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Cohesion Failure as a Source of Memory Illusions

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One source of ''false'' memories may be that often only memory fragments are retained. This would then result in a person being unable to distinquish a false conjunction, constructed of memory components, from what had been actually experienced. Experiment 1, employing two-syllable words in a continuous recognition paradigm, found that patients with left hippocampal damage classified more new verbal conjunctions as ''old'' than did normal subjects or patients with only right hippocampal damage. Experiment 2, employing simple face drawings in a study-test paradigm, found that patients with damage to either side of their hippocampal formation made more conjunction errors with pictorial stimuli than did normal subjects. The results are seen as supporting the hypothesis that binding is an important early step in the consolidation process and that the hippocampal system is a critical component of the neural system involved in the appropriate binding of memory components.

Memory ''illusions''—the remembering of events that did not occur—as well as other memory ''distortions,'' are well known to students of memory (Roediger, 1996; Schacter, 1995; Schacter & Curran, 1995) and demonstrations to prove their existence are no longer needed. The central problem today is to account for them in terms that make them a natural expression of the workings of human memory. The explanation of memory illusions should not require the postulation of any special memory structures, processes, or mechanisms, but rather should be readily derivable from our current theories of the cognitive and neural basis of memory processing.

In this paper we report data in support of the hypothesis that one source of memory illusions is the defective process of ''binding'' or ''cohesion.'' ''Cohesion'' is a term used by Moscovitch (1994) to refer to a rapid form of consolidation that plays an important role in the process of transforming the incoming information into long-term storage. A slower form, consolidation proper, contributes to the solidity of the coherent trace.

Our ''cohesion hypothesis,'' if true, would contribute to the understanding of what appears to be a rather puzzling phenomenon—how can anyone ''remember'' nonexistent events? If it is possible to capture evidence for the hypothesis, albeit under laboratory conditions, we might have the beginnings of a more general model of memory illusions. Perhaps even more importantly, the pursuit of the cohesion hypothesis may contribute to our knowledge of the workings of memory as a

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whole. Just as studying perceptual illusions helps us to better understand how the perceptual system interprets complex and sometimes ambiguous sensory inputs in such a way as to construct—most of the time—useful representations of the world around us, studying memory illusions should help us to improve our understanding of a system that gives rise to veridical remembering—most of the time.

We describe two experiments done with normal subjects and patients with mesial temporal-lobe lesions. The results of these experiments suggest that components of perceived items can be mismatched in the course of post-perceptual processing, with the consequence that some of the items that are stored do not correspond to items presented as such at study. On the basis of existing research, discussed below, we expected that this kind of a memory illusion would occur much more frequently in patients with damage in the hippocampal system than it would in normals, and that this, in turn, would corroborate the growing conviction that these brain regions play a critical role in consolidation.

Memory consolidation is a venerable idea that originated in Müller and Pilzecker's (1900) work on retroactive inhibition. They explained their findings by assuming that the brain activity associated with learning continues (''perseveres'') in time beyond the learning episode and can be inhibited by subsequent learning. However, the idea of consolidation has not been popular among ''verbal learners'' and cognitive psychologists. For example, in the subject indexes of all the 23 volumes of *Journal of Verbal Learning and Verbal Behavior* that were ever published, from 1962 to 1984, ''consolidation'' appears exactly once. The main reason for such disinterest probably lies in the absence of any data originating from the laboratory study of human learning and memory whose interpretation requires the use of the concept. The outcomes of the Müller and Pilzecker type of interference experiments can be, and have been, explained in terms of concepts such as response competition (McGeoch, 1932), transfer (Webb, 1917), ''disruption'' (Britt, 1936), extinction and ''factor X'' (Melton & Irwin, 1940), unlearning (Barnes & Underwood, 1959), and response-set suppression (Postman, Stark, & Fraser, 1968). With such a wealth of psychological explanations, there seemed to be no need for physiologically tinged ideas. In the physiologically oriented study of memory, on the other hand, the concept of consolidation has been accepted not only as useful but even indispensable (Alvarez & Squire, 1994; Curran, 1986; Glickman, 1961; Izquierdo, 1989; John, 1967; Lynch, 1986; McGaugh, 1966; Parker, Morihasa, Wyatt, Schwartz, Weingartner, & Stillman, 1980; Warburton, 1992; Weingartner & Parker, 1984; but see also Crowder, 1982).

Traditionally, ''consolidation'' has referred to the processes that form the bridge between primary memory and secondary memory (Scoville & Milner, 1957) and that determine the ''whether'' and ''what'' of the storage of the information. The basic paradigm is one in which the learner is given a learning trial, the experimenter administers a specific post-trial treatment, and, as a consequence, the learner either does not retain what it learned as effectively as would have occurred in the absence of the treatment, or retains it more effectively. In the former case, the retrograde effect is interpreted in terms of interference with consolidation, in the latter case, as facilitation of consolidation. The treatments that have been used are typically ''systemic,'' such as electroconvulsive shock, or the administration of drugs. The duration of the period of consolidation varies with the specifics of the situation, but is always finite.

Like many other concepts that have been around for a long time, consolidation too is very broad, and calls out for refinement. Moscovitch's (1994) distinction between cohesion and consolidation proper is a step in the right direction. We find it useful, adopt it for our present purposes, and suggest one method, borrowed from Underwood and Zimmerman (1973), as a means of empirically tapping the cohesion process.

Along with Moscovitch (1994) we think of cohesion as an early component process of consolidation whose function is to ‘‘bind’’ or ‘‘glue’’ aspects of incoming information into separately retrievable engrams (Chalfonte & Johnson, in press; Johnson & Chalfonte, 1994; Metcalfe, Cottrell, & Mencl, 1992; Wickelgren, 1979). It has been suggested that the hippocampal formation plays an important role in such binding (Cohen & Eichenbaum, 1993, pp. 286–288; Eichenbaum & Bunsey, 1995). This binding process has been represented as an additional constituent of encoding as it is conceptualized in the General Abstract Processing System (Tulving, 1983). The memory binding is similar to perceptual binding postulated by Treisman and others (Treisman & Gelade, 1980; Treisman & Schmidt, 1982), the main difference being that it occurs after the act of perception, and that its product is a coherent engram of the perceived event.

Cohesion has the following hypothetical properties: (a) it begins when a stimulus object appears and is perceived, (b) it runs its course over a short interval after the perceptual event, (c) it is not under the conscious deliberate control of the learner, (d) it assembles (or, ‘‘binds’’) the engram in secondary memory from the neuronal/informational elements available in primary or working memory, (e) it ends when the engram has been constituted (‘‘bound’’), and (f) it is independent of primary memory. (But see Baddeley, 1994, whose working memory model includes binding as an additional operation performed by the ‘‘central executive.’’)

Although one function of primary memory *may* be to increase the likelihood and efficiency of cohesion, the correct perception of an event and its initial registration in primary memory will not necessarily guarantee its consolidation into secondary memory. Indeed some people (e.g., Rawlins, 1985) have argued that the hippocampal system’s main function is to act as a temporary memory store or buffer, but a growing body of data suggests that this is not the case (cf. Eichenbaum, Otto, & Cohen, 1994). Patients with damage to their hippocampal system behave relatively normally in many tasks designed to measure short-term memory, but tend to be extremely deficient in tasks measuring explicit longer term memory.

If, as appears to be the case, cohesion is not guaranteed by anything like registration of information into the primary memory system, or its ‘‘recycling’’ or ‘‘rehearsal,’’ it is difficult to draw inferences about cohesion simply by measuring short-term memory. Nor can the results of interference experiments be very informative, because, as we have seen, they can be too easily explained in other ways.

