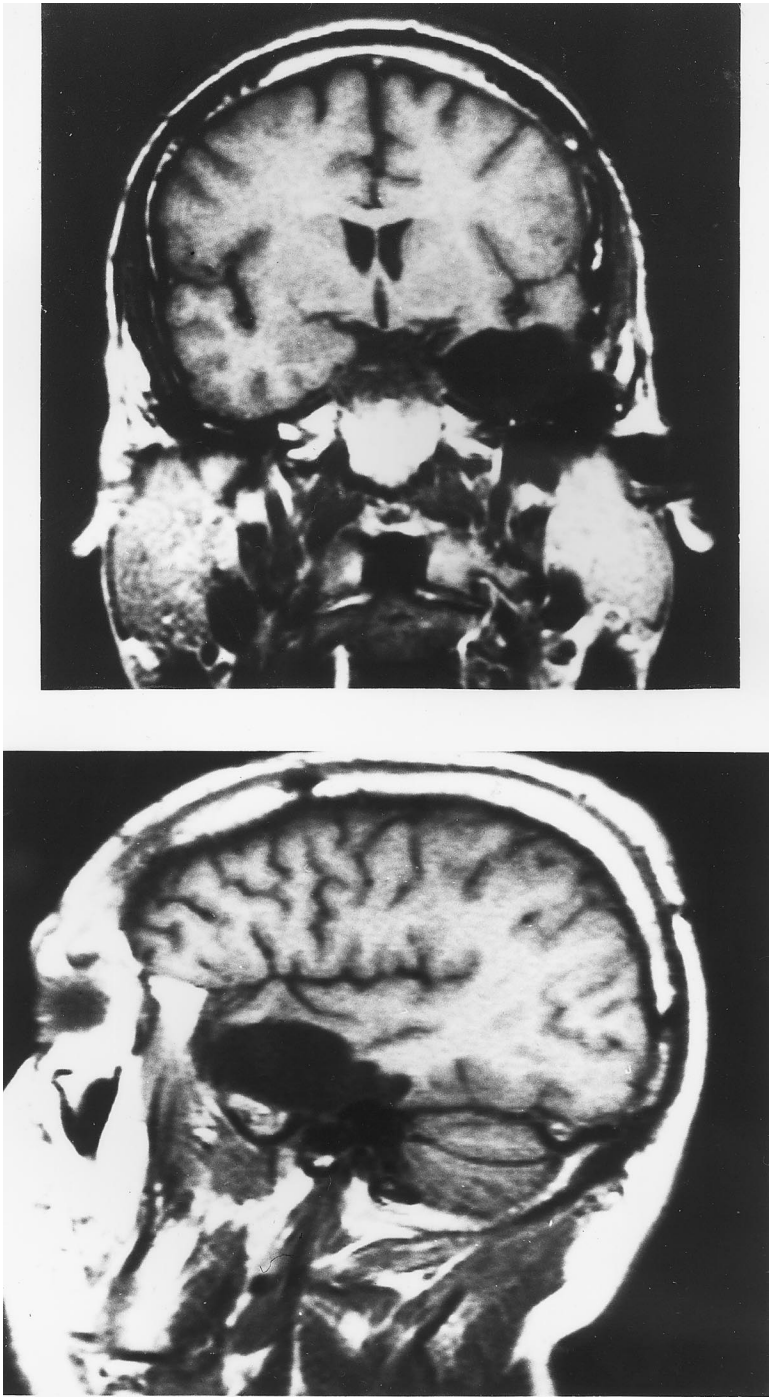

FIG. 2. Representative postoperative T1-weighted MRI scans demonstrating the extent of the surgical procedure: Coronal and parasagittal sections.

TABLE 1
Demographic and Neuropsychological Profile of Patient Population

Group	Age	Gender	Education (yrs)	Seizure Onset (age)	WAIS-R (12 months post-op)	WMS (12 months post-op)
LTL ($N = 13$)	36 ± 10	7M, 6F	13 ± 2	9 ± 10	99 ± 14 F 102 ± 13 P 104 ± 14 V	11 ± 2 IP 8 ± 3 DP 14 ± 5 IV* 7 ± 6 DV*
RTL ($N = 13$)	38 ± 9	3M, 10F	14 ± 2	7 ± 7	107 ± 12 F 110 ± 11 P 104 ± 14 V	12 ± 3 IP 10 ± 5 DP 20 ± 5 IV 16 ± 6 DV

Note. All values represent means (±*SD*) unless otherwise indicated.
* Statistically significant (LTL vs. RTL, $p < .05$).

through a local newspaper advertisement. All subjects provided informed consent and were paid for their participation.

