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Successful Performance by Monkeys With Lesions of the Hippocampal Formation on AB̄ and Object Retrieval, Two Tasks That Mark Developmental Changes in Human Infants

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In this study, (a) what determines success or failure on the AB̄ and object retrieval tasks and (b) the relation between brain maturation and cognitive development as indexed by these tasks were examined. Specifically, does improved performance on these tasks with age reflect maturation of memory functions dependent on the medial temporal lobe? In AB̄, the S watches a reward being hidden in 1 of 2 wells; after a brief delay S reaches for that reward. The AB̄ error consists of the S continuing to reach to the first location (A) when side of hiding is shifted to the second location (B). In object retrieval, a reward is placed in a transparent box open on 1 side. Although the reward is visible through all sides of the box, it can only be retrieved through the 1 open side. Intact cynomolgus monkeys and those with bilateral lesions of the hippocampal formation (H^+) were tested. Although H^+ monkeys exhibited impaired memory by performing poorly on the delayed nonmatching to sample task, they performed well on AB̄ at delays of 2–15 s. Performance declined as delays increased to 30 s, but H^+ monkeys never showed the AB̄ error pattern. On object retrieval, H^+ monkeys succeeded quickly and efficiently, even when required to detour to the box opening. This research demonstrates that memory impairment alone cannot account for deficits on AB̄ or on object retrieval and strengthens the conclusion (Diamond, 1988a, 1988b, in press) that improved performance on AB̄ and object retrieval during infancy reflects maturation of dorsolateral prefrontal cortex.

The AB̄ task (pronounced "A not B"), first devised by Piaget in the 1930s (Piaget, 1937/1954), is a reliable, much studied marker of developmental change during the second half of the 1st year of life (for excellent reviews, see Gratch, 1975; Schuberth, 1982; Harris, 1986; Wellman, Cross, & Bartsch, 1987). In the task, the subject watches as a desired object is hidden in one of two identical wells. A delay of 0–10 s is imposed, and the subject is then allowed to reach.

Infants of 7½–9 months err on the AB̄ task at delays of 2–5 s, exhibiting the pattern of behavior from which the task derives its name (Diamond, 1985; Fox, Kagan, & Weiskopf, 1979; Gratch & Landers, 1971). (Infants under 7½ months cannot uncover a hidden object and so cannot be tested on this task.) Specifically, they find the object correctly at the first hiding place (Location A), but when side of hiding is reversed to Location B, they reach back to Location A. Thus, they find the object at A, but not at B. Performance improves with age, so that by 12 months, infants find the hidden object correctly at both A and B with delays as long as 10 s (Diamond, 1985).

The AB̄ task clearly involves memory. When there is no delay, infants do not err (Diamond, 1985; Gratch & Landers, 1971), and with increasing age longer delays are required to produce errors (Diamond, 1985; Fox et al., 1979). Even infants who are making the AB̄ error begin to perform correctly *within the same session* when the delay is reduced (Diamond, 1985). Infants also perform well if they are allowed to circumvent the memory requirements of the task by looking at the correct well throughout the delay, positioning themselves in front of the correct well, or straining toward the correct well throughout the delay (Cornell, 1979; Diamond, 1985; Fox et al., 1979; Goldfield & Dickerson, 1981; Gratch, Appel, Evans, LeCompte, & Wright, 1974).

In contrast to the failure of 7½- to 9-month-old infants on the AB̄ task at delays as brief as 2–5 s, infants of only 2–5 months can demonstrate the effects of conditioning, show habituation, and succeed on visual paired comparison tasks over delays of hours, days, and weeks (conditioning: Lipsitt, 1969; Papousek, 1961; Rovee-Collier, 1984, 1986; habituation: Baillargeon, Spelke, & Wasserman, 1985; Cohen, Gelber, & Lazar, 1971; visual paired comparisons: Fagan, 1973; Fantz, 1964). One possible explanation for the finding

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that infants fail AB at brief delays, despite succeeding on other tasks at long delays, is that AB requires both memory and some additional ability. When the subject reaches to A and retrieves the reward, the response of reaching to A is reinforced. Successfully retrieving the reward when it is subsequently hidden at B thus requires resisting a repetition of the reinforced response of reaching to A. To perform correctly, subjects need to remember that the reward had just been hidden at B, and they must also inhibit a tendency to reach to A.

If success on AB depends on memory *and* the ability to inhibit a strong response tendency, then correct performance on AB would be expected to depend crucially on the integrity of prefrontal cortex. Damage to prefrontal cortex in monkeys and humans disrupts the normal flexibility of behavior, resulting in failures to inhibit prepotent responses (Diamond, 1988b, in press; Luria, 1966; Milner, 1963; Perret, 1974). Tasks that require both memory and inhibition are the ones most firmly linked to prefrontal function. Indeed, AB is very similar to the delayed response task, and delayed response in monkeys is severely impaired by lesions of dorsolateral prefrontal cortex (for reviews, see Fuster, 1980; Goldman-Rakic, 1987; and Rosenkilde, 1979).

An alternative explanation is that the kind of memory required for AB is different from the kind of memory required by conditioning, habituation, and visual paired comparisons. The latter tasks may depend on procedural or implicit memory (Cohen, 1984; Schacter, 1987; Squire, 1982, 1986), whereas AB may require declarative or explicit memory. If success on AB depends on declarative memory, then correct performance on AB should depend crucially on the hippocampal formation and related structures in the medial temporal region. Monkeys with lesions of the hippocampal formation (Mahut, Zola-Morgan, & Moss, 1982; Mishkin, 1978; Zola-Morgan & Squire, 1985) and patients with amnesia attributable to similar damage (Scoville & Milner, 1957; Zola-Morgan, Squire, & Amaral, 1986) are impaired on declarative, explicit memory tasks but not on procedural, implicit memory tasks.

Recently, monkeys with dorsolateral prefrontal lesions were shown to fail AB at delays similar to those failed by 7½- to 9-month-old human infants (Diamond & Goldman-Rakic, 1989). This finding is consistent with the suggestion (Diamond, 1985, 1988a, 1988b) that the AB task depends on prefrontal cortex and that successful performance requires both memory and the inhibition of prepotent response tendencies. It remains possible, however, that the AB error pattern may be a typical feature of impaired memory alone. When memory is impaired, the two hiding places may not be forgotten to the same degree. Thus, if the second hiding place, Location B, is remembered less well than the first hiding place, Location A (e.g., because of proactive interference), AB errors would tend to occur.

In the present study, we evaluated the AB performance of monkeys with bilateral lesions limited to the hippocampal formation. The questions of interest were the following: (a) Would monkeys with bilateral lesions of the hippocampus fail AB at the same delays as do human infants? (b) Would hippocampal monkeys show the characteristic AB error pattern at any delay? In order to obtain an independent measure of memory function during the time that AB was administered, we tested monkeys on the trial-unique delayed nonmatching to sample task, a test of recognition memory for objects (Gaffan, 1974; Mishkin & Delacour, 1975), both before and after AB testing.