A more promising method for the purpose is one introduced by Underwood and Zimmerman (1973) and recently adopted by Reinitz (Reinitz & Demb, 1994; Reinitz, Lammers, & Cochran, 1992; Reinitz, Verfaellie, & Millberg, 1996). This method allows one to observe memory illusions by presenting complex stimuli and then measure the extent to which subjects false alarm when the components of the stimuli are recombined into new units during a subsequent test. To the extent that subjects do so, they can be said to ‘‘remember events that did not occur.’’ Following Reinitz (e.g., Reinitz et al., 1996), we will distinguish ‘‘memory conjunction errors,’’ which appear to be the result of memory processes, from ‘‘illusory conjunctions’’ which Treisman (e.g., Treisman & Schmidt, 1982) believed are due to perceptual processes (but see Navon & Ehrlich, 1995).

Underwood and Zimmerman had their subjects study two-syllable words and obtained memory conjunction errors which ‘‘while highly reliable statistically, were not large in an absolute sense’’ (p. 705). Reinitz et al. (1992) found evidence of memory conjunction errors following the study of complex line drawings of faces, and Reinitz (Reinitz & Demb, 1994; Reinitz et al., 1996), found evidence of memory conjunction errors following the study of compound words. All but the last of these studies measured the effect with college students. We reasoned that if the hippocampal system was responsible for the binding of the individual components of a stimulus into an integrated memory trace, then patients with damage to their hippocampal system should be much more likely to experience memory conjunction errors than are either students or older adults without such damage. In particular, given the specialization of the left hemisphere for language processing (e.g., Gazzaniga, 1995; Gazzaniga & Sperry, 1967; Hellige, 1993; Moscovitch, 1979), we expected that, when studying verbal material, this difference between people with intact and impaired hippocampal systems would be greater when the left hippocampal system is damaged. On the other hand, although the standard view is that the right hemisphere is predominant in the processing of pictorial–spatial stimuli, results from Palmer and Tzeng (1990) indicate that *both* hemispheres may be required for the complete processing, memory, and later discrimination of complex visual stimuli. Consequently, we expected memory conjunction errors with pictorial stimuli to be either greatest for patients with right hippocampal damage or to be large in all of our patient subjects, regardless of the laterality of their hippocampal damage.

In our first experiment, we presented the subjects with lists of two syllable words and tested their brief memory through the use of a continuous recognition paradigm. Our hypothesis was that patients with left hippocampal damage would be more likely than normals or patients with right hippocampal damage to classify as ‘‘old’’ new words that were constructed out of syllables from recently presented words (e.g., FICTION . . . BUCKLE . . . FICKLE). Notice that while this test is similar to the verbal test developed by Reinitz (e.g., Reinitz et al., 1996), it differs in several important ways. First, Reinitz had his subjects learn a list of words and then tested for recognition after a brief retention interval. Our test, on the other hand, is using a continuous recognition test which, among other things, allows for the testing over much shorter retention intervals. Second, Reinitz used compound words (e.g., SHOTGUN) so that each component is itself a word. In our test, the words consisted of two nonword syllables (e.g., MUSTANG).

SUBJECTS

Patients

We tested seven patients with lesions to their left hippocampal system[1] (LHc), four resulting from strokes and three from lobectomy, eight with lesions to their right hippocampal system (RHc), five resulting from strokes and three from temporal lobectomy, and one patient with a bilateral hippocampal lesion (BHc) resulting from anoxia. All strokes were due to infarction of the posterior cerebral artery from embolus or atherosclerotic occlusion except for one patient (DR) whose stroke was due to vasospasm after a subarachnoid hemorrhage. All of the patients suffered from variable degrees of anterograde amnesia. The BHc patient presented the most severe anterograde amnesia and the LHc patients tended to show more severe anterograde amnesia symptoms than the RHc patients. Aside from these memory problems, all patients were capable of understanding complicated instructions and of carrying on intelligent conversations with the examiner. However, the patients with the more severe symptoms are unlikely to remember these conversations a short time later. The effects from unilateral posterior cerebral infarction and the resulting persistent acute anterograde amnesia have been reviewed elsewhere (von Cramon, Hebel, & Schuri, 1988; DeRenzi, Zambolin, & Crisi, 1987; Ott & Saver, 1993).

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[1] By using the term ‘‘hippocampal system’’ we mean to refer to the same area designated by Eichenbaum *et al.* (1994, *pp.* 450–451). The patient with a bilateral lesion resulting from anoxia would not be expected to have any parahippocampal damage and based on postmortem data from other patients should have predominantly CA1 damage. There may, however, be cellular damage in other regions of the brain in addition to the CA1 region in the posthypoxic patients that eludes quantification. The hippocampal stroke patients all have parahippocampal in addition to hippocampal damage. The lobectomy patients have minimal, if any posterior parahippocampal damage although they do have anterior parahippocampal damage.

The patients with infarcts all had variable degrees of homonoymous field defects due to calcarine damage. Two of the LHc patients also suffered some damage to their splenium resulting in some degree of alexia without agraphia. Patient (AL) could read only a few letters at a time and the other (EM) only a syllable at a time. These patients were asked to look at the screen while the words were read to them. Previous testing sessions had demonstrated that they were capable of reporting when the experimenter said a word different than the one shown on the screen. Their results mirrored that of the other patients and thus they were included in the overall analysis.

Unfortunately, standard test scores (e.g., complete WMS-R) were not available for most of the patients used in this study. In order to provide some indication of the severity of their anterograde amnesia, Table 1 presents the standardized scores for the patients on those subtests of the WMS-R that were obtained most of the patients. (These standardized scores were found by using the mean and standard deviations for each subject's age group as given in the WMS-R manual.) The immediate and delayed versions of the Prose (or ''Logical'') and Visual Reproduction subtest scores were obtained from all of the patients, and the immediate and delayed versions of the Verbal Paired Associate subtests were obtained from the infarction and anoxia patients. Of these sets of subtests, the delayed Verbal Paired Associate test seems to best capture the severity of their anterograde amnesia. Note that on this test, the bilateral hippocampal patient scores over four standard deviations below normal (−4.83), the average of the patients with left hippocampal damage is more than three standard deviations below normal (−3.39), and the average for the patients with right hippocampal damage is over one standard deviation below normal (−1.36). Table 1 also gives the sexual and age compositions of patient and normal groups.

Computerized reconstructions of computed tomography (CT) or magnetic resonance (MR) brain scans of the patients are presented in Figures 1a and 1b.

Healthy Control Subjects

Two groups of healthy controls were tested. One was a group of 18 older adults who lived in Davis, California and who were contacted via a newspaper advertisement. These subjects, like the patients, were paid $10/hour for their participation. The other consisted of 18 students from introductory psychology courses at the University of California, Davis, or at Dartmouth College. These subjects received minor course credit for their participation.

EXPERIMENT 1

Method

Design. Three lists of common two-syllable nouns were constructed such that each word presented fell into one of the following four categories: (a) *First:* this is the first time this word or either of its syllables appeared in the list; (b) *Syllable-Repeat:* this is the second time that *one* of the syllables appeared in the list; (c) *True-Repetition:* this is the second time that this exact word appeared in the list; or (d) *Conjunction:* this is the second time for *each* of the syllables, but the first time that they appeared together. (See Table 2: Word Categories.)