Materials. A list of 27 words, 9 each of three categories—positive (e.g., lucky), negative (e.g., victim) and neutral (e.g., stamp)—was used in this study (see Appendix 1). This list was drawn from a master list of affective words generated by Kitayama (1989). The words were chosen so that all categories were similar in word frequency and length.

SCR was recorded bilaterally with Thought Technology skin conductance units (Lafayette Instruments, Lafayette IN), consisting of Ag-AgCl electrodes attached by velcro straps to the middle phalanges of the subjects third and fourth digits. Lafayette instruments electrode gel was used as electrolyte. The incoming analog signal was amplified and digitized by a Lab Master A/D converter controlled by ASYST software on an IBM AT computer. Skin conductance was sampled at 100 Hz throughout a trial and was stored for off-line amplitude analysis. The minimum SCR resolution was .0144 μSiemen (μS).

Procedure. Subjects were told that they would see a list of words and were asked to rate each word for affect on a scale of 1 (negative) to 5 (positive). Each word was presented on a computer screen for 4 sec during which time SCRs were recorded. Subjects were asked to respond with the affective rating only after the word disappeared from the screen. A new word was presented every 14 ± 2 sec. Words were presented in a pseudorandom order such that no more than 2 words of the same affect were presented in succession. After a delay of 1 min, subjects were given a surprise recall test.

Results

SCR. For each word presentation, the peak SCR amplitude in the 1- to 4-sec interval following stimulus onset was calculated. All SCR data were normalized using a square root transformation. There was no difference in responses for right and left hands for any subject group, so these responses were combined. The results can be seen in Fig. 3.

A two-way ANOVA revealed a main effect for affect, $F(2, 86) = 3.76$, $p < .05$, no effect for subject group and no affect × group interaction. Post-hoc tests did not reveal a significant effect of affect in any individual subject

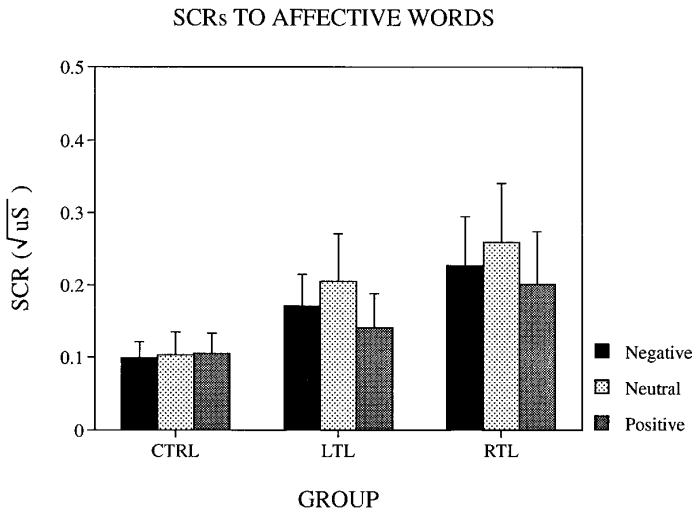

FIG. 3. Mean SCRs to negative, neutral, and positive words for normal controls (CTRL), left temporal lobectomy patients (LTL), and right temporal lobectomy patients (RTL).

group. The main effect is most likely due to the slightly greater responding to the neutral words.

Recall. There was a marginally significant difference in overall number of words recalled between subject groups, $F(2, 43) = 2.85$, $p < .07$, due primarily to the poor recall of left temporal lobectomy subjects (see Fig. 4).

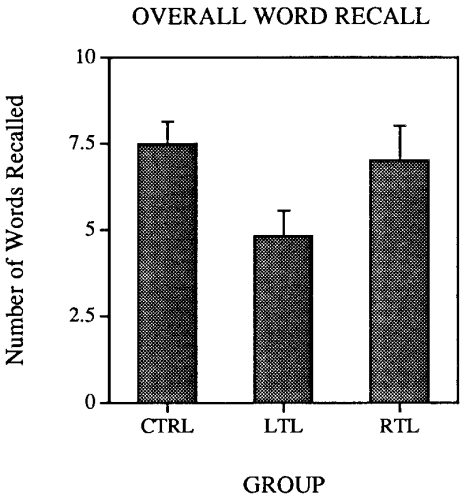

FIG. 4. Mean number of words recalled by normal controls, left temporal lobectomy patients, and right temporal lobectomy patients.