We also evaluated the performance of monkeys with lesions of the hippocampus on the object retrieval test, which is a detour problem using a transparent barrier (Diamond, 1988c). Human infants improve on this task during the same period (7½–9 months) that they improve on AB. Unlike AB, object retrieval does not appear to place demands on memory. It resembles AB, however, in that infants must overcome a predominant response tendency. They must resist reaching directly for the visible reward and instead detour around the barrier. Like AB, success on object retrieval depends on the integrity of dorsolateral prefrontal cortex (Diamond & Goldman-Rakic, 1985).

Method

Subjects

Six cynomolgus monkeys (*Macaca fascicularis*) were used. Three monkeys (all female) received bilateral lesions of the hippocampal formation, and 3 unoperated monkeys (2 male and 1 female) were used as a control group for AB. Two of the 3 unoperated monkeys who were tested on AB also served as the control group for the object retrieval task. All monkeys had prior testing experience. The 3 operated monkeys had undergone surgery 3 years prior to the start of the present study. They had completed testing on a series of tasks (delayed nonmatching to sample, pattern discrimination, retention of object discriminations, concurrent object discrimination, delayed response, and motor skill learning). Their performance on tests is reported elsewhere (Zola-Morgan & Squire, 1986; Zola-Morgan, Squire, & Amaral, 1989). The control monkeys had been tested on several of the same tasks (Salmon, Zola-Morgan, & Squire, 1987).

Finally, to evaluate the performance of the monkeys with hippocampal formation lesions on the delayed nonmatching to sample task, we used data for 3 monkeys from another study (Zola-Morgan, Squire, & Amaral, in press) for comparison. These monkeys, in which the amygdaloid complex had been damaged bilaterally without damage to the surrounding cortex, had performed normally on several different memory tasks. They had been given the delayed nonmatching to sample task on two occasions, once as their first postoperative test and again 1½ years after surgery.

Surgery

All surgery was performed under sodium pentobarbital anesthesia (30 mg/kg). The hippocampus on each side was approached by elevating the occipitotemporal convexity and entering the brain medial to the occipitotemporal sulcus and caudal to the entorhinal cortex. The hippocampus, including dentate gyrus and subicular cortex, was removed. The removal also included most of the parahippocampal gyrus (area TF–TH of Bonin & Bailey, 1947) and the posterior half of the entorhinal cortex. The upper surface of the lateral ventricle served as an identifiable dorsal boundary along the entire length of the removal. In this way it was possible to spare the temporal stem during surgical removal of the hippocampus (Zola-Morgan, Squire & Mishkin, 1982). We use the designation H^+ for this operated group, because the removal includes the hippocampus proper plus adjacent cortex.

Table 1
Three Types of Trials That Occur in the "A not B" Task

Trial type	Response on previous trial	Side of hiding on new trial
1. Repeat following correct	Correct	Same
2. Reversal	Correct	Reversed
3. Repeat following error	Error	Same

Repeat = the reward is hidden in the same place as on the preceding trial.

Behavioral Testing

All testing was carried out in a Wisconsin General Test Apparatus (WGTA; Harlow & Bromer, 1944). Monkeys were tested first on the AB̄ task and then on the object retrieval task. In addition, the H+ monkeys were administered the trial-unique delayed nonmatching to sample task just prior to AB̄ and again after completion of object retrieval testing. Administration of the delayed nonmatching to sample task provided an independent measure of memory impairment before and after the AB̄ and object retrieval tests were administered.

The AB̄ task. The experimenter sat facing the caged monkey across a table that supported the stimulus tray. An opaque screen could be lowered between the monkey and the stimulus tray to prevent the monkey from observing the stimuli during the delay intervals. The stimulus tray contained three food wells located 16 cm in front of the cage bars and spaced 16 cm apart. All monkeys underwent a 2-week pretraining procedure to familiarize them with the apparatus, experimenter, and experimental procedure.

To administer the AB̄ task, the opaque door was raised while one of the two lateral wells was baited with a raisin, or another preferred food, as the monkey watched. If there was any doubt about whether a monkey had seen where the bait was hidden, the entire hiding procedure was repeated. Both food wells were then covered simultaneously with identical 3-in. (7.6-cm) square, black plastic plaques, and the opaque screen was lowered between the monkey and the food wells. After the delay, the opaque screen was raised, and the monkey was allowed to choose between the two covered wells and, if correct, to retrieve the food. If the monkey reached incorrectly, the experimenter directed the monkey's attention to the correct well, exposed the reward, but did not permit the monkey to retrieve it.

The reward was hidden in the same well until the monkey made two correct responses in a row. The side of hiding was then reversed, and the same sequence was repeated until two responses in a row were made to the correct side. In each daily session, every monkey received at least four of these reversal sequences. If four reversals were successfully completed in fewer than 20 trials (the minimum number of trials for a perfect session was 12), monkeys were given one additional reversal. As a result, unless a monkey was performing poorly, five reversals were given in a session. In this way, monkeys that made fewer errors per reversal obtained about the same number of trials each day as did monkeys that made more errors per reversal. Testing sessions continued until a monkey made two consecutive correct responses on the fourth (or fifth) reversal, or until the monkey had completed 30 trials. The intertrial interval was approximately 20 s. The side of hiding on the 1st trial alternated from day to day.

The response requirements of the AB̄ task were such that monkeys could eventually respond correctly on all trials by developing a double alternation strategy (i.e., L, L, R, R, L, L, etc.). The following procedure was used to minimize this possibility: If a monkey performed errorlessly in any daily session, the response requirement for one of the reversals in the following session was changed from two consecutive correct responses to three consecutive correct responses. The side of this reversal and whether it occurred early or late in the session was varied randomly.

All monkeys were tested at four successive delay intervals: 2 s, 5 s, 10 s (for 14 sessions), and 15 s (for 8 sessions). The 3 H+ monkeys, but not the control monkeys, were also tested for 8 sessions at a 30-s delay. All monkeys were tested 5 consecutive days per week.

Before each delay increment, all monkeys received five sessions in which the delay was increased gradually over days. Thus, for example, there were five sessions in which the delay was increased from 0 to 2 s before testing at the 2-s delay and five sessions in which the delay was increased from 2 to 5 s before testing at the 5-s delay. Performance during these transition sessions was not included in data analysis.

Scoring criteria for the AB̄ task. When AB̄ is administered as just described, three types of trials can occur depending on whether performance on the previous trial was correct or incorrect and on whether the side of hiding changes or remains the same (see Table 1). The scoring of the AB̄ task in this investigation was the same as that used for human infants (Diamond, 1985) and for monkeys with lesions of prefrontal cortex (Diamond & Goldman-Rakic, 1989). The essential feature of the AB̄ error is that the subject makes an error when the side of hiding is reversed (i.e., errors occur on Reversal trials).

