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The Development and Neural Bases of Memory Functions as Indexed by the $A\bar{B}$ and Delayed Response Tasks in Human Infants and Infant Monkeys[a]

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One of the classic markers of developmental change in infants between 7½–12 months of age is improved performance on "the $A\bar{B}$ task" (pronounced "A not B"). This task was first devised by Piaget (1954 [1937]) and has been used by researchers throughout the world to study psychological development in babies (for reviews see Gratch, 1975; Schuberth, 1982; Harris, 1986; Wellman, Cross & Bartsch, 1987). One of the most useful tasks in the study of brain–behavior relations is a task called "delayed response." It was first introduced for this purpose by Jacobsen (1935; 1936) and has been widely used ever since to study brain function in monkeys and other animals (for reviews see Nauta, 1971; Rosvold, 1972; Rosenkilde, 1979; Fuster, 1980). Delayed response has been particularly useful in this regard because success on the task has been systematically linked to proper functioning of a discrete neural circuit that comprises dorsolateral prefrontal cortex and the structures with which it is interconnected.

As it turns out, the $A\bar{B}$ and delayed response tasks are very similar, even though they were developed independently and for almost 50 years scientists working with one task did not know of the work of scientists with the other. Indeed, it has recently been established that infants show the same developmental progression on delayed response as they show on $A\bar{B}$ (Diamond & Doar, 1989), and success on $A\bar{B}$ depends on functioning of the same neural circuit as does success on delayed response (Diamond & Goldman-Rakic, 1989).

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The purposes of this paper are: One, to review the evidence that what is being indexed by improved performance with age on A$\overline{B}$ and delayed response is, at least in part, an improvement in *memory*. Two, to review the evidence that at least part of the reason why functioning of dorsolateral prefrontal cortex is required for success on the delayed response and A$\overline{B}$ tasks is because of the memory requirements of the tasks. Three, to consider the nature of the memory ability required by these tasks. That is, what is the nature of the memory ability that appears to be maturing during the latter months of the first year of life and that appears to depend on dorsolateral prefrontal cortex? The fourth and final purpose of the present paper is to compare and contrast the characteristics of the memory ability required for success on A$\overline{B}$ and delayed response with the characteristics of the memory abilities required for other tasks used with young children or in the study of brain–behavior relations.

Memory is *not* the only ability required for success on the A$\overline{B}$ or delayed response tasks. These tasks require *both* inhibitory control and memory (Diamond, 1985; 1988a; 1988b; in press). In this paper, however, only the contribution of memory will be considered. The role of inhibitory control in performance of these and other tasks is discussed in Diamond (this volume, a).

TESTING PROCEDURE FOR THE A$\overline{B}$ AND DELAYED RESPONSE TASKS

For either A$\overline{B}$ or delayed response testing the subject is centered between two identical hiding wells, one to the left and one to the right. The experimenter holds up an object of keen interest to the subject, and the subject watches the experimenter place this object in one of the two hiding wells. Care is taken to make sure that the subject has seen where the object was placed. The experimenter covers both hiding wells simultaneously with identical covers and a brief delay of 0–10 sec is imposed during which the subject is prevented from looking at, or moving or straining toward, the correct well. Then the subject is allowed to reach. When a subject reaches incorrectly to the empty well, the experimenter shows the subject that the reward had been hidden in the other well, but removes the reward without allowing the subject to have it. This procedure is used for both A$\overline{B}$ and delayed response. In these details the two tasks are identical.

In A$\overline{B}$, the reward is consistently hidden in the same well until the subject is correct to a specified criterion (typically, 2 correct responses in a row), then the reward is hidden in the other well and the procedure repeated.[b] In delayed response, where the reward is hidden is varied randomly by a predetermined schedule. Thus, the A$\overline{B}$ and delayed response tasks differ in the rule for deciding where the reward is to be hidden, but once one has decided where to hide the reward, the procedure on any trial is the same in both tasks.

[b]Infants make a characteristic error on the A$\overline{B}$ task, from which the task derives its name. They typically reach correctly at the first place the reward is hidden (A), but when the reward is then hidden at well B, infants still search at well A, although they just saw the reward hidden at B moments earlier. That is, they are correct when the reward is hidden at A but not when it is hidden at B; they reach to A but not to B. Hence, the name of the task "A-not-B."

EVIDENCE THAT IMPROVED PERFORMANCE WITH AGE ON THE A$\overline{B}$ AND DELAYED RESPONSE TASKS REVEALS A DEVELOPMENTAL PROGRESSION IN MEMORY

The principal change in performance of A$\overline{B}$ and delayed response over age is in the delay that can be tolerated. As human infants or infant monkeys grow older they are able to tolerate longer and longer delays on these tasks.

A$\overline{B}$: Human Infants

For example, we tested 25 human infants biweekly from 5 or 6 months to 12 months of age. Testing on A$\overline{B}$ began in the session when the infant could first reach for a hidden object (Diamond, 1985). We found that infants succeeded at longer and longer delays as they grew older. The mean delay at which the A$\overline{B}$ error occurred increased gradually and continuously at the rate of approximately 2 sec per month (see FIG. 1). The A$\overline{B}$ error was found in all children and persisted for many months, as long as the delay was incremented. Individual differences in the shape of the growth function and between children of the same age in the delay needed to produce the A$\overline{B}$ error were quite large, however.

We also tested another 84 infants only once on A$\overline{B}$ between the ages of 6–12 months (12 infants each at 6, 7, 8, 9, 10, 11, and 12 months; see TABLE 1). A similar developmental progression in the length of delay that could be tolerated at each age could be seen in the performance of these infants. At 9–10 months they performed well when a 2-sec delay was imposed, by 10–11 months they could tolerate delays of 5 or 8 sec, and by 11–12 months their performance was excellent even with a 10-sec delay. They could not tolerate delays quite as long as the infants tested longitudinally, however. The infants tested only once made the A$\overline{B}$ error at delays about 1½ to 2 sec shorter on average than did the infants tested every 2 weeks, although by 12 months of age this difference had largely disappeared (see TABLE 2).