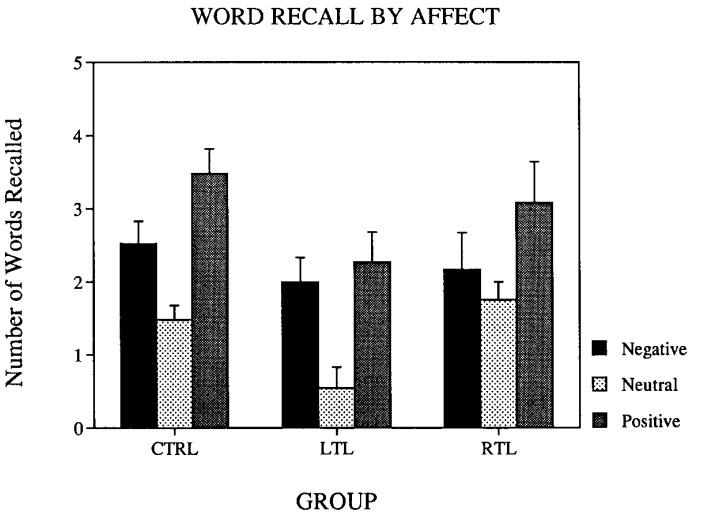

FIG. 5. Mean number of negative, neutral, and positive words recalled for normal controls, left temporal lobectomy patients, and right temporal lobectomy patients.

There was a significant main effect for affect, $F(2, 86) = 22.92$, $p < .001$. Post-hoc Tukey HSD analysis revealed that all groups showed superior recall for positive and negative words in comparison to neutral words ($p < .05$). In addition, positive words were recalled significantly more often than negative words for the right temporal lobectomy subjects and normal controls ($p < .05$). Left temporal lobectomy subjects showed a similar trend (see Fig. 5).

Discussion

The SCR results indicate that while the words are affective, they are not consistently arousing for subjects. This is not particularly surprising given the types of emotional words used. These words are semantically emotional, but not surprising or shocking in any way.

The results on the recall test indicate that, consistent with previous studies and the neuropsychological data for these subjects, the left temporal lobectomy subjects showed slightly worse overall levels of recall. In spite of this mild decrement, the pattern of performance was the same for all subject groups. All of the subjects recalled more of the affective words than the neutral words. Furthermore, all of the subjects showed slightly better recall for positive than negative words. These results indicate that the temporal lobectomy subjects show normal patterns of performance when recalling affective words. This result is in contrast to the emotional memory study on fear conditioning (LaBar et al., 1995) and suggests that patients with unilateral amygdala damage are not impaired on all memory tasks which are influenced by emotional factors.

In the earlier review of the role of the amygdala in emotional memory it was suggested that the amygdala does not seem to be necessary for the experience of emotion in humans, but rather is necessary for assigning emotional valence to a previously neutral stimuli. It may be the case that we did not find a difference in mnemonic performance for emotional stimuli in the temporal lobectomy subjects in Experiment 1 because the stimuli were emotional words, as opposed to neutral words whose memory is influenced by emotional factors. If the amygdala is uniquely involved in learning the emotional significance of neutral events (as is the case in fear conditioning) then the results of Experiment 1 might be expected. In Experiment 2, we attempt to address this issue using a paradigm introduced by Banaji (1986) in which neutral words are embedded in emotional and neutral sentence contexts.

EXPERIMENT 2

Method

Subjects. There were 23 temporal lobectomy subjects (11 left, 12 right) drawn from the same pool of subjects described in Experiment 1. There was an additional control group of 5 epileptic subjects who received surgical excision of other brain areas (4 partial left occipital lobectomy, 1 anterior corpus callosotomy). There were 28 normal control subjects matched for age and gender with the epileptic patients. All subjects gave informed consent and were paid for their participation.