Object retrieval. A transparent Plexiglas box (7 cm × 7 cm × 7 cm), open on one side, was affixed to a round wood base (10 cm in diameter) and attached to the tray area of the WGTA. The box and base were mounted on a track that ran parallel to the front of the testing cage. The box could be rotated 360° in the horizontal plane and could be positioned anywhere along the length of the track. When the box was appropriately positioned for a trial, a locking mechanism prevented movement.

All sessions were videotaped with a camera mounted in a stationary position directly in front of the WGTA. A monitor placed out of view of the monkey allowed the experimenter to watch the monkey unobserved. For several days prior to formal testing, monkeys were acclimated to the presence of the video camera and to the video lights.

During testing, the opaque screen was lowered, the experimenter positioned the box and the reward, and then locked the box in place. The reward was either a raisin or a small piece of fresh fruit. The box was oriented in one of three ways: the open side faced the monkey (front open) or it faced to the left or right of the monkey. To vary the difficulty of the trials, two other features of the task were manipulated: The box was positioned along the track in one of three positions: at the center of the testing tray (where it was for most of the trials), to the far left, or to the far right. Second, the reward was located in one of three places: partly outside the opening of the box, inside the box approximately ¼ in. (0.635 cm) from the opening, or deep inside the box. When the box and the reward had been appropriately placed, the experimenter moved out of sight of the monkey and raised the opaque screen. The opaque screen remained raised until the monkey retrieved the reward or until the monkey made no further attempt at retrieval for 30 s.

Each daily session consisted of 21 trials, always in the same sequence (see Table 2). On 3 of the trials the open side of the box faced the monkey, on 9 trials it faced left, and on 9 trials it faced right. Monkeys were tested on consecutive days until the reward was retrieved within 3 s on every trial in a session. The measures of interest were the number of sessions required to reach this performance criterion and the number of trials required to succeed at each of the three orientations. Monkeys were considered to have succeeded at a particular orientation of the box opening when they retrieved the reward at that orientation on 2 consecutive trials within 3 s on each trial.

Trial-unique delayed nonmatching to sample. This test was administered in the same way on two occasions, once prior to AB̄ and

Table 2
Testing Sequence for Object Retrieval

Trial	Orientation of box opening	Position of box on track	Position of reward in box
1	Front	Center	Deep inside
2	↓	↓	↓
3	Right		Partly outside
4	↓		Deep inside
5			Partly outside
6			Inside, near opening
7	↓		Deep inside
8	Left		Partly outside
9	↓		Deep inside
10			Partly outside
11			Inside, near opening
12			Deep inside
13	↓		↓
14	Right	↓	
15	↓	Far right	
16	Left	↓	
17	Right	Far left	
18	Left	↓	
19	↓	Center	
20	Right	↓	
21	Front	↓	↓

once after completion of the object retrieval task. The interval between the two delayed nonmatching to sample tests was approximately 40 months for the H+ monkeys and 16 months for the control monkeys. Each trial consisted of two parts: Monkeys first displaced an object covering the central well to obtain a raisin reward; then, after 8 s, they saw two objects—the original one and a new one—and they had to displace the new object to obtain the reward. Twenty such trials were presented daily with an intertrial interval of 20 s. Each trial used a new pair of objects, selected randomly from a collection of more than 300 junk objects. After reaching the learning criterion of 90 correct choices in 100 trials, monkeys were tested with successively longer delays of 15 s, 60 s, and then 10 min between presentation of the sample and the choice parts of the trial. One hundred trials were given at both the 15-s and 60-s delays, and 50 trials were given at the 10-min delay. (For a more detailed description of this task, see Zola-Morgan & Squire, 1985.)

Results

Histological Findings

The brains were blocked in the coronal plane, placed in fixative, and encapsulated in egg albumin prior to cutting. Frozen sections were cut at 50 μm, and every fifth section was mounted on slides and stained with thionine. Monkeys H+1 and H+2 sustained complete bilateral hippocampal removals (see Figure 1). The entorhinal cortex and the parahippocampal gyrus were also extensively damaged bilaterally. Monkey H+3 sustained a smaller lesion, involving about half of the hippocampal formation bilaterally. Damage to the parahippocampal gyrus was less extensive in this animal, and the entorhinal cortex was only slightly involved. This animal evidenced some atrophy in the lateral geniculate nucleus bilaterally, and the optic radiations appeared to have been damaged on the left side. In Monkeys H+1 and H+3, the amygdaloid complex was entirely spared. In Monkey H+2 there appeared to be slight direct damage of the amygdaloid complex involving the ventral limit of the posterior border of the lateral nucleus. Detailed descriptions of the extent of damage in each of the 3 H+ monkeys can be found in Zola-Morgan et al. (1989).

Behavioral Findings

The AB̄ Task

For each delay interval, we calculated the overall percentage of correct trials in each daily session, as well as the percentage correct for each of the three trial types that could occur during a session (Repeat following correct, Reversal, and Repeat following error; see Table 3). Figure 2 shows the scores on each of the three trial types.

Delays of 2–5 s. For the 2-s and the 5-s delay intervals, the control (C) group and the group with hippocampal formation lesions (H+) performed similarly (see Figure 2). Both groups performed better than 90% correct overall (C = 91%;

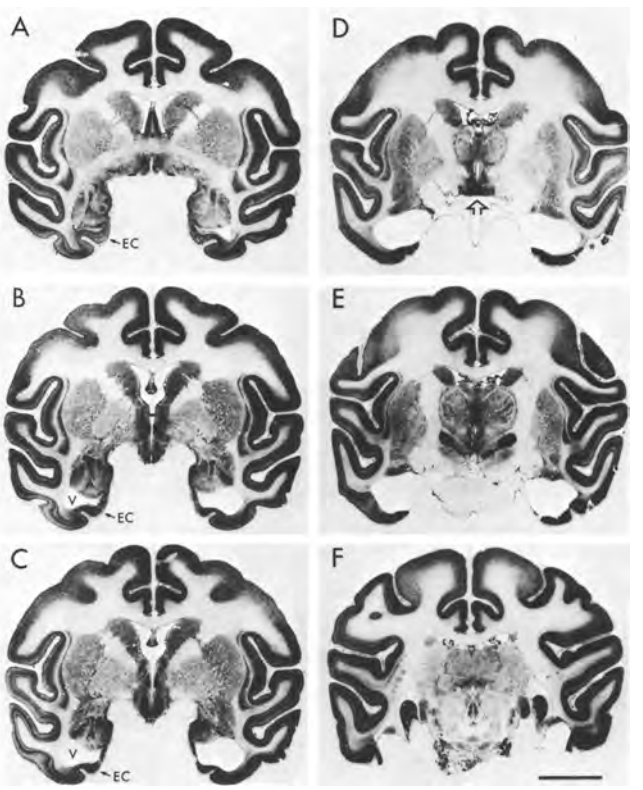

Figure 1. Representative thionine-stained coronal sections arranged from rostral (Panel A) to caudal (Panel F) through the hippocampal formation of Monkey H+1. (The amygdaloid complex [labeled *A* in Panels A–C] was not involved in the lesion nor was the rostral half of the entorhinal cortex [Labeled *EC* in Panels A–C]. The posterior half of the entorhinal cortex and the full rostrocaudal extent of the hippocampal fields were completely and bilaterally removed, as were the parahippocampal fields TH and TF. Asterisks in Panels D and E indicate damage produced in the histological processing. V = ventricle. Bar equals 10 mm. See Plate S.)