A *Conjunction* set consisted of the two initial words containing the key syllables and the test word. The ''lag'' of a *Conjunction* set was the number of words between the two initial words. In this experiment, the lag wa either one (i.e., one word intervened between the first and second initial words) or five. The ''retention interval'' of a *Conjunction* set was the number of words between the second of the initial words and the test word. This is also the definition of the retention interval for *True-Repetition* and *Syllable-Repeat* sets. The retention intervals in this experiment varied between five and forty words. There were a minimum of six *Conjunction* sets of each lag/retention interval combination and an equal number of *True-Repetition*

TABLE 1

SUBJECT SUMMARY

Patient Groups

		Age at			Wechsler memory scale					
					Immediate			Delayed		
Subject	Sex	Test	Lesion	Dominant hand	Prose	Visual repro	Verbal PAL	Prose	Visual repro	Verbal PAL
Left hippocampus (infarction)										
AL	m	62	58	Right	−1.98	1.54	−2.92	−2.18	1.61	−4.92
WM	m	71	56	Right	−1.08	0.52	−3.45	−1.05	−1.99	−2.64
JS	m	72	55	Right	−0.67	−0.44	−2.45	−0.73	−0.18	−3.36
EM	m	78	76	Left	−1.22	0.15	−1.70	−1.27	−1.02	−2.64
Left hippocampus (lobectomy)										
PM	f	33	30	Right	−0.38	1.60		−0.53	1.62	
BG	m	29	27	Right	−0.22	0.27		−0.20	0.07	
JA	m	32	31	Right	−2.00	1.04		−1.73	1.06	
Right hippocampus (infarction)										
JC	f	55	perinatal	Right	−0.87	0.38	−1.88	−1.52	0.64	−1.58
FN	m	56	50	Right	−0.24	1.73	0.63	−0.35	0.50	0.92
CB	m	72	49	Right	−1.22	3.16	−1.70	−0.95	0.06	−2.64
DO	m	70	?	Right	−0.67	1.52	−1.45	−1.05	−1.02	−1.21
DR	m	43	41	Left	−1.00	−0.85	−1.53	−1.51	−0.49	−2.29
Right hippocampus (lobectomy)										
JE	f	32	30	Right	−0.44	−0.04		−2.13	−0.46	
SA	f	40	38	Right	1.64	1.14		0.77	1.36	
Tk	m	25	22	Right	−0.45	1.31		−1.70	−1.74	
Bilateral hippocampus (anoxia)										
RD	m	68	63?	Right	−1.76	−3.17	−3.18	−1.95	−2.39	−4.83

Nonpatient Groups

	Female/Male	Age
Adults	11/7	56.7 (40–70)
Students	7/11	20.3 (18–25)

and *Syllable-Repeat* sets tested at each retention interval. In addition, the number of *Conjunction* sets in which the first syllable of the test word was presented before the second was equal to the number of sets in which the second syllable was presented before the first for each lag and retention interval. Across the *Syllable-Repeat* sets, the first and second syllables were equally often chosen to be the syllable repeated.

List 1 and list 2 were presented in session 1 and did not repeat words or syllables unless the design required it. List 3 was pre-

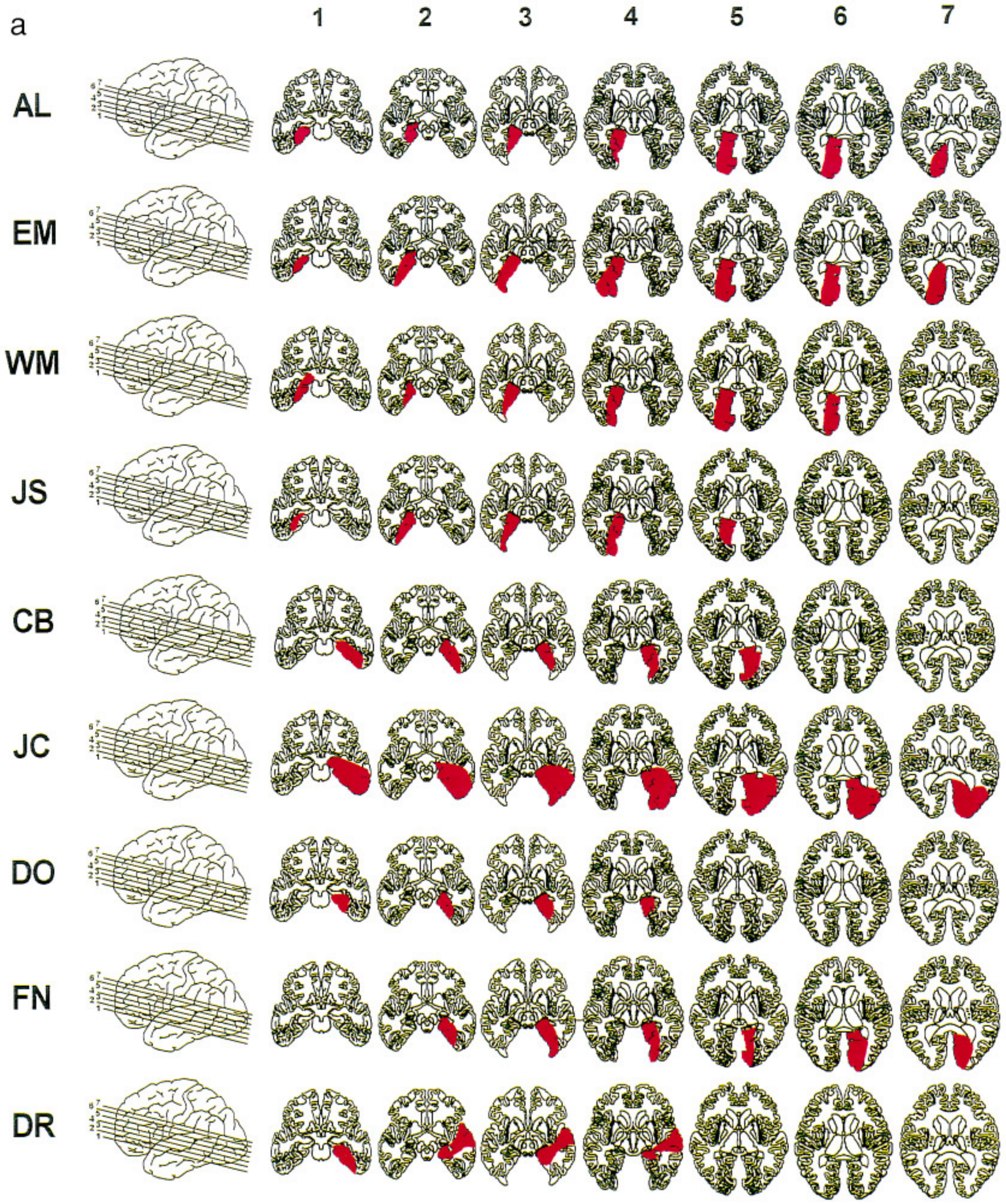

FIG. 1. Computerized reconstructions of CT or MR brain scans for nine patients with hippocampal lesions resulting from infarctions of the posterior cerebral artery. Red areas represent site of lesion on transverse sections. The lateral view illustrates the level and orientation of each section from the most ventral section (1) to the most dorsal section (7) (b). Computerized reconstructions of CT or MR brain scans for six patients with hippocampal lesions resulting from temporal lobectomies. The lateral views show the amount of anterior temporal resection employed to get at the mesial temporal structures including amygdala and hippocampus. Thus, the resections of these mesial structures is not seen on the lateral view of the brain. This is why a coronal cut through the amygdala is given for the resection patients. The axial cuts do show the mesial temporal damage in both the resection group and the stroke group. A coronal cut is not provided for the hippocampal stroke group since the amygdala is not damaged in this group. In addition a lateral view for the stroke group is not provided since there is no damage to the anterior temporal lobe in this group.