Materials and procedure. Thirty nouns were selected from a list of affective word norms (Belleza, Greenwald, & Banaji, 1986). All words were selected to be neutral in affective valence. These words were randomly assigned to one of three sentence valences: positive, negative, or neutral (see Appendix 2). These assignments were constant across subjects. Each word and its associated sentence valence were typed at the top of a half-sheet of paper. Subjects were instructed to write a hypothetical, self-referent sentence using the word at the top of the page. They were instructed that this sentence should reflect an experience of the assigned emotional valence (e.g., chair, negative—''When I sat in the chair, it broke and I hurt my back''). In this manner, neutral words were encoded in positive, negative, and neutral sentence contexts. The presentation order was pseudorandomized for each subject and subjects were allowed to take as much time as needed. After completing all 30 sentences, the subjects conversed with the experimenter for 1 to 5 min and then were given a surprise recall test for the neutral words. After the recall test, subjects were asked to rate each sentence they wrote on a scale of 1 (negative) to 5 (positive) for emotional valence.

Results

Affective ratings and length of sentences. The sentences written by the subjects were first evaluated for affect based on the subjects' ratings following recall. Subject ratings of the sentence valences revealed a main effect for affect, $F(2, 102) = 215.32$, $p < .001$, and no effect for group or group by affect interaction (see Fig. 6). In other words, all subjects rated the sentences they wrote as consistent with the assigned emotional valence.

In order to assess how elaborate the encoding sentences were for different assigned valences, a word count was conducted for all sentences (see Fig.

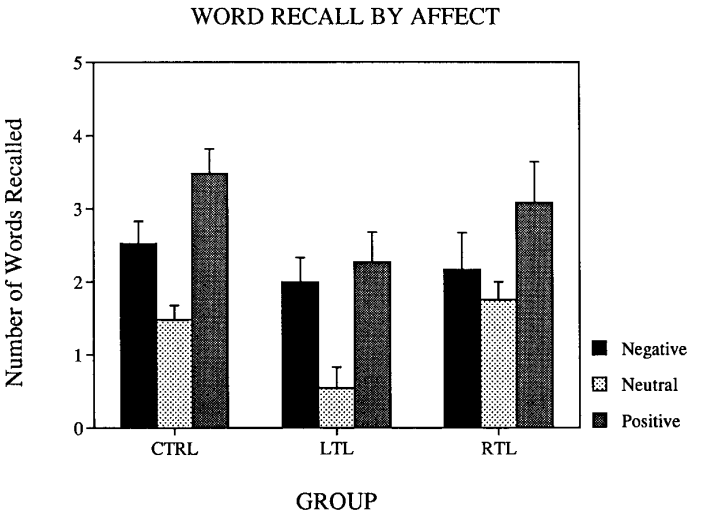

FIG. 6. Mean sentence ratings using a scale from 1 (negative) to 5 (positive) for negative, neutral, and positive assigned valences for normal controls, left temporal lobectomy patients, right temporal lobectomy patients, and epileptic controls with other brain lesions (OL).

7). There was a main effect for affect, $F(2, 96) = 27.60$, $p < .001$, and a significant affect by group interaction, $F(2, 96) = 2.57$, $p < .05$. Post-hoc dependent t-tests adjusted by the Bonferroni correction showed that, in general, both positive ($t(52) = 9.16$, $p < .001$) and negative ($t(52) = 6.76$, $p < .001$) sentences contained more words than neutral sentences. The magnitude of this effect, however, varied by experimental group: post-hoc ANOVAs with adjusted significance levels indicated that the effect was highly significant for the normal controls ($F(2, 54) = 44.10$, $p < .001$); marginally significant in right temporal lobectomy subjects ($F(2, 22) = 5.08$, $p < .06$) and epileptic controls ($F(2, 8) = 6.40$, $p < .09$); and not significant for the left temporal lobectomy subjects. These data indicate that, overall, subjects wrote more elaborate sentences for words encoded in an affective sentence context than words encoded in a neutral sentence context, but this effect was not uniform across subject groups.