Table 3
Performance of Monkeys With Lesions of the Hippocampal Formation and Unoperated Controls on the "A not B" Task by Type of Trial and Length of Delay

Experimental groups	All trials	Repeat following correct	Reversal	Repeat following error
		2-s delay		
Hippocampal				
H+1	98 (185)	99 (97)	98 (70)	100 (4)[a]
H+2	100 (182)	100 (98)	100 (70)	— (0)[a]
H+3	95 (190)	97 (96)	91 (70)	100 (10)
M	98 (186)	99 (97)	96 (70)	100 (5)
Controls				
C1	92 (199)	93 (98)	90 (70)	88 (17)
C2	99 (183)	99 (97)	99 (70)	100 (2)[a]
C3	87 (210)	90 (99)	84 (69)	89 (28)
M	92 (197)	94 (98)	91 (70)	93 (16)
		5-s delay		
Hippocampal				
H+1	93 (199)	92 (100)	93 (70)	100 (14)
H+2	88 (205)	93 (97)	93 (70)	67 (24)
H+3	95 (185)	99 (91)	89 (70)	90 (10)
M	92 (196)	95 (96)	92 (70)	86 (16)
Controls				
C1	96 (187)	100 (96)	91 (70)	100 (8)[a]
C2	91 (198)	93 (98)	87 (71)	100 (17)
C3	84 (218)	76 (97)	83 (70)	89 (35)
M	90 (211)	90 (97)	87 (70)	96 (20)
		10-s delay		
Hippocampal				
H+1	87 (204)	92 (94)	84 (70)	85 (26)
H+2	86 (219)	87 (105)	91 (70)	71 (31)
H+3	80 (239)	79 (107)	84 (69)	82 (49)
M	84 (221)	86 (102)	85 (70)	79 (35)
Controls				
C1	95 (193)	96 (99)	94 (70)	80 (10)
C2	91 (196)	94 (95)	87 (70)	94 (17)
C3	85 (211)	92 (95)	73 (70)	84 (32)
M	90 (200)	94 (96)	85 (70)	86 (20)
		15-s delay		
Hippocampal				
H+1	69 (158)	73 (64)	60 (40)	78 (49)
H+2	92 (123)	92 (58)	90 (40)	90 (15)
H+3	73 (145)	81 (59)	56 (39)	82 (39)
M	80 (142)	82 (60)	69 (40)	83 (34)
Controls				
C1	96 (111)	95 (57)	95 (40)	100 (5)[a]
C2	90 (120)	92 (59)	83 (40)	100 (13)
C3	94 (110)	100 (55)	88 (40)	86 (7)[a]
M	93 (114)	96 (57)	85 (40)	96 (8)
		30-s delay		
Hippocampal				
H+1	62 (119)	79 (43)	64 (28)	47 (43)
H+2	71 (177)	72 (62)	70 (37)	49 (69)
H+3	59 (175)	73 (59)	56 (36)	56 (71)
M	64 (157)	75 (55)	63 (34)	51 (61)

Note. Numbers show percent correct. Number of trials is given in parentheses. Only monkeys with lesions of the hippocampal formation were tested at the 30-s delay. They were tested at that delay to determine if once their memory was strained they would show the "A not B" error pattern. They did not. Note that the average percent correct score for all the trials includes performance on the first trial of each daily session, but performance on this trial does not contribute to the score for any of the three trial types.
[a] Data based on fewer than 10 trials do not yield a reliable percentage.

H+ = 95%). The control group required an average of 14.6 trials per session to achieve criterion performance at the initial hiding place and to complete all reversal sequences. Monkeys with H+ lesions required 13.6 trials per session. An analysis of variance (ANOVA; Two Groups × Three Trial Types) revealed no group difference, $F(1, 4) = 0.7$, $p > .10$; no difference across the three trial types, $F(2, 8) = 1.2$, $p > .10$; and no interaction, $F(2, 8) = 1.2$, $p > .10$.

Delays of 10–15 s. The monkeys with H+ lesions performed marginally worse overall than the control monkeys (81% correct vs. 91% correct, respectively), $t(4) = 2.29$, $p < .09$. H+ monkeys required 16.8 trials per session to complete all reversal sequences; control monkeys required 14.3 trials per session, $t(4) = 3.7$, $p < .05$. Nevertheless, the two groups performed similarly across the three trial types. An ANOVA (Two Groups × Three Trial Types) revealed no effect of group or trial type, and no interaction (*p*s > .10). Within each group there were no significant differences in performance across the three trial types, and there were no significant differences between the two groups on any of the three types of trials at either the 10-s or the 15-s delay. Thus, at delays of 10–15 s, the H+ monkeys exhibited no tendency to commit the AB̄ error.

Delays of 30 s. Monkeys with hippocampal formation lesions performed more poorly at the 30-s delay than at the shorter delays, requiring 20 trials per session to complete all reversal sequences. Their overall score on all trials at the 30-s delay was 64% correct, compared with 95% at delays of 2–5 s and 82% at delays of 10–15 s, $F(2, 4) = 42.6$, $p < .01$. Although control monkeys were not tested at the 30-s delay, the overall score of 64% obtained by the H+ monkeys is likely to represent impaired performance. First, the H+ monkeys were marginally impaired ($p < .09$) at the shorter delays of 10–15 s. Second, these same H+ monkeys were severely impaired at a 30-s delay on the similar delayed response task (Zola-Morgan et al., 1989).

Despite this indication that performance was poor overall at the 30-s delay, H+ monkeys did not exhibit the AB̄ error. H+ monkeys performed similarly on Reversal trials and Repeat-following-correct trials (63% vs. 75%, $p > .10$). Moreover, H+ monkeys performed significantly above chance on Reversal trials, $t(2) = 4.0$, $p < .05$, and they scored about the same on the Reversal trials (63%) as they did overall (64%).