When no delay was used or when infants managed to strain or look toward the correct well throughout the delay, they typically reached correctly. This was true for the infants tested longitudinally and for the infants tested only once. If allowed to circumvent the memory demands of the task, they reached correctly. Errors on the A$\overline{B}$ task did not disappear by 12 months of age, but delays longer than 10 sec were typically needed before babies of this age made errors.

To more systematically look at the effect of delay on performance, all 25 infants tested longitudinally also received one test session where half the trials were at a delay predicted to produce the A$\overline{B}$ error, and half were at a delay 2–3 sec shorter. Order of delay presentation was counterbalanced across subjects and within sex and age groups. Ten of these infants also received a second testing session where the order of delay presentation was reversed. The other 15 infants received one test session where half the trials were at the delay predicted to produce the A$\overline{B}$ error, and half were at a delay 2–3 sec *longer*.

We found that within the same session, infants who were making the A$\overline{B}$ error performed correctly when the delay was reduced only 2–3 sec and reached randomly or severely perseveratively when the delay was increased only 2–3 sec (Diamond, 1985). There was no effect of order of delay presentation. Here one can see the

marked sensitivity of infants' performance to even small changes in delay. Infants reached correctly at delays just 2–3 sec shorter than the delays at which they showed the A$\overline{B}$ error, and their performance was severely disrupted by delays just 2–3 sec longer than the delays at which they showed the A$\overline{B}$ error.

Results consistent with this have been found by others. For instance, other longitudinal studies of A$\overline{B}$ performance have found that 8-month-old infants make

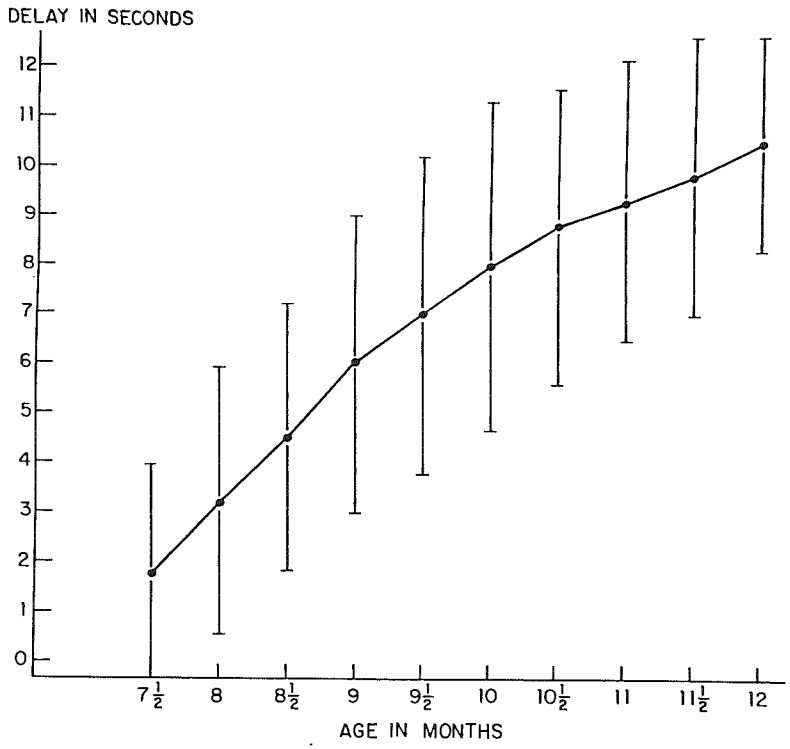

FIGURE 1. Developmental progression in the delay at which human infants tested longitudinally every 2 weeks make the A$\overline{B}$ error on the A$\overline{B}$ task (from Diamond, 1985).

The A$\overline{B}$ error is characterized by a pattern of behavior in which errors occur on reversals and over the next few consecutive trials at the new hiding location, in the face of otherwise excellent performance. If errors occur with equal frequency over all trials, the subject is reaching randomly, and not committing the A$\overline{B}$ error.

the A$\overline{B}$ error at delays of 3 sec, although they perform well if there is no delay (Gratch & Landers, 1971; Fox, Kagan & Weiskopf, 1979). Fox *et al.* also found that by 9 months infants succeeded with delays of 3 sec, but failed with delays of 7 sec. Cross-sectional studies have yielded a similar progression in the delays infants of different ages can tolerate, although here, as in our own work, infants tested only once have been found to make the A$\overline{B}$ error at delays a few seconds briefer than do

TABLE 1. Number of Subjects at Each Age Tested at Each Delay

Age in Months	Age Range in Weeks (days)	Delay in Seconds					
		0	2	5	8	10	12
6	26 (0)–30 (0)	2					
7	30 (2)–34 (0)	4	4				
8	34 (6)–38 (4)	4	4	4			
9	40 (2)–43 (6)		4	4	4		
10	44 (1)–47 (1)		4	4	4		
11	47 (5)–51 (0)			4	4	4	
12	53 (2)–55 (3)				4	4	4

NOTE: Twelve infants were tested with a hidden object at each age, but 10 6-month-olds and 4 7-month-olds could not uncover a hidden object and so were not tested on A$\overline{B}$.

infants tested repeatedly. For example, it is not uncommon for 9- or 10-month-old infants tested for the first time to make the A$\overline{B}$ error at delays of only 5 sec (e.g., Harris, 1973; Bremner, 1978). It is very uncommon, however, for an infant over 8 months, even on first testing, to make the A$\overline{B}$ error if no delay is imposed (Gratch, Appel, Evans, LeCompte & Wright, 1974; Harris, 1973). Similarly, if infants are allowed to circumvent the memory requirements of the task by looking at, or straining toward, the correct well throughout the delay, they perform well regardless of the length of delay used (Cornell, 1979; Fox *et al.*, 1979).