Recall. There was a significant difference in overall level of recall between groups, $F(3, 52) = 4.40$, $p < .05$, once again due primarily to the poor recall performance of the left temporal lobectomy subjects (see Figure 8). There was a significant effect for affect, $F(2, 104) = 16.27$, $p < .001$. There was no affect by group interaction (see Fig. 9). All of the groups showed the same pattern of performance, specifically better recall for neutral words embedded in affective sentences than those embedded in neutral sentences. Post-hoc *t* tests adjusted by the Bonferroni correction showed that words encoded in the negative context were recalled more often than words embed-

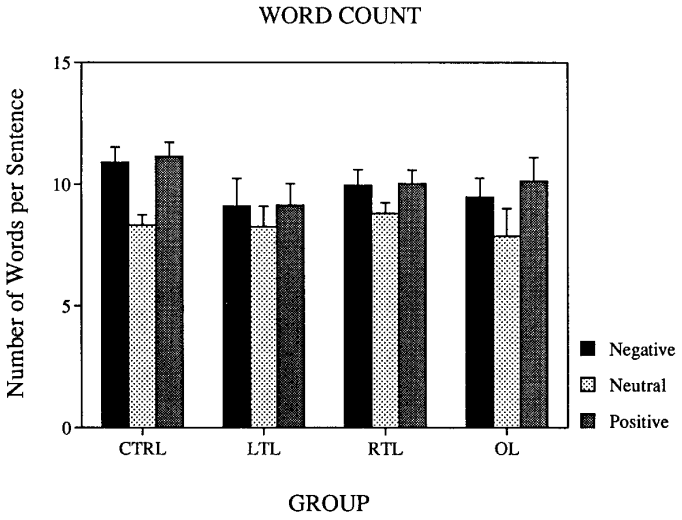

FIG. 7. Mean number of words in negative, neutral, and positive sentences generated by normal controls, left temporal lobectomy patients, right temporal lobectomy patients, and epileptic controls with other brain lesions.

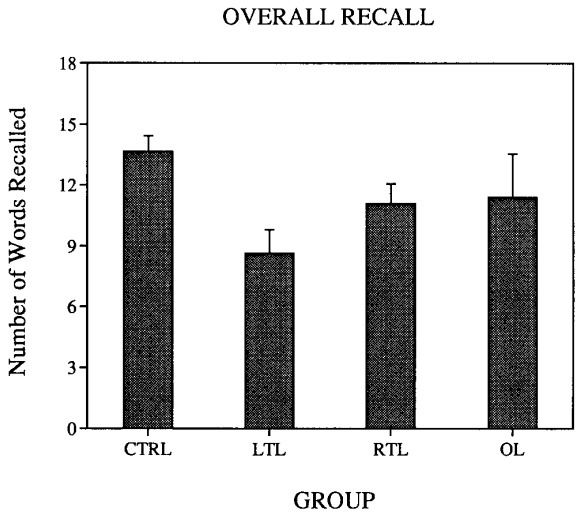

FIG. 8. Mean number of neutral words recalled for normal controls, left temporal lobectomy patients, right temporal lobectomy patients, and epileptic controls with other brain lesions.

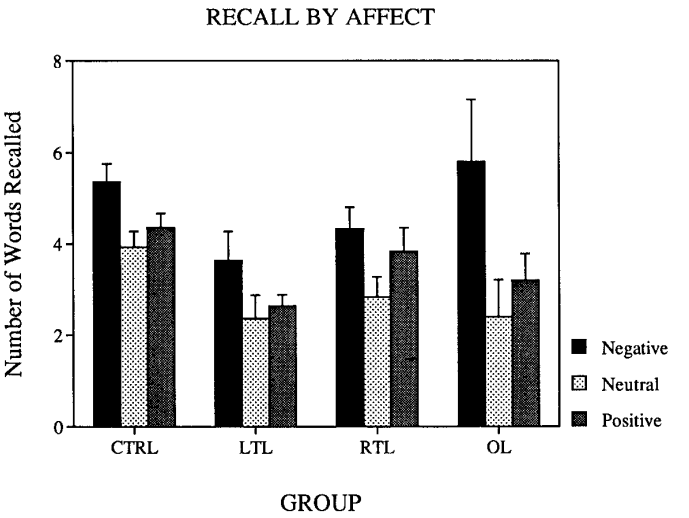

FIG. 9. Mean number of neutral words embedded in negative, neutral, and positive sentences recalled by normal controls, left temporal lobectomy patients, right temporal lobectomy patients, and epileptic controls with other brain lesions.

ded in either the positive ($t(55) = 5.65$, $p < .005$) or neutral ($t(55) = 3.3.4$, $p < .005$) sentence contexts. Words encoded in a positive context were recalled marginally better than words encoded in a neutral context ($t(55) = 2.22$, $p < .10$).