We also examined in more detail the performance of H+ monkeys on Repeat-following-error trials. Their score for this type of trial was poor (51%) and significantly worse than for Repeat-following-correct trials (75%; $p < .05$). Repeat-following-error trials indicate that a string of at least two errors has occurred. The first error of such a string can occur on a Reversal trial or on a Repeat-following-correct trial. Because errors on Reversal trials are indicative of the AB̄ error, if the subject continues to reach incorrectly over the next series of trials, that may reasonably be taken as further evidence for the AB̄ error. However, an error or string of errors following a correct reach when the side of hiding has not changed (i.e., a string of errors beginning on a Repeat-following-correct trial) would *not* be indicative of the AB̄ error. Thus, an important question is the following: For each

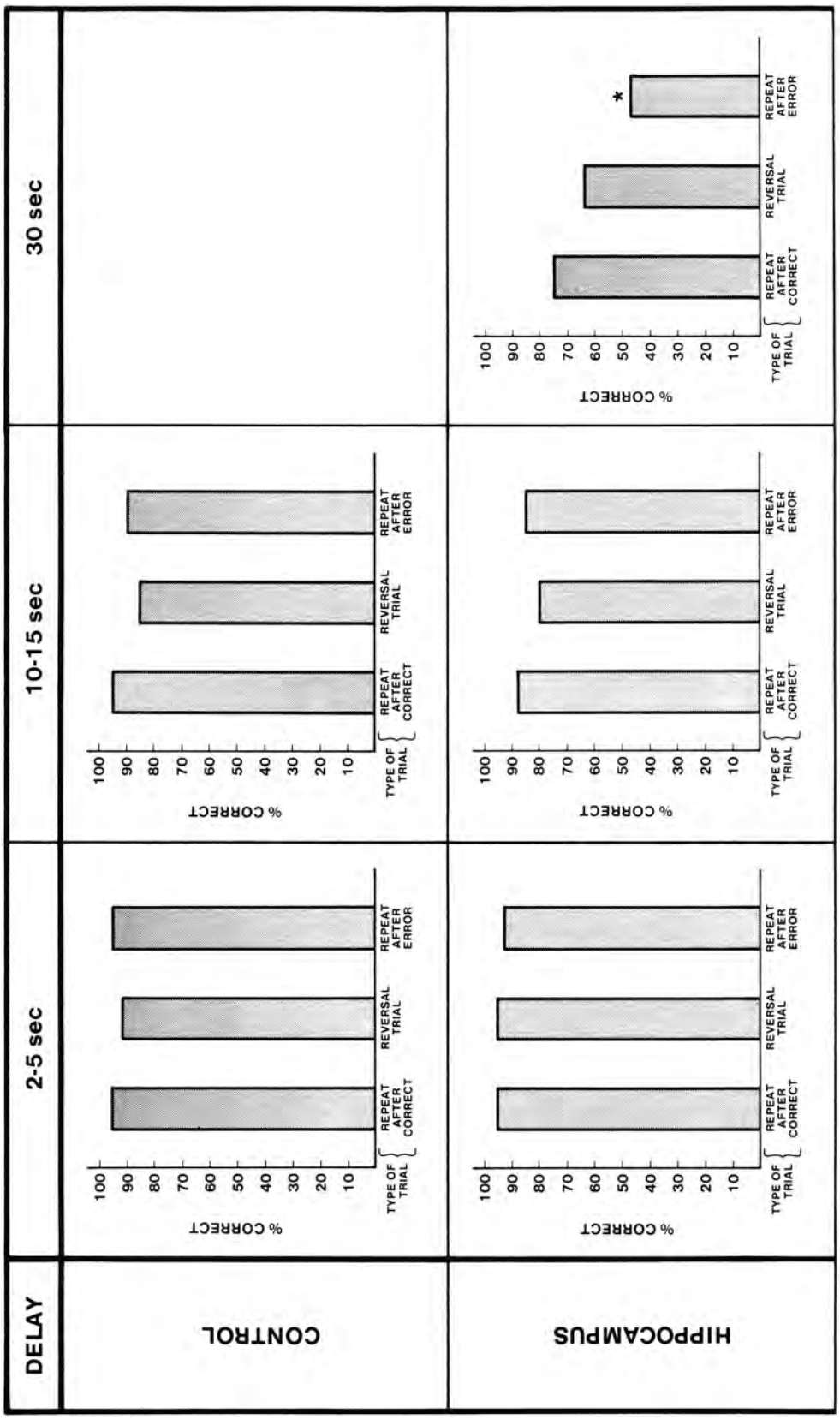

Figure 2. Performance of monkeys on the three types of trials that occur during administration of the "A not B" task. (The results for control monkeys [$N = 3$] and for monkeys with hippocampal formation lesions [$N = 3$] are presented. Delay refers to the interval between when the experimenter hid the reward and when the subject was allowed to reach. *Significantly different from performance on Repeat-following-correct trials, $p < .05$.)

Table 4
Trials Needed to Reach Criterion on the Object Retrieval Task

Group	Orientation of the opening of the box		
	Front open	Right open	Left open
Control (C)			
C1	2	8	10
C2	2	26	12
M	2.0	17.0	11.0
Hippocampus (H^+)			
H^+1	4	20	25
H^+2	2	12	11
H^+3	2	12	12
M	2.7	14.7	16.0

string of Repeat-following-error trials, did that string immediately follow an error on a Reversal trial or not?

H^+ monkeys were no more likely to repeat an error after a Reversal trial than after a Repeat-following-correct trial. At the 30-s delay, they erred on a total of 37 Reversal trials. They were then correct on 46% of the Repeat-following-error trials immediately following these 37 reversal errors. At the 30-s delay, H^+ monkeys also erred on a total of 36 Repeat-following-correct trials. They were then correct on 53% of the Repeat-following-error trials immediately following those errors. Accordingly, the performance of H^+ monkeys on Repeat-following-error trials does not reflect a tendency to commit the $A\bar{B}$ error. Their low score on Repeat-following-error trials seems to reflect poor performance in general and not a tendency to make errors, especially on trials that followed errors on Reversal trials.

The usefulness of this analysis is supported by findings from a recent study of monkeys with prefrontal lesions (Diamond & Goldman-Rakic, 1989). At 2–5 s, the prefrontal monkeys showed the $A\bar{B}$ error, that is, their performance on Reversal and Repeat-following-error trials (49% and 55%, respectively) was significantly worse than their performance on Repeat-following-correct trials (83%). The prefrontal monkeys erred on 64 Reversal trials. They were then correct on 40% of the Repeat-following-error trials following these reversal errors. They also erred on 21 Repeat-following-correct trials. However, on the Repeat-following-error trials following these errors, they were 72% correct. Thus, when prefrontal monkeys erred on a Reversal trial they tended to repeat that error, but when they erred on a Repeat-following-correct trial they tended *not* to repeat that error. Therefore, in addition to their errors on Reversal trials, their low score on Repeat-following-error trials is further evidence of their tendency to make the $A\bar{B}$ error. By contrast, H^+ monkeys did not tend to err on Reversal trials, and on the few trials when they did, they were no more likely to repeat that error than when they erred on a Repeat-following-correct trial.