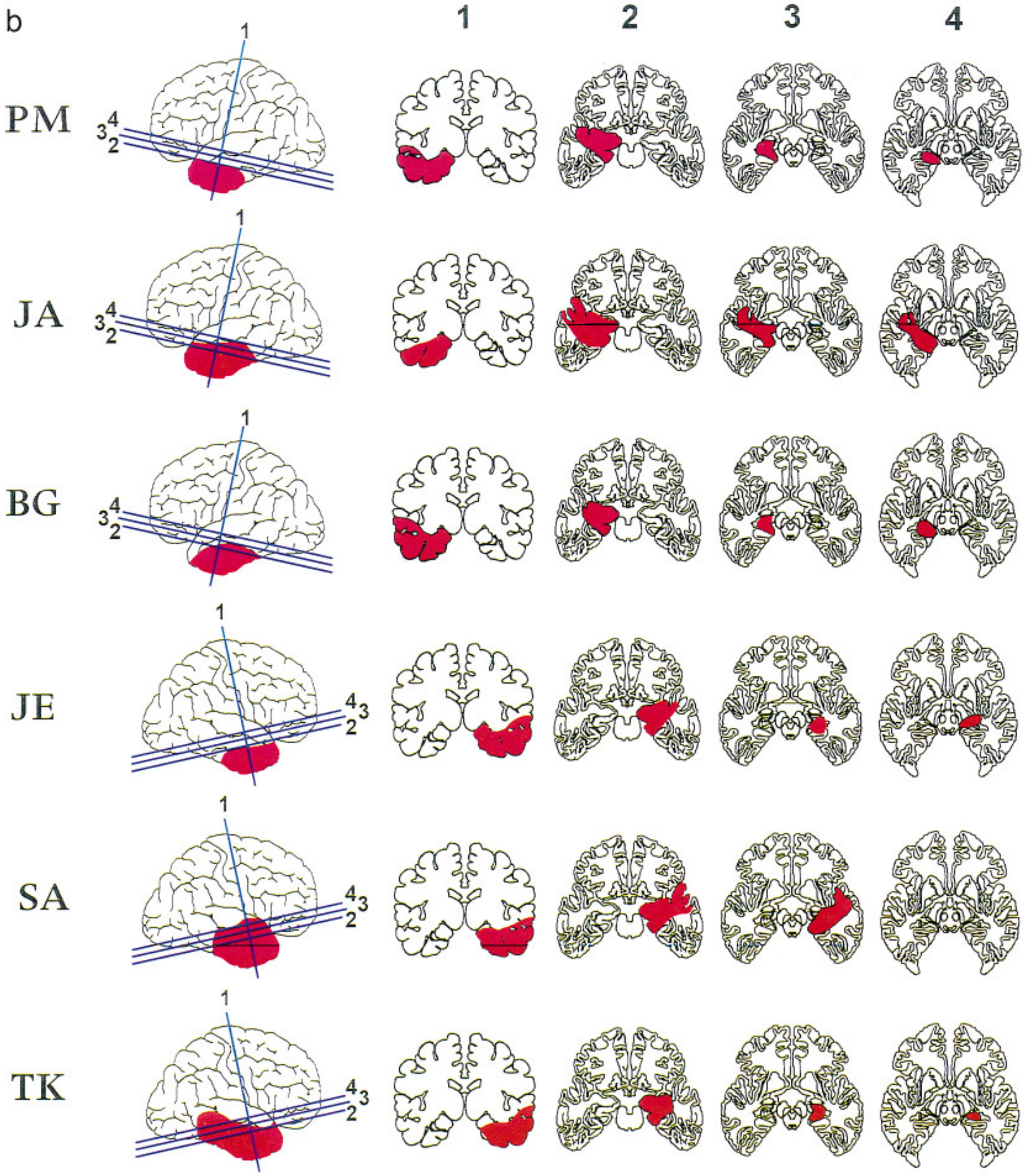

FIG. 1—*Continued*

sented in session 2, which took place at least two weeks later. In order to best compare different groups of subjects, all subjects received the exact same lists (i.e., the words were not counterbalanced across conditions), but list 3 was composed of many of the same words used in lists 1 and 2 with the words assigned to different categories. The lengths of the lists, including some filler words, were 99, 109, and 182 words respectively, with list 3 essentially replicating the combined conditions of lists 1 and 2.

Procedure. A continuous recognition paradigm was employed. The words were pre-

TABLE 2

WORD CATEGORIES: PRECEDING WORDS

Category			Lag interval		Retention interval	Test word
Repetition	. . .	. . .	. . .	**SIGNAL**	. . .	**SIGNAL**
Syllable-Repeat	. . .	. . .	. . .	FER**TILE**	. . .	REP**TILE**
Conjunction	. . .	VAL**LEY**	. . .	**BAR**TER	. . .	**BARLEY**

sented sequentially in the center of a computer monitor. The subject read aloud the word on the screen and judged whether the word was ‘‘old’’ (i.e., had occurred previously in the list) or ‘‘new’’ (i.e., is occurring for the first time in the list). Note that in this experiment, saying ‘‘old’’ to the second member of a *True-Repetition* pair is a ‘‘hit’’ and saying ‘‘old’’ to any other word is a ‘‘false alarm.’’ The percentage of ‘‘old’’ responses to *First*-words is the base false alarm rate.

Non-patient subjects indicated their judgment by pressing one of two keys on the computer keyboard. Patient subjects told their judgments to the experimenter, who pressed the appropriate key. The average number of seconds per word required to report a decision was 1.74, 1.65, and 1.47 for the patients, normal adults, and students, respectively. After completing the first list, subjects were given several visual memory tests (see Experiment 2), and then reminded of the instructions before receiving list 2. List 3 was given in session 2, which occurred at least two weeks later.[2]

Results

The overall pattern of results did not change with the different retention intervals. Consequently, to simplify an already complicated set of results, the data were collapsed over retention intervals. Table 3 presents the average scores for each of the groups in each of the conditions.

First, normal subjects showed a very high rate of saying ‘‘old’’ to true repetitions, a much lower rate of saying ‘‘old’’ to conjunctions, an even lower rate of saying ‘‘old’’ to syllable repeats, and the lowest rate of saying ‘‘old’’ to totally new words. This is basically the same pattern found by Reinitz and Demb (Experiment 1, 1994), in spite of the many differences in the experimental paradigms and materials.

The patients did *not* have systematically lower ‘‘base’’ percentage correct scores (i.e., percentage of hits minus the percentage of false alarms to *First* words that are unrelated in any obvious way to words occurring previously in the list). That is, the student and adult controls averaged 83.8% and 69.4% correct, and the RHc, LHc, and BHc patients averaged 80.9%, 77.2%, and 84.0%, respectively. This is, in itself, notable. That is, these patients all exhibit some degree of anterograde amnesia, especially the LHc and BHc patients; yet, at least under these conditions and time parameters, their ability to recognize the true repetitions and to reject completely new words is very similar to that of the subjects without hippocampal damage. The main difference among the groups was seen in the false alarm rate to the conjunction words. The most interesting aspect of this pattern is that the LHc subjects (including the BHc patient) have very large false alarm rates to the conjunction words whereas the other subjects, the controls and the RHc subjects, do not.

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[2] The lobectomy patients, by reasons outside of the control of the authors, could be tested for only one session and thus were only tested on two of the lists. All three of the lists were used for the remaining subjects, however, in order to improve the stability of their scores and in order to test words in different conditions. No systematic differences were observed across lists.