Given that (a) infants usually succeed when there is no delay, (b) even infants who are making the A$\overline{B}$ error, reach correctly *within the same session* if the delay is decreased, and (c) infants perform well if allowed to circumvent the memory requirements of the task by looking at the correct well, positioning themselves in front of the correct well, or straining toward the correct well throughout the delay, it would seem that one of the sources of infants' errors is a failure to remember where the toy has been hidden. When their memory is not taxed, they perform well; when even a small memory demand is imposed by a brief delay, they begin to err.

TABLE 2. Comparison of the Delays at Which the A$\overline{B}$ Error Occurred in Infants Tested Only Once and Infants Tested Every Two Weeks

Age in Months	Average Delay for the A$\overline{B}$ Error, Cross-Sectional Sample	Average Delay for the A$\overline{B}$ Error, Longitudinal Sample
7	1.25	1.52
8	2.67	3.86
9	4.83	6.56
10	6.0	8.48
11	8.33	9.58
12	10.33	10.55

Further support for this comes from studies where the reward is always visible in the hiding well, eliminating the need to remember where the reward has been placed. Thus, infants perform beautifully when covers are not placed over the wells or when transparent covers are used (e.g., Butterworth, 1977). Although infants make some errors with transparent covers, the errors are fewer here (e.g., Butterworth, 1977), and largely disappear if the infants are given time to familiarize themselves with the covers (Yates & Bremner, 1988). In short, any procedure that reduces the memory requirements of the task yields superior performance.

Moreover, with increasing age, infants perform well at increasingly long delays. This suggests that their ability to remember is improving over these months. All the evidence indicates that the improvement is gradual and linear, at a constant rate of about 2 sec per month.

A$\bar{B}$ with Multiple Wells: Human Infants

For a time the role of memory in A$\bar{B}$ performance was called into question by findings that infants perform better when 3–7 hiding wells are used (where one would think the memory requirements would be more severe) than they do when only the traditional 2 wells are used (see e.g., Cummings & Bjork, 1983; Wellman, Cross & Bartsch, 1987). It now seems, however, that this apparent discrepancy was due to inadvertent differences in procedure.

When more than 2 wells are used, and the spacing between wells is not reduced, the 2 endpoint wells will necessarily be farther apart than when only 2 wells are used. Most studies using multiple wells have hidden the reward only in the 2 wells at the endpoints. Horobin and Acredolo (1986) demonstrated that performance improves with only 2 wells if those wells are farther apart, comparable to their spatial separation in multiple wells studies. Thus, it would appear that part of the reason infants have performed better with multiple wells is the greater separation between wells A and B in those studies.

A second difference in procedure arises because when 2 wells are used both wells are usually uncovered and then covered simultaneously. When multiple wells are used, however, since we have only 2 hands, the correct well alone is usually uncovered and then re-covered after the toy is hidden there. Here, the last action after the hiding draws the infant's attention to the correct well. When the experimenter covers all the wells simultaneously (as is done with 2 wells), that covering might be considered a distraction, drawing the infant's attention away from where the toy was hidden. Indeed, Harris (1973) showed that performance with 2 wells is significantly better on the all-important reversal trial (the first time the hiding changes from well A to well B) if the correct well is covered last than if both wells are covered simultaneously. Thus, Cruttenden, Neiderman, and I (Diamond *et al.*, 1989) reasoned that the manner in which the wells were covered might have made the task with multiple wells easier for infants. We tested this using 3 conditions of hiding with a 7-well apparatus.

The wells were arranged in a semicircle to offset the tendency of infants to reach along the midline. In condition 1, all wells were uncovered, the toy hidden, and then a tray that sat between the tabletop and the wells was pushed in place simultaneously

covering all wells. With the tray in place, each well was covered by a cloth with a slit cut into it; infants could retrieve the toy by reaching through a slit (see FIG. 2). In condition 2, the tray was in place throughout testing. The toy was hidden by lowering it through the slit into the well. Hence, the last action before the delay (lowering the toy) was at the correct well. Condition 3 replicated the standard procedure with multiple wells: Cloth covers were used and only the correct well was uncovered and

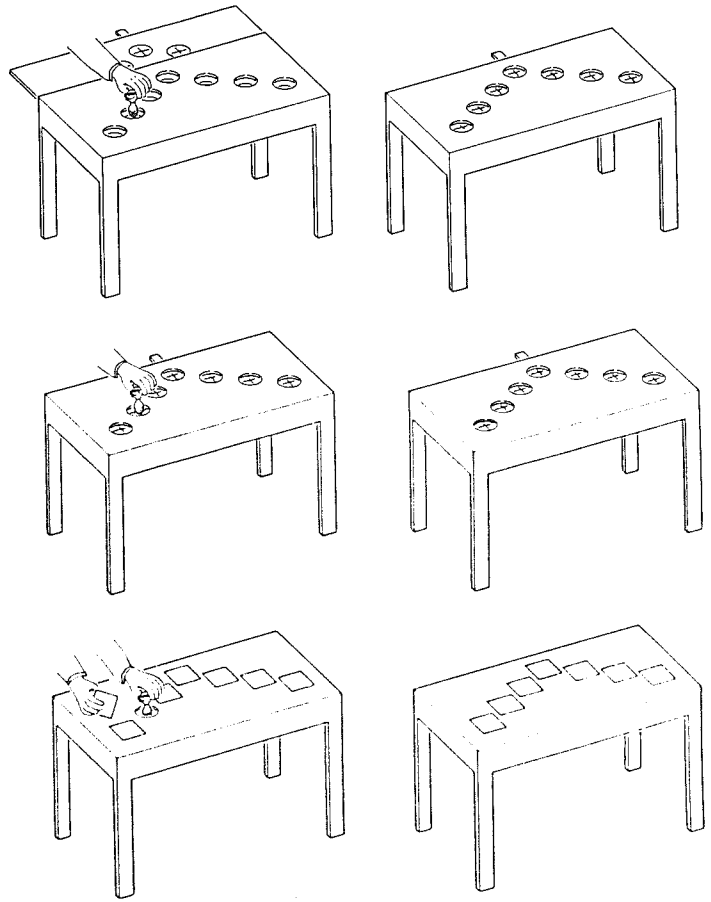

FIGURE 2. The 3 conditions used for multiple well testing by Diamond, Cruttenden, and Neiderman (1989).