Monkeys were tested for 14 sessions at 2-, 5-, and 10-s delays but for 8 sessions at 15- and 30-s delays. If performance ordinarily improved over the test sessions at a given delay, then the overall percent correct score at the longer delays would be misleadingly low and difficult to compare with scores at shorter delays. However, improvement across test sessions did not occur. The H^+ monkeys and control monkeys obtained about the same overall score on the first 7 test sessions (94%, 91%, and 86% for the 2-, 5-, and 10-s delays, respectively) as on the final 7 test sessions (97%, 90%, and 88%; Fs < 1.0). The same was true when the two groups were considered separately. The H^+ monkeys obtained the same score (90%) for both blocks of 7 sessions (averaging across 2-, 5-, and 10-s delays). The unoperated group obtained a score of 91% for the first 7 sessions and 93% for the last 7 sessions ($F < 1.0$).

Object Retrieval

All monkeys completed testing within 3 days (i.e., on the 2nd or 3rd day, they succeeded in retrieving the reward within 3 s on all 21 trials). There were no differences between the hippocampal and control groups in terms of the number of trials required to achieve the performance criterion at each of the three box orientations (see Table 4). A two-way ANOVA (Two Groups × Three Orientations) revealed no effect of group and no interaction (Fs < 0.70, ps $> .10$). There was a significant effect of orientation, $F(2, 6) = 10$, $p < .01$, indicating that trials in which the box opening faced the monkey were easier than trials in which the box opening faced to the left or right for all monkeys. Trials in which the reward was placed deep inside the box were more difficult for both groups than trials in which the reward was placed closer to the opening of the box. The position of the box along the track did not affect performance.

Delayed Nonmatching to Sample

This task was first administered prior to $A\bar{B}$. Monkeys with lesions of the hippocampal formation learned the basic task with a delay of 8 s somewhat more slowly than did control monkeys (540 trials vs. 213 trials to criterion), $t(4) = 1.9$, $p > .10$. Having learned the basic task, they were then impaired when the delays were extended to 15 s, 60 s, and 10 min (see the left side of Figure 3). A two-way ANOVA revealed a marginally significant effect of group, $F(1, 4) = 5.4$, $p < .08$, and significant effects of delay, $F(2, 8) = 24.3$, $p < .001$, and a Group × Delay interaction, $F(2, 8) = 4.1$, $p < .05$. At a delay of 10 min, the H^+ group obtained an average score of 64%, significantly above chance, $t(2) = 6.1$, $p < .02$, but significantly lower than the average score for the control group (77%), $t(4) = 3.7$, $p < .05$.

The same task was administered again following completion of object retrieval testing. Both the control and the hippocampal groups relearned the basic task rapidly, and the performance of the H^+ group was not impaired (mean trials to learning criterion: C = 0, H^+ = 7), $t(4) = 1.0$, $p > .10$. As the delay was increased from 8 s to 10 min, the performance of the H^+ group was again impaired (see the right side of Figure 3). A two-way ANOVA revealed a marginally significant effect of group, $F(1, 4) = 6.1$, $p = .06$, delay, $F(2, 8) = 11.3$, $p < .001$, and a significant Group × Delay interaction, $F(2, 8) = 4.8$, $p < .05$. At a delay of 10 min, the H^+ group obtained an average score of 63%, significantly above chance, $t(2) = 4.3$, $p < .05$, but significantly lower than the average score

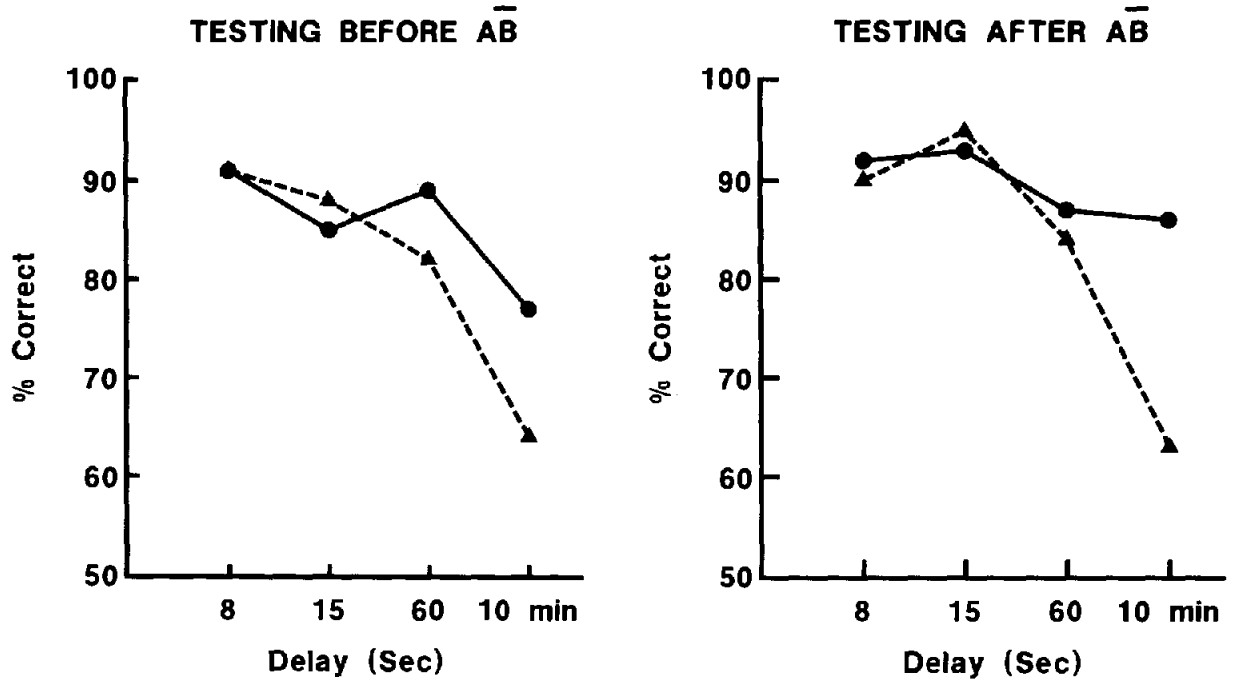

Figure 3. Performance on the delayed nonmatching to sample task by control monkeys (N = 3, circles), and monkeys with hippocampal formation lesions (N = 3, triangles). (The two tests were separated by an interval of 40 months. AB̄ = "A not B" test.)

for the control group (86%), $t(4) = 5.9$, $p < .01$. The H+ group was impaired to a similar extent on both administrations of the task ($p > .10$).

Discussion

The purpose of the present study was to determine whether performance on two tasks that mark developmental changes in infants—the AB̄ and object retrieval tasks—is dependent on the integrity of the hippocampus. The main finding was that monkeys with bilateral lesions of the hippocampus performed well on both tasks.