Discussion

The results of Experiment 2 indicate that unilateral temporal lobectomy subjects show intact patterns of recall for neutral words embedded in emotional sentence contexts. Specifically, for all subjects neutral words embedded in negative and positive sentence contexts were recalled more frequently than neutral words embedded in neutral sentence contexts. Although there were some differences in the length of the sentences generated for the different valences by the subject groups, these differences do not appear to be related to the patterns of recall for the neutral words.

These results are consistent with those in Experiment 1 and indicate that unilateral temporal lobectomy patients do not show an impairment on all emotional memory tasks. They seem to show normal enhanced memory for a neutral word when it is embedded in an emotional sentence context. These results are in contrast to the study of fear conditioning (LaBar et al., 1995) in which these patients demonstrated a deficit in acquiring an emotional response to a neutral stimulus. This finding indicates that patients with unilateral temporal lobectomy are unimpaired on some tasks where a mnemonic response for a neutral event is mediated by emotion.

GENERAL DISCUSSION

The results from these two studies indicate that unilateral temporal lobectomy patients demonstrate normal patterns of performance on some tests of emotional memory. The earlier studies on fear conditioning (LaBar et al., 1995) show that these same patients are impaired on other emotional memory tasks. The present studies and the fear conditioning studies differ in many ways and it is not clear which factors may lead to the discrepant results. There are several possibilities as to what the critical differences between these emotional memory tasks might be. Four possibilities, along with our hypothesis concerning them, are discussed below.

Verbal vs. Visual or Auditory Memory

One difference between the present studies and the fear conditioning studies is the type of stimuli. Both of the studies in the present are verbal memory studies, while the CSs in the fear conditioning studies were auditory or visual (Bechara et al., 1995; LaBar et al., 1995). The left temporal lobectomy subjects in the present studies demonstrated a mild verbal memory deficit, however this did not seem to influence the pattern of responding for emotional and nonemotional words.

Although it is impossible to rule out that type of stimuli was a factor, it does not seem likely for a few reasons. First, there were no differences between patterns of memory performance for right and left temporal lobectomy subjects in the present study or the fear conditioning study. One might expect a material specific deficit in emotional memory tasks to mirror that of nonemotional memory tasks. Second, the anatomy and physiology of the amygdala suggests that it is massively interconnected with the neocortex, receiving input from all sensory modalities (Amaral, Price, Pitkanen, & Carmichael, 1992). Given this, it would be surprising if the deficit in emotional memory were specific to the visual or auditory domain.

Unilateral vs. Bilateral Amygdala Damage

It is possible that no deficit was detected in the current studies because the patients only had unilateral damage to the amygdala. The research on the hippocampus and memory might give credence to this interpretation since bilateral lesions lead to a far more severe deficit than unilateral lesions (Milner, 1970). In our earlier study (LaBar et al., 1995) we demonstrated a deficit in fear conditioning following unilateral lesions, but conditioning may be a special case. Daum et al. (1991) found an impairment on an eyeblink discrimination task, thought to be mediated by the hippocampus, in unilateral patients who only show subtle deficits in explicit memory.

As stated earlier, there are very few individuals identified with bilateral amygdala damage without concurrent hippocampal damage and those that have been described have either congenital disorders or behavioral problems

which make it difficult to generalize to normal populations. However, for these few patients, it has not been reported that they have severe deficits that might be related to a global deficit in emotional memory. Although the reports of performance on emotional memory tasks are limited to fear conditioning and memory for an arousing story, it seems likely that there would at least be some anecdotal reports of deficits that would be consistent with a deficit of emotional memory. Furthermore, some of the unilateral temporal lobectomy patients show mild forms of the deficit observed following bilateral hippocampal damage (declarative/explicit memory). This was seen in the left temporal lobe subjects in the present study. If there were a deficit on the types of emotional memory tasks examined in the current studies following bilateral amygdala damage, one might expect to see a hint of an impairment in some of the unilateral patients. This was not the case in the present studies. The pattern of emotional memory performance was consistent across groups. Therefore, we might expect patients with bilateral amygdala lesions to show similar patterns of results.

Learned Emotional Response Deficit

Another possible factor that could explain the discrepancy between present studies and the fear conditioning studies is the nature of the learned response. In the two verbal memory studies, the subjects learned words that were semantically affective or neutral. These words, while meaning something that could be emotional, do not by themselves elicit an emotional response. Memory for the words was influenced by emotional factors, but could not be described as an emotional response. In the fear conditioning studies, the learned response (i.e., arousal) was an indication that the subject experienced an emotion. In other words, the learned response was an emotional response.