Top row: After the toy is placed in a well, the tray containing the slits is slid over the wells, covering all the wells simultaneously (condition 1). *Middle row:* The toy is hidden by lowering it through a slit into a well (condition 2). The slits are in place the whole time. The last action before the delay (lowering the toy) occurs at the correct well. *Bottom row:* One well is uncovered, the toy is placed in that well and then that well is re-covered (condition 3). This is the procedure by which multiple wells testing has typically been conducted. The last action before the delay (covering the well) occurs at the correct well.

then re-covered on each trial. Here again, the last action before the delay was at the correct well (covering the well). Testing in all conditions was conducted with 9-month-old infants using a delay of 5 sec. Well B was 2 wells from the endpoint and 2 wells away from well A. Half the infants in each condition were tested with the hiding first on the right, and half with the hiding first on the left.

Infants performed well when the last action before the delay drew their attention to the well where the toy was hidden (conditions 2 and 3), but performed significantly worse when the wells were covered simultaneously (condition 1). This was true for all dependent measures (see TABLE 3). Performance in conditions 2 and 3 was comparable to that observed by others with multiple well testing. When all wells were covered simultaneously, however (condition 1), the infants' performance was severely impaired and fell below that typically found when only 2 wells are used. Previous reports that infants perform better with multiple wells than with only 2 wells can apparently be accounted for, in large measure, by the order in which the hiding places were covered: uncovering and re-covering of only the correct well with multiple wells, simultaneous covering with 2 wells. Infants do not perform better with multiple wells when the order of covering the wells is the same in the 2-well and multiple-well conditions.

Delayed Response: Human Infants

We tested 12 infants every 2 weeks from 6–12 months of age (Diamond & Doar, 1989). Delayed-response testing began as soon as an infant could uncover a hidden object. To control for the effect of repeated testing, another 36 infants were tested only once (12 each at 8, 10, and 12 months of age). The testing procedure within a trial was exactly the same as that for A$\overline{B}$, where the toy was hidden was determined by a pseudo-random Gellerman series. Initial side of hiding was counterbalanced across children for the first testing session and counterbalanced across testing sessions for each child tested longitudinally.

We found that infants improved in performance on delayed response over the same ages and at the same constant rate (approximately 2 sec per month) as is found for A$\overline{B}$ (see FIG. 3), despite the fact that delayed response and A$\overline{B}$ were administered in different laboratories by different testers with infants from different parts of the country. The mean difference in the delays that could be tolerated on delayed response and A$\overline{B}$ at each age was only 0.3 sec (see TABLE 4). The performance of infants tested only once on delayed response lagged somewhat behind the performance of infants of the same age tested longitudinally, as had been found on A$\overline{B}$.

If the memory requirements of the task were reduced (by shorter delays or by uninterrupted orientation toward the correct well) infants succeeded on delayed response, just as they do on A$\overline{B}$. For example, the performance of infants tested cross-sectionally was significantly better at each age at the briefer delay than at the longer delay (see TABLE 5). Moreover, older infants performed significantly better at each delay than did younger infants.

These results are also corroborated by the work of others. Harris (1973) found that infants of 9½–10 months performed significantly better on delayed response when the delay was 0 sec than when it was 5 sec. Brody (1981) found that infants of 8

TABLE 3

Condition	Percent Correct				First Reversal Trial	Mean Number of Reaches until Two Correct in a Row		Mean Number of Consecutive Errors After First Reversal
	All Trials	All Reversal Trials	All Repeat Following Correct Trials	All Repeat Following Error Trials		At First Well	After First Reversal	
(1) Slits: All wells covered simultaneously	32	17	34	25	18	5.9	7.2	4.9
(2) Slits: Attention drawn to correct well	55	47	59	50	55	5.2	4.2	2.7
(3) Covers: Attention drawn to correct well	60	50	60	60	53	4.0	4.0	1.5
Linear Contrasts[a]:								
(1) versus (2)	24.92[b]	14.75[c]	23.03[b]	18.95[b]	2.99[d]	1.39, ns	3.67[c]	4.95, $p < 0.03$
(1) versus (3)	39.42[b]	17.59[b]	25.90[b]	35.26[b]	2.81[d]	3.54[c]	3.54[c]	14.56[c]
(2) versus (3)	1.45, ns	0.11, ns	0.07, ns	2.36, ns	0.14, ns	0.46, ns	0.25, ns	1.81, ns

[a]Linear contrasts are used rather than less stringent *t*-tests, to take into account that multiple comparisons are being tested.
[b]$p < 0.0001$
[c]$p < 0.001$
[d]$p < 0.01$
[e]$p = 0.06$

months succeeded on delayed response with a 0.25-sec delay, but failed with a 3-sec delay. By 12 months, they succeeded with delays even 9 sec long.