Comparison of H+ Monkeys and Monkeys With Dorsolateral Prefrontal Cortex Lesions on the AB̄ Task

On AB̄, monkeys with hippocampal formation lesions performed well at delays of 2 and 5 s, the delays at which human infants of 7½–9 months (Diamond, 1985) and infant monkeys of 1½–2½ months (Diamond & Goldman-Rakic, 1986) make the AB̄ error. In contrast to the performance of H+ monkeys, monkeys with dorsolateral prefrontal cortex lesions make the AB̄ error at these delays (adult prefrontal monkeys: Diamond & Goldman-Rakic, 1989; infant monkeys operated at 4 months, tested at 5 months:[1] Diamond & Goldman-Rakic, 1986). They required an average of 19.0 trials per session (compared with 13.6 trials for monkeys with hippocampal formation lesions), $t(4) = 12.6$, $p < .001$, and they performed at a level of 66% correct overall (H+ = 95%), $t(4) = 32.1$, $p < .001$. Indeed, prefrontal monkeys (Diamond & Goldman-Rakic, 1989) performed nearly as well as the H+ monkeys on Repeat-following-correct trials (83% correct vs. 95% correct), but prefrontal monkeys performed much worse than H+ monkeys on Reversal trials and Repeat-following-error trials (roughly 50% on both vs. roughly 95% on both). Poor performance on Reversal trials in the face of good performance on Repeat-following-correct trials is the hallmark of the AB̄ error.

H+ monkeys also performed well on AB̄ at delays of 10 and 15 s. In contrast, monkeys with lesions of prefrontal cortex were severely impaired at a 10-s delay (Diamond & Goldman-Rakic, 1989). Indeed, their performance was so poor at the 10-s delay that testing was not continued to the 15-s delay.

At delays of 30 s, the performance of H+ monkeys declined to a level comparable to that shown by prefrontal monkeys (Diamond & Goldman-Rakic, 1989) when the prefrontal monkeys showed the AB̄ error (delays of 2 or 5 s) (based on percent correct over all trials: H+ = 64%, prefrontal = 65%). However, even when performance was at this level, monkeys with H+ lesions did not show the AB̄ error pattern. At delays of 2–5 s, prefrontal monkeys performed well on Repeat-following-correct trials (83%), but they performed poorly on Reversal trials (49% correct), and they tended to repeat their errors on the trials immediately following the Reversal trials (i.e., they committed the AB̄ error). In contrast, the performance of H+ monkeys on Reversal trials, even at the 30-s delay, was not significantly different from their performance

[1] By 4 months of age, infant monkeys succeed on AB̄ at delays as long as 12 s. Lesions of dorsolateral prefrontal cortex at this age produce the AB̄ error at delays of 2–5 s as these lesions do in the adult.

on Repeat-following-correct trials (63% vs. 75%); that is, they did not show the AB̄ error. Moreover, the H+ monkeys performed better on Reversal trials at the 30-s delay than did the prefrontal monkeys at delays of 2–5 s (63% vs. 49%), $t(4) = 2.9$, $p < .05$.

One should consider the possibility that monkeys with hippocampal formation lesions might have shown the AB̄ error pattern at a delay between 15 s and 30 s. The window for the AB̄ error is small. At brief delays, performance is perfect; at long delays, performance is equally poor across all types of trials (cf. performance of 7½- to 9-month-old infants and prefrontal monkeys at 10-s delay [Diamond, 1985; Diamond & Goldman-Rakic, 1986, 1989]). At delays of 15 s, performance of H+ monkeys was not perfect, and it had begun to decline. At delays of 30 s, monkeys with hippocampal formation lesions did show some signs of deteriorated performance. They were inclined to perseverate after erring and showed signs of distress and refusal to continue testing. Nevertheless, their performance was not so deteriorated at the 30-s delay as to be at chance levels, nor was their performance equivalent across trial types.

The important point is that the overall score of the H+ monkeys at the 30-s delay matched the overall score obtained by prefrontal monkeys (Diamond & Goldman-Rakic, 1989) at the 2–5 s delay. Although it is difficult to compare results across two different studies, different patterns of performance were observed in these two groups. Despite the fact that they were matched with respect to overall score and that performance was impaired in both groups, monkeys with lesions of dorsolateral prefrontal cortex showed the AB̄ error, and monkeys with lesions of the hippocampal formation did not. If AB̄ errors were a consequence of weak memory, AB̄ errors should have been observed after hippocampal damage.

Comparison of H+ Monkeys and Monkeys With Dorsolateral Prefrontal Cortex Lesions on the Object Retrieval Task

Monkeys with hippocampal lesions also performed well on the object retrieval task, succeeding on all trials by the 3rd day of testing. On the other hand, monkeys with bilateral prefrontal cortex lesions tested in a similar way (Diamond & Goldman-Rakic, 1986) required 4–5 days to complete object retrieval testing, significantly longer than the time required by monkeys with hippocampal lesions, $t(4) > 4.95$, $p < .05$.

Human infants of 7½–9 months, infant monkeys of 1½–2½ months, and monkeys with lesions of dorsolateral prefrontal cortex fail the object retrieval task (human infants: Diamond, 1988c; infant monkeys: Diamond & Goldman-Rakic, 1986; prefrontally operated monkeys: Diamond & Goldman-Rakic, 1985). Human infants of 7½–8 months are unable to solve the left- and right-open trials when the bait is deep in the box, because they fail whenever their line of sight to the bait is through a closed side of the box. Human infants of 8½–9 months solve left- and right-open trials by leaning over to look in the opening and reaching in awkwardly with the contralateral hand. Infant monkeys of 1½–2½ months and monkeys with dorsolateral prefrontal lesions show these same kinds of errors (Diamond & Goldman-Rakic, 1985, 1986). Monkeys with hippocampal formation lesions performed normally at all orientations of the box opening and never showed the awkward reach with the hand contralateral to the box opening.

It should be noted that the monkeys who received lesions of dorsolateral prefrontal cortex (Diamond & Goldman-Rakic, 1985, 1989) were rhesus (*Macaca mulatta*), whereas the monkeys who received lesions of the hippocampal formation were cynomolgus (*Macaca fascicularis*). However, it is unlikely that the superior performance of the H+ monkeys on AB̄ and object retrieval can be attributed to this species difference. The unoperated cynomolgus monkeys performed slightly but not significantly worse than the unoperated rhesus monkeys on both AB̄ and object retrieval (rhesus on AB̄: Diamond & Goldman-Rakic, 1989; rhesus on object retrieval: Diamond & Goldman-Rakic, 1985). Thus, if anything, a species difference would have predisposed the cynomolgus monkeys with hippocampal formation lesions to perform worse than rhesus monkeys with prefrontal lesions. Just the reverse was observed: The cynomolgus monkeys with hippocampal formation lesions performed better.

H+ monkeys were tested on AB̄ and object retrieval at different intervals after surgery from those used for monkeys with prefrontal lesions (Diamond & Goldman-Rakic, 1985, 1989), but this difference probably was not important. Prefrontal monkeys were tested on AB̄ and then object retrieval beginning either at 3 weeks or at 2 years after surgery. H+ monkeys were tested on these tasks at approximately 2½ years after surgery. The prefrontal monkeys tested 2 years after surgery were at least as impaired as those tested soon after surgery. Similarly, the memory impairment of the H+ monkeys remained severe 3 years after surgery: Their performance on delayed nonmatching to sample 3 years after surgery continued to show a significant deficit, as had their performance shortly after surgery.