TABLE 3

AVERAGE PERCENTAGE OF 'OLD' RESPONSES FOR EACH OF THE GROUPS IN EACH OF THE CONDITIONS IN EXPERIMENT 1

Group	No.	True *repetition* (hit)	*Conjunction* lag		Single *syllable* repetition	*First* word (new)
			One word	Five words		
Students	18	88.4 (1.71)	9.7 (1.92)	10.3 (1.66)	4.0 (0.90)	4.6 (0.59)
Older adults	18	78.9 (3.09)	14.5 (2.97)	14.3 (3.09)	7.7 (1.81)	9.5 (2.90)
RHc	8	83.9 (2.41)	12.0 (3.20)	12.2 (3.60)	8.3 (3.82)	2.9 (1.62)
LHc	7	84.7 (3.81)	41.3 (7.61)	30.6 (7.05)	16.3 (4.12)	7.6 (2.76)
BHc	1	88.9	52.8	19.2	24.0	4.9

Note. SE in parentheses.

The difference in false alarm rates to the syllable-repetition words was not significant between the RHc patients and the LHc (including BHc) patients, $t_{14} = 1.67$, $SE_M = 5.32$. However, in order to test the degree to which conjunction stimuli resulted in a greater false alarm rate than that obtained from syllable-repetition stimuli, a 2 × 2 (patient group × lag) analysis of variance was performed on the difference scores found by subtracting each subject's false alarm rate to the syllable-repetition words from that subject's false alarm rates to the conjunction words with one word lags and from that subject's false alarm rates to the conjunction words with five word lags. This analysis found significantly greater difference scores (i.e., more false alarms to conjunction words) for the LHc subjects: 18.7 vs 3.7, $F(1,14) = 7.22$, $MS_E = 716.28$; more false alarms for the conjunction words following short lags: 14.6 vs 7.9, $F(1,14) = 11.72$, $MS_E = 2{,}773.64$; and a significant interaction: $F(1,14) = 6.13$, $MS_E = 1{,}452.17$, i.e., the LHc subjects had a greater false alarm rate to the conjunction words following short lags (25.5) than to those with long lags (11.9), while the RHc subjects did not (3.6 and 3.8).

DISCUSSION

In all cases, performance of the RHc patients appeared very similar to that of the subjects in the two groups without lesions. Thus, it appears that high false alarm rates to the conjunction words on this task are specifically related to left hippocampal malfunction. It also may require that the two syllables occur together within a short period of time in order for the conjunctions to produce a higher false alarm rate than a repetition of a single syllable.

It should be emphasized that what the LHc patients are doing when they show a high rate of responding ''old'' to the conjunction words, but not doing so to first words or even to syllable-repeat words, is not simply showing a ''weak'' memory. For one thing, neither normals nor RHc patients develop this pattern over longer retention intervals. For another thing, subjects instructed to respond ''old'' to both repeats and conjunctions, but not to syllable-repeats, had a very difficult time doing so. Of 45 University of California, Davis and Dartmouth students tested with these new instructions, only 13 were able to keep their base false alarm rate below 10%, and these subjects, deliberately trying to respond ''old'' to the conjunction words, only managed an average of 27.7 at the short lag and 25.0 at the long lag. The remaining 32 subjects obtained scores of 61.5 and 57.3 for the conjunction words at the short and long lags, but at the cost of an average base false alarm rate of 25.8 and a false alarm rate to single syllable repetitions of 39.2. Thus, normal subjects trying to respond ''old'' on the basis of physical, as opposed to semantic, similarity tend to have either much higher false alarm rates or much lower conjunction scores than the LHc patients.

Another possibility might be that LHc patients are not perceiving the words as words, but only as their individual component syllables. Although this would explain the difference between their performance and that of the normal subjects, who would be expected to perceive the actual word and, thus, find it difficult to respond to repetitions of the components; it is unlikely that this is the explanation. The verbal intelligence of these patients is still within the normal range and their conversational skills are at least average. In addition, these patients were well-aware of their memory problem and most had developed attempts at compensation. They frequently said the word on the screen a second time and occasionally would even make a side comment on the word in an attempt to better commit it to memory.

The general pattern of results from Experiment 1, then, seem to support the contention that the left hippocampal system is critically important to the binding of the memory components of verbal stimuli. The full implication of these results will be considered in the final discussion. Next, however, we will report an experiment that attempts to isolate the consolidation process required for visual/spatial stimuli.

EXPERIMENT 2

Method

Design. Seven sets of visual stimuli were created: (1) abstract figures, (2) circle faces, (3) cartoon faces, (4) complex line-sketch faces (Reinitz, Lammers, & Cochran, 1992), (5) egg faces, (6) simple drawings of female faces, and (7) simple drawings of male faces. An example of the perceptual test with the abstract figures is presented in Fig. 2 and examples from each of the face sets is provided in Fig. 3.

The abstract figures each consisted of two designs inside of a frame. These were used to teach the tasks to the subjects.

Five of the face sets were used for the measurement of the conjunction effect on faces. The complex faces from Reinitz were too difficult to use to differentiate normals and hippocampal patients, but were used to compare our experimental technique with that of Reinitz et al. (1992).[3]

Each of the face sets was composed of a study-subset and a test-subset. All five faces within a study subset were different, but the eight test faces were related to the study faces in the following ways: two of the test faces were identical to two of the study faces, two test faces were ‘‘conjunctions’’ of the features of two of the study faces (e.g., one of the conjunction circle faces had the eyes of one of the study faces and the nose of another), two test faces had one of the features of a study face and one feature that had not appeared on any of the study faces, and two test faces were completely new. For example, if the study faces are designated by *Aa*, *Bb*, *Cc*, *Dd*, and *Ee*, the test faces might be *Aa* and *Bb* (true *Repetitions*), *Cd* and *Dc* (*Conjunctions*), *Ex* and *Xe* (single *Feature* repetitions), and *Yy* and *Zz* (totally *New* faces). Note that because of the sexual differences of the cartoon faces, eight study faces were required to obtain all of the types of test faces. Four of the study faces were male: *Aa*, *Bb*, *Cc*, and *Dd* and four were female: *Ee*, *Ff*, *Gg*, and *Hh*; with the test faces *Aa*, *Ee* (*Repetitions*), *Cd*, *Hg* (*Conjunctions*), and *Dx*, *Zh* (*Features*). For the circle faces, only the eyes and noses varied across the faces. For the cartoon, simple female, and simple male faces, the entire frontal face (eyes, nose, mouth, facial lines) constituted one feature set, and the head (hair, chin, ears) constituted the other. For the egg faces, the eyes and eye brows constituted one feature set, and the nose and mouth constituted the other. For the complex faces, one feature set consisted of the hair and the mouth and the other of eyes and nose. All subjects received the exact same set of stimuli in the exact same

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[3] The authors would like to thank Mark Reinitz for providing copies of his stimuli.