In short, infants' performance on delayed response seems exquisitely sensitive to the memory requirements of the task, as is also true for their performance on A$\bar{B}$. Anything that reduces the memory requirements (such as a briefer delay or continued fixation on the correct well throughout the delay) serves to improve performance. Moreover, from the time when they can first be tested on delayed response (approximately 7½ months) until 12 months of age (when testing stopped), infants show a clear developmental progression in the delays they can tolerate on the task, as is also found on A$\bar{B}$.

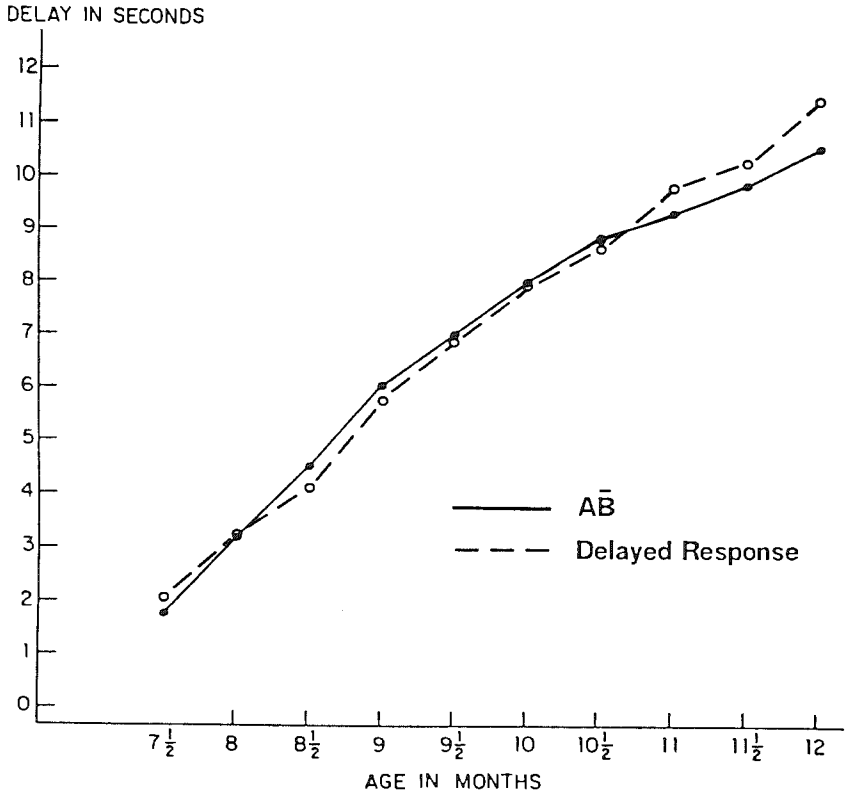

FIGURE 3. Developmental progression in the delay human infants can tolerate on delayed response and A$\bar{B}$.

A$\bar{B}$ results are usually reported in terms of the age at which the A$\bar{B}$ *error* occurs. In an attempt to use a comparable measure for the delayed response task, results are plotted here in terms of the delay at which errors occurred (i.e., the delay at which performance was below the criterion of 88% correct). The A$\bar{B}$ results are shown by the solid line and are the same as those shown in FIGURE 1. The delayed response results are shown by the dashed line and are based on the infants studied longitudinally by Diamond and Doar (1989).

TABLE 4. Mean Delay for the A$\bar{B}$ Error and for Less Than 88% Correct on Delayed Response for Infants Studied Longitudinally[a]

Age in Months	Delayed Response (N = 12)	A$\bar{B}$ (N = 25)	Difference between DR & A$\bar{B}$ Means
7½	2.1	1.7	0.4
8	3.2	3.2	0
8½	4.0	4.5	−0.5
9	5.9	6.1	−0.2
9½	6.9	7.0	−0.1
10	8.0	8.0	0
10½	8.8	8.9	−0.1
11	9.9	9.3	−0.6
11½	10.3	9.8	0.5
12	11.6	10.6	1.0

[a]A$\bar{B}$ results are usually reported in terms of the age at which the A$\bar{B}$ *error* occurs. In an attempt to use a comparable measure for delayed response, results are reported here in terms of the age at which errors occurred (i.e., the delay at which performance was below the criterion of 88% correct).

A$\bar{B}$: Infant Monkeys

We tested 4 infant rhesus monkeys on A$\bar{B}$ in an infant Wisconsin General Testing Apparatus (WGTA) every day (5 days a week) from the age when they could first uncover a hidden object until they passed A$\bar{B}$ with a delay of 12 sec (Diamond & Goldman-Rakic, 1986). The testing procedure was the same as that used for human infants except (a) a small piece of food was hidden rather than a toy, (b) an opaque screen was lowered during the delay to break visual fixation on the wells[c] (with human infants visual fixation had been broken by the experimenter calling to the infant), and (c) the subjects were not physically restrained from straining or position cuing during the hiding or delay as human infants had been, but were trained not to do so (a trial was interrupted and repeated if a subject tried to reach toward a well or tried to position cue).

Subjects were tested at a given delay until they met the criterion of no more than 1 error in a given session (i.e., at least 92% correct). Delay was then incremented by 1 sec for the next testing session. One monkey was tested daily on A$\bar{B}$ until he succeeded with a delay of 12 sec. The other 3 monkeys were tested daily on A$\bar{B}$ until 80 days of age. From then on they were tested 3 days a week on A$\bar{B}$ and 2 days a week on delayed response until they succeeded with a 12-sec delay.

Infant monkeys, like human infants, showed a clear developmental progression in the length of delay they could tolerate on the A$\bar{B}$ task (see FIG. 4), although infant monkeys could begin testing at a younger age and progressed more quickly to longer delays than human infants. The mean age at which infant monkeys could first be tested on A$\bar{B}$ was 48 days (1.59 months, compared with 7.42 months for the human

[c]Because it takes approximately 2 sec to lower and raise the screen, infant monkeys were not tested at delays of 0 or 1 sec.