It may be that the amygdala is only involved in emotional memory tasks when the learned response is an emotional response. If this were the case, one would not expect to see a deficit in the types of tasks used in the present studies. The memory deficit seen following amygdala damage would be limited to tasks where the subject learns to fear (or, perhaps, favor) previously neutral stimuli. This emotional response may not necessarily be related to any measurable arousal (for instance, affection) or it may be limited to emotional responses that also induce arousal. The research on emotional memory following amygdala damage has primarily focused on fear or arousal, probably because these are easier to measure (especially in non-human animals) than other emotional responses.

Arousal Memory Deficit

Almost all of the behaviors reported to be associated with amygdala function in the earlier review of the literature could be described as related to arousal or, perhaps, fear. It is hard to dissociate these two since they often

co-occur and we assume the behavior we measure (usually arousal) is related to an internal state (fear). For the studies with humans, at least, we prefer the term arousal since the response measured in the two fear conditioning studies (SCR) is a measure of arousal and the subjects in the LaBar et al. (1995) study did not report being afraid of the US, although they certainly were aroused by it and somewhat annoyed.

It could be the case that the amygdala is only involved in emotional memory tasks when the emotional component leads to arousal. This interpretation is consistent with the proposal by Damasio (1994) that the amygdala is necessary for coupling exteroreceptive sensory information with information concerning somatic states. In the two verbal memory studies, the emotional stimuli (words or sentences) were emotional in meaning, but did not seem to lead to an emotional response in the subjects. In Experiment 1, arousal to the stimuli was measured using SCR and no consistent response was seen for the emotional words. In Experiment 2, the subjects, not surprisingly, did not appear to actually experience the emotions consistent with the hypothetical events described in the sentences. It is possible that in tasks like these, where the emotional component is more semantic than arousing, the benefit to memory is one of organizational strategies, that is, emotion benefits encoding or serves as a retrieval cue much in the same way an overarching category might.

If the amygdala is uniquely involved in tasks where arousal influences memory performance then the results of the present studies would be expected. Arousal did not appear to be a factor in these studies, unlike the studies on fear conditioning or the study described earlier on memory performance following β-adrenergic receptor blockers (Cahill et al., 1992). It is possible that unilateral temporal lobectomy patients would only show deficits on tasks where arousal influences memory performance and the role of the amygdala in human memory is limited to these types of emotional memory tasks.

Conclusions

All of these possible explanations for the discrepancy between the present results and the impairment on the fear conditioning studies merit further examination. The first two possibilities, that is, the deficit is only apparent for non-verbal stimuli or following bilateral amygdala damage, do not seem likely for the reasons cited above. The last two possibilities, that is, the deficit is specific to learned emotional responses or arousal, seem reasonable and may both be true. If so, one would expect to see a memory deficit following amygdala damage in very limited circumstances. Patients with amygdala lesions may show a deficit when learning to respond in an emotional manner to a neutral stimulus and/or when learning is mediated by arousal. This limited deficit would not significantly impact the patients' behavior in everyday life and may go unnoticed. This is consistent with the reports of patients

following unilateral temporal lobectomy as being relatively unimpaired (with the exception of those that show a mild explicit memory deficit). Although there is much more to be discovered about the deficit in emotional memory following unilateral temporal lobectomy, the finding of intact performance in the present studies indicates that these patients do not show a global deficit on all emotional memory tasks.

APPENDIX 1

Words from Experiment 1 by Emotional Valence

Positive

Lucky
Funny
Trust
Talent
Proud
Joke
Comedy
Smile
Glory

Neutral

Stamp
Spare
Switch
Locate
Habit
Chair
Stone
Border
Track

Negative

Victim
Error
False
Damage
Fault
Waste
Fool
Cancer
Devil

APPENDIX 2

Neutral Words from Experiment 2 by Assigned Emotional Valence

Positive

Pamphlet
Cork
Ink
Stool
Dish
Riddle
Bowl
Kerosene
Merchant
Hat

Neutral

Trumpet
Statue
Ginger
Key
Foot
Pajamas
Thermometer
Clay
Racket
Bag

Negative

Trunk
Door
Office
Wagon
Scissors
Museum
Monkey
Boat
Bundle
Engine

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