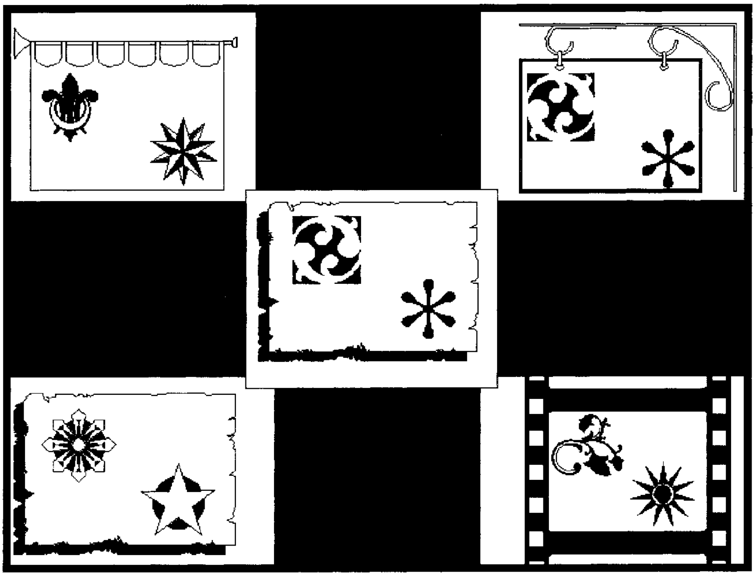


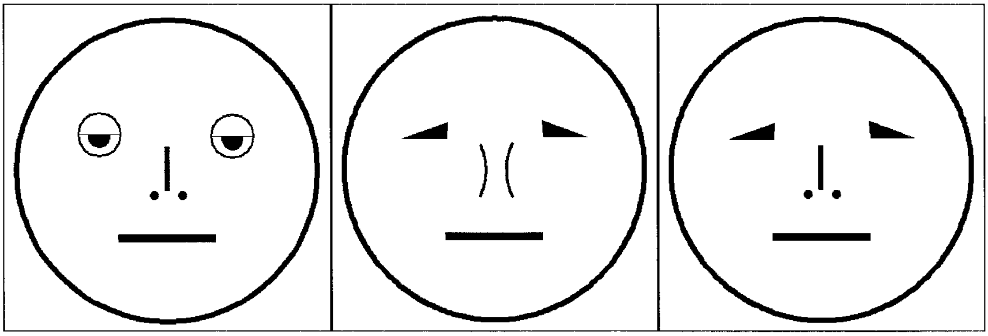
FIG. 2. Example of a perceptual test trial, with the abstract figures. The four study figures are in the four corners and a test figure is in the middle. In this example, the test figure is a *conjunction* of the upper right and lower left study figures.

order, but the ordering of the relationships were different across the different face sets. The purpose of having very different face sets was to reduce the probability that memory of features from earlier sets would influence a subject's classification of faces in the later sets.

For the abstract (practice) figures, the two internal designs constituted one feature set and the border the other feature. Only four study figures were used, but twelve test figures were created—four repeats, four conjunctions, and four feature repetitions.

Procedure. Each subject began with the practice tasks with the Abstract figures—first a memory task, then a perceptual task to help insure that the subject understood the instructions. All subjects were instructed that a test stimulus was to be designated as ''old'' only if both features were repeated *and* paired as they had been in the study set. (Some of the patients found it easier to say ''same'' for exactly like a study stimulus, or ''different'' if it were different in any way.) In the practice memory task, all four of the study figures appeared together on the screen for 30 seconds. Then the test figures appeared sequentially and subjects first judged each test figure as ''new'' or ''old'' and then rated their confidence in their judgment on a 1 (guessing) to 4 (confident) scale.

Immediately after the memory task, the same figures were used in the perception task. In the perception task, the same four study figures remained in the four corners of the monitor screen throughout the test, while the test figures appeared sequentially in the middle of the screen (Fig. 2). The subject's task was to judge if the center figure was identical to any of the corner figures, and then to give this decision a confidence rating. Although the primary purpose of this part of the procedure was to train the subject how to do the task and how to look for mispairings of old components, it also served as a measure of a subject's ability to perceive the figures. Although a few of the students were able to finish this part of the experiment in under 4 min most required approximately 12 min. The amount of time required for the patients and adult normals to



Circle Face: Study **Circle Face:** Study **Circle Face:** Test (Conjunction)

Cartoon Face: Study **Cartoon Face:** Study **Cartoon Face:** Test (Conjunction)

FIG. 3. Examples from each of the face sets.

complete this first set of tasks varied from 11 to 14.7 min. For the patients, the most time consuming and confusing aspect of the task had to do with the confidence ratings. Consequently, the confidence rating data will not be reported.

After the perceptual task, subjects began the facial memory tests. Each of the face tests consisted of a study phase and a test phase. Before each study phase, subjects were warned to pay close attention to how the components of the faces were put together and were shown examples of a ''new'' test face which consisted of components of ''old'' study faces.[4] In the study phase, the subject saw the current set of faces three times. The faces were shown for 10 per face during the trial, then 5 s each during the second and third trials.

The circle faces were tested immediately after the third study trial. Immediately following the testing of the circle faces, the subject received three study trials of the cartoon faces. This was followed by a list from the verbal experiment (lasting approximately 10 min) before presentation of the test of the cartoon faces. After the test of the cartoon faces, the study phase of the complex faces were presented. This was followed by the second list from the verbal conjunction experiment, prior to the test of the complex faces. The remaining three face tests were given in the second session,[5] the egg and simple female face tests

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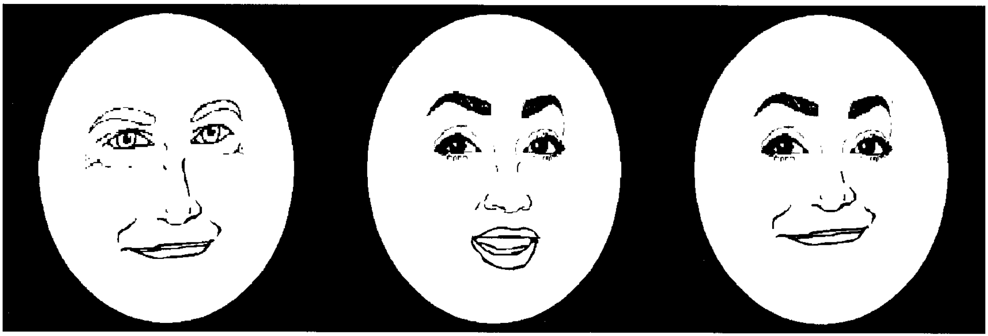
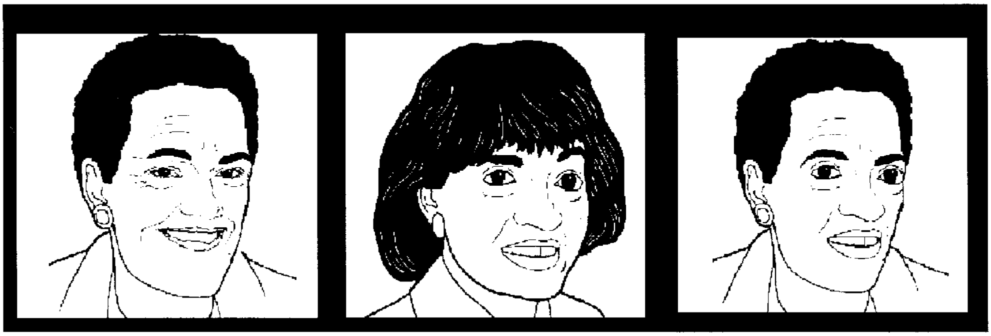
[4] Example faces were different from actual study and test faces.

[5] The lobectomy patients were only tested for one session and therefore were not tested on the last three face tests. All of the face sets were used for the remaining subjects, however, in order to improve the stability of their scores and in order to balance the order of the different conditions. No systematic differences were observed across lists.