TABLE 5. Performance on Delayed Response by Infants Tested Only Once as a Function of Age and Length of Delay

	0-Sec Delay			3-Sec Delay		
8-Month-Olds	All ($N = 6$)	Boys ($N = 3$)	Girls ($N = 3$)	All ($N = 6$)	Boys ($N = 3$)	Girls ($N = 3$)
Percent passing criterion[a]	67	33	100	0	0	0
Percent correct on first reversal trial[b]	67 (6)	67 (3)	67 (3)	33 (3)	50 (2)	0 (1)
Mean percent correct	85	79	90	61	65	56
Difference between mean percent correct at the two delays: $t(10) = 3.25, p < 0.01$						

	3-Sec Delay			8-Sec Delay		
10-Month-Olds	All ($N = 6$)	Boys ($N = 3$)	Girls ($N = 3$)	All ($N = 6$)	Boys ($N = 3$)	Girls ($N = 3$)
Percent passing criterion	83	67	100	17	0	33
Percent correct on first reversal trial	83 (6)	67 (3)	100 (3)	25 (4)	33 (3)	0 (1)
Mean percent correct	86	80	92	63	60	65
Difference between mean percent correct at the two delays: $t(10) = 3.09, p = 0.01$						

	8-Sec Delay			12-Sec Delay		
12-Month-Olds	All ($N = 6$)	Boys ($N = 3$)	Girls ($N = 3$)	All ($N = 6$)	Boys ($N = 3$)	Girls ($N = 3$)
Percent passing criterion	50	33	67	0	0	0
Percent correct on first reversal trial	40 (5)	0 (3)	100 (2)	20 (5)	33 (3)	0 (2)
Mean percent correct	79	77	81	60	66	54
Difference between mean percent correct at the two delays: $t(10) = 3.33, p < 0.01$						

[a]Criterion = 88% correct (correct on 14 out of 16 trials).
[b]This measure is included to compare performance here with the standard dependent measure reported in studies of $A\bar{B}$. Because reversals are only administered in $A\bar{B}$ after a subject has reached correctly, only infants who reached correctly on the trial preceding the first reversal are included here. The number in parentheses gives the number of infants on which the percentage is based.

infants tested longitudinally). The mean age at which the $A\bar{B}$ error occurred at 2 sec was 53.25 days (1.77 months, compared with 7.9 months for human infants), at 5 sec it was 74 days (2.45 months, compared with 8.9 months for human infants), and at 10 sec it was 112.75 days (3.75 months, compared with 11.63 months for human infants). In short, infant monkeys showed a developmental progression between 1½ to 4 months on $A\bar{B}$ comparable to that seen in human infants between 7½ to 12 months. Whereas human infants progressed at the rate of approximately 2 sec per month,

infant monkeys progressed at the rate of about 2 sec per half-month (or 1 sec per week). The $A\bar{B}$ error was seen at delays of 2–5 sec in human infants of 7½ to 9 months and in infant monkeys of 1½ to 2½ months. Excellent performance was seen with delays of 10 sec in human infants of 12 months and infant monkeys of 4 months. When no delay was used or when subjects managed to orient their bodies toward the correct well throughout the delay, they typically reached correctly. That is, if allowed to circumvent the memory demands of the task, infant monkeys, like human infants, reached correctly.

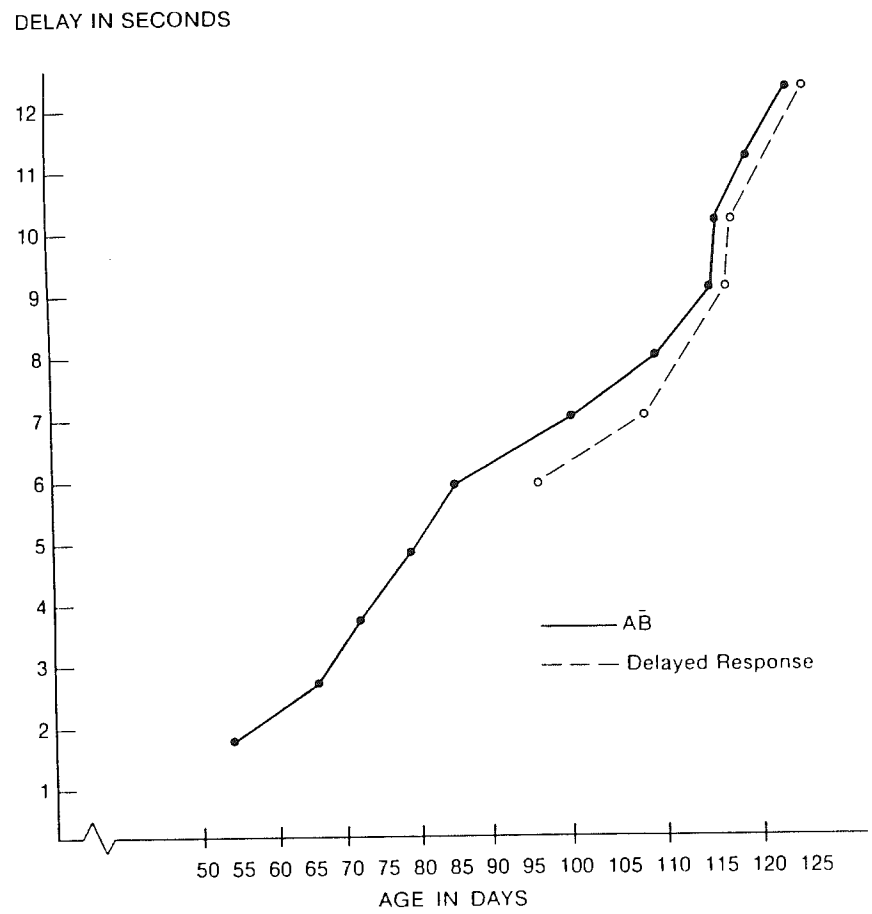

FIGURE 4. Developmental progression in the delay infant monkeys can tolerate on $A\bar{B}$ and delayed response.