In summary, two tasks on which human infants show improved performance between 7½–12 months (AB̄ and object retrieval) are sensitive to dorsolateral prefrontal cortex damage but not to hippocampal damage. This finding suggests that these developmental changes in the performance of human infants may be linked to the maturation of prefrontal cortex. Memory impairment alone is not sufficient to cause errors on these tasks.

Studies of AB̄ and Related Tasks in Brain-Injured Humans

Recent neuropsychological studies of brain-injured patients who have been tested on tasks similar to AB̄ (Freedman & Oscar-Berman, 1986; Schacter, Moscovitch, Tulving, McLachlan, & Freedman, 1986) have yielded results consistent with the conclusions drawn from our research. Freedman and Oscar-Berman used the delayed response task with delays of 0[2], 10, 30, and 60 s, summing the results over all

[2] After the covering of the wells, a curtain was quickly lowered and raised between the wells and the subject. Thus, the 0-s delay was probably at least 1–2 s long.

delays. (For a discussion of the similarities between delayed response and AB̄, see Diamond, in press; Diamond & Doar, 1989). Patients with bilateral frontal lobe damage, which included dorsolateral prefrontal cortex in some cases, failed delayed response, whereas amnesic patients (some of whom were reported to have signs of frontal lobe dysfunction) and alcoholic control subjects performed well.

Schacter et al. (1986) also tested amnesic patients with signs of frontal lobe dysfunction. An object was either hidden in a room rich in objects and landmarks (room search) or in one of four drawers (container search). The delay for both tasks was 150 s (2½ min). The amnesic patients correctly retrieved the object from the first hiding place (Location A), but when the object was hidden at a second location (B) they continued to search at A (similar to the AB̄ error). Unlike human infants, however, these patients were as likely to err when the object was uncovered as when it was covered. Human infants make very few errors when there are no covers (Butterworth, 1977). Patients with damage to medial frontal cortex succeeded on these tasks and perseverated less on the Wisconsin Card Sorting Test than did the amnesic patients. The good performance of these medial frontal patients is consistent with the finding that perseverative errors on the Wisconsin Card Sorting Test are associated with damage to dorsolateral prefrontal cortex (Drewe, 1974; Milner, 1963).

The good performance of the amnesic patients in the Freedman and Oscar-Berman (1986) study and the poor performance of the amnesic patients in the Schacter et al. study (1986) might have been due to the difference in length of delay. The 150-s delay used by Schacter and colleagues might have taxed the memory of the amnesic patients more than the shorter delays (0–60 s) used by Freedman and Oscar-Berman. A second possibility is that the amnesic patients tested by Schacter et al. might have had more severe frontal lobe dysfunction than the amnesic patients studied by Freedman and Oscar-Berman.

Taken together, the results of those two studies are consistent with the conclusion from the present study that damage to dorsolateral prefrontal cortex alone is sufficient to produce the AB̄ error and that memory impairment alone does not produce the AB̄ error. First, patients with dorsolateral prefrontal cortex damage failed the delayed response task (which resembles AB̄) even at relatively short delays (Freedman & Oscar-Berman, 1986). Second, the amnesic patients who failed tasks similar to AB̄ had signs of frontal lobe dysfunction, as indicated by their poor performance on the Wisconsin Card Sorting Test task (Schacter et al., 1986). Third, patients with *medial* frontal cortex damage succeeded on AB̄-like tasks (Schacter et al., 1986).

Abilities Required for Successful AB̄ and Object Retrieval Performance

Work on the AB̄ task and tasks similar to AB̄ sharpens the contrast between the contributions to memory performance of dorsolateral prefrontal cortex and the hippocampus. Monkeys with hippocampal formation lesions performed well on the AB̄ task at short delays and never showed the AB̄ error. They did have a memory impairment, however. Specifically, they were impaired on the delayed nonmatching to sample task as the delay was increased from 8 s to 10 min both before and after testing on AB̄ and object retrieval. They were also impaired on AB̄ as the delay increased from 5 to 30 s. Despite their memory impairment, however, they did not show the AB̄ error or have difficulty with object retrieval. Thus, the AB̄ error cannot be due to impairment in the type of memory function subserved by the hippocampus.

The excellent performance of monkeys with hippocampal formation lesions at short delays is consistent with extensive findings of good performance by H^+ monkeys at delays of 10 s or fewer, even on tasks particularly sensitive to damage of the hippocampus (Diamond, 1988a; Squire & Zola-Morgan, 1983; Zola-Morgan & Squire, 1986; Zola-Morgan et al., 1989). Amnesic patients, including patients with known hippocampal damage, similarly perform well at short delays provided that the material to be retained does not exceed short-term memory capacity (Squire, 1987; Zola-Morgan et al., 1986).

By contrast, monkeys with prefrontal cortex lesions perform poorly even at very short delays (e.g., 2 s), and they make the AB̄ error at those delays (Diamond & Goldman-Rakic, 1986, 1989). The hippocampus appears to be required in order for information to be available beyond short-term memory, whereas prefrontal cortex is needed to use information effectively while it is within short-term memory (e.g., it is needed to keep information on-line for current use).

One of the abilities required for AB̄ is the ability to relate hiding to retrieval over a brief temporal separation. All subjects succeed when there is no separation (i.e., no delay). Young infants and prefrontal monkeys fail even when a small separation is introduced (e.g., 2 s). Object retrieval requires a similar ability to relate information over a separation: The reward must be related to the box opening over a small spatial separation. When the reward is in the opening, all subjects succeed. Young infants and monkeys with prefrontal cortex lesions fail as the separation between the reward and box opening increases. This problem occurs particularly when the box opening faces left or right.

Both AB̄ and object retrieval appear to require a second ability in addition to spanning a temporal or spatial separation (i.e., the ability to inhibit a dominant response tendency). If memory were the only ability required for success on AB̄, errors should appear equally on all types of trials. The AB̄ error, however, consists of good performance when the reward is hidden where the subject just reached correctly (Repeat-following-correct trials) and repeated errors when the side of hiding is reversed (Reversal and Repeat-following-error trials), even though the delay is held constant across all trials. When the side of hiding changes, the AB̄ task sets up a conflict between a subject's memory of where the reward has just been hidden and a subject's tendency to repeat a rewarded response. To succeed on AB̄, a subject must remember where the reward has been hidden *and* inhibit the response tendency to reach back to Location A. Object retrieval also requires inhibition of a dominant response. Here, the dominant response is reaching directly toward a visible goal rather than making a detour. The tasks most sensitive to damage of prefrontal cortex are those that require both inhibition and integration of information over a temporal or

spatial separation. Maturation of dorsolateral prefrontal cortex may underlie improvement in these abilities during the 1st year of life. In contrast, tasks that require long-term memory are the most sensitive to hippocampal damage. The ability to retain information across very short delays and the ability to inhibit dominant responses do not require the integrity of the hippocampus.

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