Complex Face: Study
Complex Face: Study
Complex Face: Test (Conjunction)



Egg Face: Study
Egg Face: Study
Egg Face: Test (Conjunction)



Simple Female Face: Study
Simple Female Face: Study
Simple Female Face: Test (Conjunction)

Simple Male Face: Study
Simple Male Face: Study
Simple Male Face: Test (Conjunction)

FIG. 3—*Continued*

TABLE 4

AVERAGE PERCENTAGE OF ''OLD'' RESPONSES FOR EACH OF THE GROUPS IN EACH OF THE CONDITIONS OF THE MEMORY AND PERCEPTION TESTS USING THE ABSTRACT FIGURES

	Memory test			Perception test		
Subject	Repeat	Conj	Feat	Repeat	Conj	Feat
Students	86.1	18.1	5.6	100.0	0.0	0.0
Older Adults	72.2	33.3	5.6	98.6	2.8	2.8
RHc	65.6	46.9	18.8	96.9	6.3	6.3
LHc	78.6	46.4	17.9	96.4	32.1	7.1
BHc	100.0	100.0	50.0	100.0	0.0	0.0

before the third verbal list and the simple male face test after. In all three, the test phase occurred immediately after the study phase.

Results

Abstract figures. The results of the practice tasks with the figural stimuli are presented in Table 4.

Judging from the false alarm rate to the conjunction stimuli on the memory task, all of the subjects except the students found the abstract figures very difficult. Of course, this was also their first test, so some of the problem might have been to their difficulty in understanding the instructions. The subjects did much better on the perceptual task, however. Only the LHc patients had a high false alarm rate on the perception task—and this was due almost entirely to two of the left lobectomy patients. The average probability of saying ''old'' for the remaining five LHc patients were: *Repetitions* = 100%, *Conjunctions* = 5%, and *Features* = 0%. These results suggest that the subjects are able to perceive the differences among even these complex figures and able to make the required judgments.

Complex faces. Table 5 presents the scores of our subjects tested with the complex faces together with those of Reinitz et al., (Experiment 6, 1992). The major differences in methodology between the two versions of the test are: they had a 45 min visual discrimination filler task, we had a 10 min visually presented verbal filler task; they had a single study trial of 30 s per face, we had three study trials of 10, 5, and 5 s per face. It appears that our students did somewhat better than theirs, in that ours had a higher hit rate and lower false alarm rates in all of the nontarget conditions. However, even our students, who performed the best of our subject groups, had considerable difficulty with these stimuli. The facial features are just too complicated; there is too much similarity between the different features; and there is too much extra ''noise'' in the faces to allow subjects to make clean, strong discriminations. This is not to say that these faces were not good stimuli for Reinitz et al. Indeed, for their purposes they were ex-

TABLE 5

AVERAGE PERCENTAGE OF ''OLD'' RESPONSES FOR EACH OF THE GROUPS IN EACH OF THE CONDITIONS OF THE REINITZ FACES TEST

Subject	Repeat	Conj	Feat	New
Students	91.7	40.3	11.1	2.8
Older Adults	80.6	66.7	25.0	5.6
RHc	78.6	64.3	42.9	21.4
LHc	64.3	78.6	35.7	14.3
BHc[a]	100.0	100.0	50.0	50.0
Students[b]	71	52	19	13

[a] Although the other subjects in this experiment had a 10 min retention interval between study and test, the BHc patient was tested immediately after his study phase.

[b] From REINITZ et al. (1992, Experiment 6), 45 min retention interval.

TABLE 6

Average Percentage of "Old" Responses for Each of the Groups in Each of the Conditions of the Faces Tests

Subject	Repeat	Conj	Feat	New
Students	95.6	7.2	.6	2.5
Older adults	96.7	30.5	6.1	0.0
RHc	100.0	58.4	12.5	1.3
LHc	100.0	56.4	10.7	7.2
BHc[a]	100.0	100.0	25.0	0.0

[a] Although the other subjects had a 10 min retention interval between study and test with the Cartoon Faces, the BHc patient was tested immediately after his study phase with all faces.

cellent. However, in order to trace down the brain structures involved in the consolidation of the visual stimuli, we required stimuli which would allow us to differentiate normal and patient groups; i.e., stimuli which the nonpatient groups could remember more easily than they could either our abstract figures or the complex faces.

Other face tests. In order to reduce the amount of noise inherent in a single test with few items, the remaining Face tests, which were approximately all of the same level of difficulty, were averaged together. Because it was only possible to test the lobectomy subjects for one session, their scores were averaged over only their circle and cartoon faces tests. For all other subjects, their scores represent their averages over the five remaining face tests (circle, cartoon, egg, simple female, and simple male). These average scores are presented in Table 6.

The subjects found these faces easier to remember than those of Reinitz et al. (1992). The student subjects discriminated almost perfectly. The difference between patients and control adults in false alarm rates to the feature stimuli did not reach significance, $t_{32} = 1.83$, $SE_M = .038$. However, to measure the extent to which conjunction stimuli resulted in false alarms over and above that obtained from feature repetition, difference scores were found by subtracting each subject's false alarm rate to feature stimuli from that subject's false alarm rate to conjunction stimuli. The test performed on these difference scores found that the patients were more affected by the conjunction stimuli than were the control adults: .47 vs .26, $t_{32} = 2.65$, $SE_M = .081$. (It should be noted that the BHc patient, one of the LHc and two of the RHc patients, were not able to differentiate true repeats from conjunctions; not one of the 36 control subjects had this difficulty.) Thus, although the data from Experiment 1 are consistant with the hypothesis that damage to the left hippocampal systems is likely to lead to inferior binding of the memory traces of verbal stimuli, this experiment finds that damage to either left or right hippocampal systems may lead to inferior binding of the memory traces of visual/spatial information.

In an experiment studying memory performance before and after anterior temporal lobectomies, Saykin, Robinson, Stafiniak, Kester, Gur, O'Connor, and Sperling (1992) also found a dissociation between verbal memory deficits and visual/spatial memory deficits. Similar to the present findings, they reported that verbal deficits were much more likely to result from damage after left rather than right temporal lobectomy. Unlike the present results, they found that visual/spatial deficits tended to be specific to damage to the right hippocampal system. In fact, they even found improvements on visual/spatial memory performance resulting from left anterior temporal resection. This discrepancy may be due to a number of factors (e.g., age of seizure onset, extent of hippocampal resection, which was not quantified in their report, or time between surgery and test). It could also be a marker of differences between the tests employed. Standard memory tests have traditionally employed either recall tests or recognition tests in which old items are pitted against novel stimuli, rather than against stimuli which are recombinations of old elements. The experiments described here were designed to assess the function of binding features into distinct episodes: the ability to discriminate familiar from novel elements is necessary but not sufficient to accomplish this task.

DISCUSSION

The main finding of Experiment 1 was that patients with damage to their left hippocampal system were much more likely to show a high false alarm rate to new words made up of previously seen components than were other subjects, including patients with right hippocampal damage. Experiment 2 showed that patients with damage to either the left or the right hippocampal system were more likely to make false alarms to new faces composed of previously seen components than were subjects without such damage.

It is a well known fact that medial temporal lesions lead to memory disorders (Markowitsch, 1995; Squire & Zola-Morgan, 1991) and it may, therefore be tempting to dismiss our findings simply as ‘‘more of the same,’’ and perhaps conclude that our results demonstrate nothing much more than the rather nonsurprising expression of memory disorder in the form of ‘‘memory illusions.’’ We believe there is more to the story, but to appreciate it, these findings of ‘‘defective binding’’ must be considered in their proper context.

First, the production of memory conjunction errors, is not associated with, nor a consequence of, defective primary or working memory. There was no evidence of impairment in primary memory in our patients. Even global amnesics perform essentially normally on short-term memory tasks, and our patients were no exception. It is in this sense that we claim that cohesion is independent of primary memory.

Second, memory conjunction errors do not result simply from the adoption of an excessively low criterion in making positive recognition judgments. There were no obvious differences among the groups in their percentage of correct responses calculated over the ‘‘standard’’ test stimuli; i.e., hit rate minus false-alarm rate for the unrelated distractors. (In fact, on the verbal tests of Experiment 1, the adult controls scored lower as a group on this measure than did the patients.) Also, the face task, in which both patient groups made more conjunction errors than did normals, is not simply a more sensitive test than the verbal task, in which only the LHc patients made more conjunction errors. That is, some LHc patients (like AL) made many conjunction errors in the verbal task, but few in the face task.