These delays represent the levels at which the $A\bar{B}$ error was seen and at which delayed response performance was below the criterion of 88% correct. The $A\bar{B}$ results are shown by the solid line and the delayed response results by the dashed line. Performance on both tasks is based on the same infant monkeys studied longitudinally.

Delayed Response: Infant Monkeys

Three of the four infant rhesus monkeys tested on A$\overline{B}$ were also tested on delayed response in the same infant WGTA beginning at the age of 80 days (Diamond & Goldman-Rakic, 1986). Delayed response testing occurred twice weekly and the testing procedure within a trial was identical to that used for A$\overline{B}$. The testing procedure was the same as that used routinely with adult monkeys to study delayed-response performance. Thirty trials were administered with side of hiding varying randomly over trials.[d]

Delayed response testing began with a delay of 6 sec. By 80 days, 2 of the 3 monkeys were being tested on A$\overline{B}$ with a delay of 7 sec and the third monkey was being tested on A$\overline{B}$ with a delay of 6 sec. They were tested on delayed response at a given delay until they met the criterion of 90% correct (27 out of 30 trials correct). The delays at which they received delayed response testing were: 6, 7, 9, 10, and 12 sec.

As can be seen from FIGURE 4, infant monkeys showed a developmental progression in the delays they could tolerate on delayed response that was comparable to their progression on A$\overline{B}$. They had some difficulty with delayed response when it was first introduced. After a few days, however, their performance on delayed response became progressively more comparable to their performance on A$\overline{B}$ so that by about 110 days and thereafter the delay they could tolerate on one task closely matched the delay they could tolerate on the other. For example, the mean age at which they performed below criterion on delayed response with a delay of 10 sec was 113.8 days (compared with 112.75 days for the mean age of the A$\overline{B}$ error with a delay of 10 sec). They progressed on delayed response at the same rate at which they progressed on A$\overline{B}$ (approximately 1 sec per week).

Conclusions

(1) For infant monkeys, as for human infants, if the memory demands of either the A$\overline{B}$ or delayed response tasks are reduced (e.g., by the subject waiting in front of the correct well throughout the delay or by imposing no delay) performance is excellent. Memory would seem to be one of the abilities required for success on these tasks. (2) Memory seems to be improving during infancy: Human infants and infant monkeys show a clear, continuous developmental progression in the length of delay they can tolerate on A$\overline{B}$ and delayed response with increasing age. Increasing demands must be placed on memory (by ever longer delays) to continue to challenge human infants or infant monkeys on either task. (3) Performance improves on delayed response over the same age period and at the same rate as performance on A$\overline{B}$, for both human infants and infant monkeys. Both tasks would appear to be tapping the same age-related improvements in memory.

[d] Delayed response testing sessions were typically longer than those for A$\overline{B}$, which ranged from 12–30 trials in length.

EVIDENCE THAT DORSOLATERAL PREFRONTAL CORTEX IS REQUIRED FOR THE MEMORY ASPECTS OF THE A$\overline{B}$ AND DELAYED RESPONSE TASKS

Delayed Response: Adult Monkeys

It has been known for some time that success on the delayed response task depends upon involvement of dorsolateral prefrontal cortex. The link between dorsolateral prefrontal cortex and delayed response has been established by an astonishing array of anatomical, physiological, pharmacological, and metabolic techniques. It has been established through the effects of permanent damage (*surgically induced by lesions:* e.g., Butters, Pandya, Sanders & Dye, 1969; Goldman & Rosvold, 1970; *pharmacologically induced by 6-hidroxydopamine injections:* Brozoski, Brown, Rosvold & Goldman, 1979), temporary and reversible inactivation (*localized cooling:* Fuster & Alexander, 1970; Bauer & Fuster, 1976; Alexander & Goldman, 1978; *localized electrical stimulation:* Weiskrantz, Mihailovic & Gross, 1962; Stamm, 1969; Stamm & Rosen, 1969), electrophysiological recording from the intact brain (*surface recording:* Stamm, 1969; Stamm & Rosen, 1969; *single cell recording:* e.g., Fuster & Alexander, 1971; Fuster, 1973; Niki, 1974; Niki & Watanabe, 1976; Funahashi, Bruce & Goldman-Rakic, 1989), and metabolic activity in the intact brain (*2-deoxyglucose metabolic labelling:* Bugbee & Goldman-Rakic, 1981). Damage to dorsolateral prefrontal cortex does not produce deficits on other tasks, such as visual discrimination, and damage to other areas of the brain does not produce deficits on delayed response.

The electrical recording work has established that the critical period within a trial when dorsolateral prefrontal cortex must fire is the delay period. Typically, cells in dorsolateral prefrontal cortex increase firing at the beginning of the delay and maintain that elevated level of activity until it is time to respond. On trials where this increased firing in dorsolateral prefrontal cortex has not occurred during the delay period, subjects tend to reach incorrectly. Evidence from lesion studies also indicates that it is the imposition of a delay that makes involvement of dorsolateral prefrontal cortex critical. If dorsolateral prefrontal cortex is removed, subjects can still succeed on the delayed response task if there is no delay; errors appear only on trials where a delay is imposed (e.g., Harlow *et al.,* 1952; Battig *et al.,* 1960; Fuster & Alexander, 1971; Bachevalier & Mishkin, 1986; Diamond & Goldman-Rakic, 1989).