Third, the verbal memory conjunction errors shown by the left mesial temporal damaged patients occurred primarily when the source materials for the erroneously joined elements were presented in close temporal proximity to one another, at short lags. This suggests at least three possibilities.

One explanation of our findings might be that everybody notices and implicitly produces memory conjunction errors, but only the patients exhibit source amnesia—do not remember that these were their own constructions. This alternative, however, is convincingly ruled out, at least in our verbal task, by the fact that control subjects were not capable of producing the ‘‘semantic’’ conjunctions even when asked to do so.

Another possibility is that the hippocampal patients have defective binding, i.e., they have stored the components of the stimuli, but not the relationships of these components. Thus, they are as likely (or nearly as likely) to respond ‘‘old’’ to the false conjunctions as they are to the true repetitions. However, the importance of the differential effect of lag suggests that there must also be some kind of temporal code that is coming into play at least over the relatively short temporal intervals used in Experiment 1.

A third possibility is, perhaps, a bit more speculative, but does seem to fit this data well. That is, the memory illusions may not reflect the ‘‘weakening’’ and attendant ‘‘disintegration’’ of engrams into ‘‘free-floating’’ components that then are recombined into episodically nonveridical units. Were this so, one might have expected to see more memory conjunction errors produced by the source words presented at longer lags. Our findings were exactly the opposite—memory conjunction errors were more prevalent at very short lags than at longer ones.

Thus, our data may imply the existence of a process, or a set of processes, that occur shortly after a perceived event, is independent of primary memory, is not a simple confusion in which elements of higher-order cognitive units are traded off against each other, is unrelated to the kinds of false alarms that all subjects make in recognition, and seems to be critically dependent on the integrity of the hippocampal system.

The previous explanation for our results was referred to as ‘‘defective binding,’’ but this label may be a misnomer, suggesting that too little binding was accomplished. This interpretation suggests, on the contrary, that the production of a large number of memory conjunction errors by the hippocampal patients was not as much defective as it was excessive. Perhaps neither the patients nor their controls exhibited any difficulty binding the elements they were expected to bind for the purpose of producing veridical engrams. Rather, the patients may have had difficulties in refraining from binding elements that should not have been so bound. The products of this excessive binding would be perfectly acceptable by the standards of semantic memory, but not by episodic memory, the actual task in which they were engaged.

As an alternative to the concept of ‘‘defective binding,’’ then, we are suggesting the possibility of a somewhat narrower hypothesis: the hypothesis of ‘‘disinhibition of binding.’’ We concur in the judgment of others (Cohen & Eichenbaum, 1993, pp. 286–288; Eichenbaum & Bunsey, 1995; Metcalfe et al. 1992; Wickelgren, 1979) that the binding process exists, and that its function consists in ‘‘gluing’’ together the elements of the incoming information into separately retrievable engrams in the long-term storage. This process is (a) facilitated by the novelty of the incoming information (Tulving & Kroll, 1995) in the hippocampal and temporal regions (Tulving, Markowitsch, Kapur, Habib, & Houle, 1994), (b) guided by the ‘‘templates’’ provided by the information already available in neocortical storage regions, and (c) inhibited by the temporal ‘‘chunks’’ of information in the hippocampal formation. The inhibitory component of the binding operation allows the formation of only those conjunctions of elements as long-term engrams that correspond to the temporally organized chunks in primary memory. When the inhibitory component fails, elements corresponding to higher-order units in long-term memory are created heedlessly.

This hypothetical scheme is largely consistent with what is known about memory phenomena in normal people and brain-damaged patients, as well as the new observations we have made in the present study. Among other things, it clarifies one puzzling aspect of the results from our first experiment, namely that the tendency to falsely recognize conjunction words was especially strong under the short lag conditions.

We did expect to find a higher rate of memory conjunction errors in the patients on the basis of the growing evidence that the hippocampal system is somehow involved in the ‘binding’ of stimulus components into composite engrams. Although normal subjects may at times have false memories caused by the conjunctions of previously seen components reconfigured into new composites (e.g., Reinitz & Demb, 1994; Reinitz et al., 1992; Underwood & Zimmerman, 1973), patients with damage to their hippocampal systems were expected to produce such false memories even with simple stimuli presented under optimal conditions. We assume that the observations we have made about binding, its inhibition and disinhibition, also hold for normal subjects. Because of their intact hippocampal systems, however, binding is kept from running out of control by the inhibitory processes, presumably of the kind that enable the hippocampus to eliminate inappropriate alternatives (McNaughton, 1994).

Other investigators have demonstrated that amnesics have particular difficulty in forming new associations (e.g., Paller & Mayes, 1994; Schacter, Church, & Bolton, 1995)—and this too may be an example of the binding deficit resulting from damage to the hippocampal system. However, to the best of our knowledge, these experiments are the first to demonstrate the importance of the hippocampal system to the binding of the constituent parts of individual stimuli—an importance that had been speculated upon by Cohen & Eichenbaum (1993, pp. 286–288).

Our ideas concerning normal inhibition of hippocampal binding, and disinhibition of the process following hippocampal damage are related to the suggestion by Eichenbaum et al. (1994) that neocortical association areas are responsible for maintaining brief representations of ‘‘specific items and events prior to hippocampal processing as well as providing the final repositories of long-term memories’’ (p. 449). They are also related to the suggestions made by Alvarez and Squire (1995) that (a) representations of stimuli presented in temporal proximity may be maintained concurrently within the neocortical system, (b) the nature of hippocampal and neocortical processing is not sequential, but rather normally extremely interactive—that the hippocampal system ‘‘directs consolidation by gradually changing the organization of cortical representations . . . by strengthening connections between the cortical sites that participate in representing a memory’’ (Squire & Alvarez, 1995, p. 172), (c) one aspect of this hippocampal contribution is to bind stimulus components as they are maintained in the neocortical system, and, thus, (d) in the absence of a fully functional hippocampal system, concurrent components are more easily reformed into false memories. With longer lags, the components are less likely to be at approximately the same strength at the same time within the neocortical system and, thus, less likely to recombine into false memories.

In summary, then, we suggest that some memory illusions—recollection of events that never occurred—have their roots in the failure of the process of binding of informational elements into coherent, separately accessible, long-term engrams. Hippocampal damage results in such failure, implying that an intact hippocampus plays a critical role in the normal binding process. In addition, our data suggests that the hippocampus may also play an inhibitory rôle so that when the hippocampal system is damaged, the inhibitory component of the binding process is nonoperational, and long-term engrams are constructed from the available elemental constituents without the early-encoding constraints. Some of the reappear at retrieval as memory illusions.

This ability of the hippocampal amnesic to remember components, combined with their inability to restrict this binding may not only be responsible for their mistakenly recognizing new composites (i.e., false memories)— but may also be what allows them, under at least some circumstances, to form prototypes even when the individually experienced items are not well remembered (e.g., Knowlton & Squire, 1993). That is, it is possible that recognition of the prototype may not imply separate systems for learning category-level and item-level knowledge (Knowlton & Squire, 1993) or a memory trace of a prototype resulting from the extraction of the central tendency from a set of similar experiences (McClelland & Rumelhart, 1986, pp. 207–208; Solso & McCarthy, 1981). Rather, the ‘‘false’’ (or ‘‘pseudo’’) memory for a prototype may simply be the result of binding failure.

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