The devastating effect that lesions of dorsolateral prefrontal cortex have on the ability to span the delay between hiding and retrieval in the delayed response task is illustrated, for example, by our finding that monkeys, who before surgery were succeeding on delayed response with delays as long as 120 sec, failed the task with delays as brief as 2 sec after removal of dorsolateral prefrontal cortex (Diamond & Goldman-Rakic, 1989). In comparison, other monkeys, who likewise had succeeded on delayed response with delays of 120 sec preoperatively, continued to succeed on the task at long delays following removal of parietal cortex.

A$\overline{B}$: Adult Monkeys

Less attention has been directed to the relation of neural activity to performance on the A$\overline{B}$ task, but the results that exist are in full accord wih those for delayed

response. We have studied the performance of rhesus monkeys (Diamond & Goldman-Rakic, 1989) and cynomolgus monkeys (Diamond, Zola-Morgan & Squire, 1989) on the $A\overline{B}$ task. The rhesus monkeys were unoperated ($N = 3$), received bilateral lesions of dorsolateral prefrontal cortex ($N = 4$), or received bilateral lesions of parietal cortex ($N = 3$). The cynomolgus monkeys were unoperated ($N = 3$) or received bilateral lesions of the hippocampal formation ($N = 3$).

The lesions of dorsolateral prefrontal cortex included cortex in both banks of the principal sulcus, the anterior bank of the arcuate sulcus, and all tissue on the dorsolateral surface rostral to the arcuate sulcus, (most of Brodmann's Area 9, Area 8, and some of Area 10; see FIG. 5), similar to lesions reported in Goldman (1971). The lesions of inferior parietal cortex included cortex in the posterior bank of the intraparietal sulcus, the superior temporal sulcus for about 10 mm, and all cortex between the two sulci including roughly 4 mm of the Sylvian fissure (most of Brodmann's Area 7). Lesions of the hippocampal formation included the entire hippocampus (Ammon's horn and the dentate gyrus) plus adjacent cortex (most of the parahippocampal gyrus including the subiculum and the posterior half of the entorhinal cortex). The temporal stem and amygdala were spared. All lesions were bilateral, symmetrical, and performed in one stage. All monkeys were given a minimum of 2 weeks following surgery to recover before testing.

The testing procedure within a trial was exactly the same as that used with infant monkeys, and very similar to that used with human infants (see FIG. 6).

All monkeys were tested for 14 days at each of 3 successive delay intervals: 2, 5, and 10 sec. In addition all cynomolgus monkeys were tested for 8 sessions using a delay of 15 sec, and those with lesions of the hippocampal formation were further tested for 8 sessions with a delay of 30 sec. Before each delay increment, each monkey received 5 sessions in which the delay was gradually increased over days.

When a subject is performing perfectly on $A\overline{B}$, it becomes a double alternation task (2 trials to the right, 2 to the left, etc.). The following procedure was used to minimize the possibility that subjects would treat $A\overline{B}$ as a double alternation task: If a monkey made no errors in a session, on the following session we required 3 correct reaches in a row following one of the reversals. The side of this reversal and whether it occurred early or late in the session were varied randomly.

The unoperated rhesus and cynomolgus monkeys, parietally operated rhesus monkeys, and hippocampally operated cynomolgous monkeys all performed well on $A\overline{B}$ at delay intervals of 2, 5, and 10 sec. The performance of these 3 subject groups was comparable at these delays, despite the species difference and despite the fact that some subjects had received surgery while others were intact (see TABLE 6).

Rhesus monkeys with lesions of dorsolateral prefrontal cortex, however, failed $A\overline{B}$ at all 3 levels of delay, including delays as brief as 2 sec. Moreover, they showed no evidence of improvement over the 14 days of testing at a given delay. In contrast, all other subjects were already performing well at each of these delays by the end of the transitional week (i.e., by the very first test day). At delays of 2 and 5 sec, monkeys with lesions of dorsolateral prefrontal cortex showed the classic $A\overline{B}$ error pattern (errors confined to reversal trials and to the trials immediately following reversal errors). At the 10-sec delay, however, they showed random and severely perseverative responding, similar to that seen in human infants when the delay is increased above the level at which the $A\overline{B}$ error occurs.

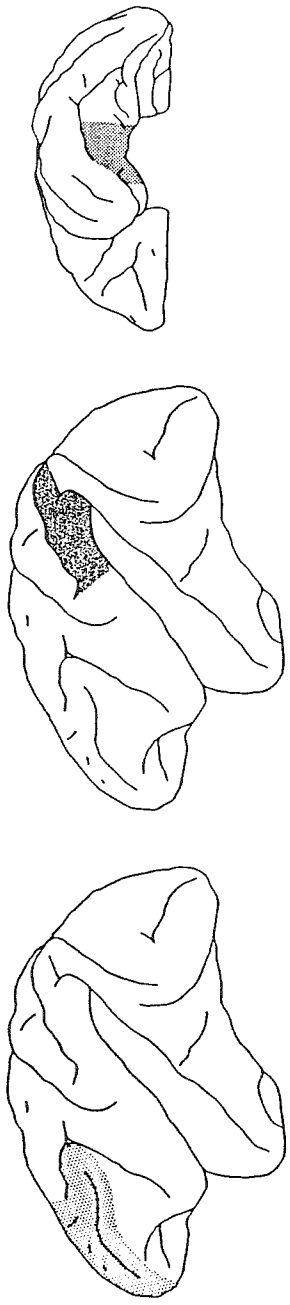

FIGURE 5. Sites of the brain lesions. From left to right: dorsolateral prefrontal cortex lesion, inferior parietal cortex lesion, and hippocampal formation lesion (including the subiculum and posterior portion of the entorhinal cortex). The prefrontal cortex and parietal cortex lesions are shown on lateral views of the brain. The hippocampus is a deep structure that cannot be seen from the brain's surface. In this ventral view of the brain the stippled area indicates the cortex that was removed in the hippocampal formation lesion. The hippocampus itself is buried deep beneath this region of cortex.

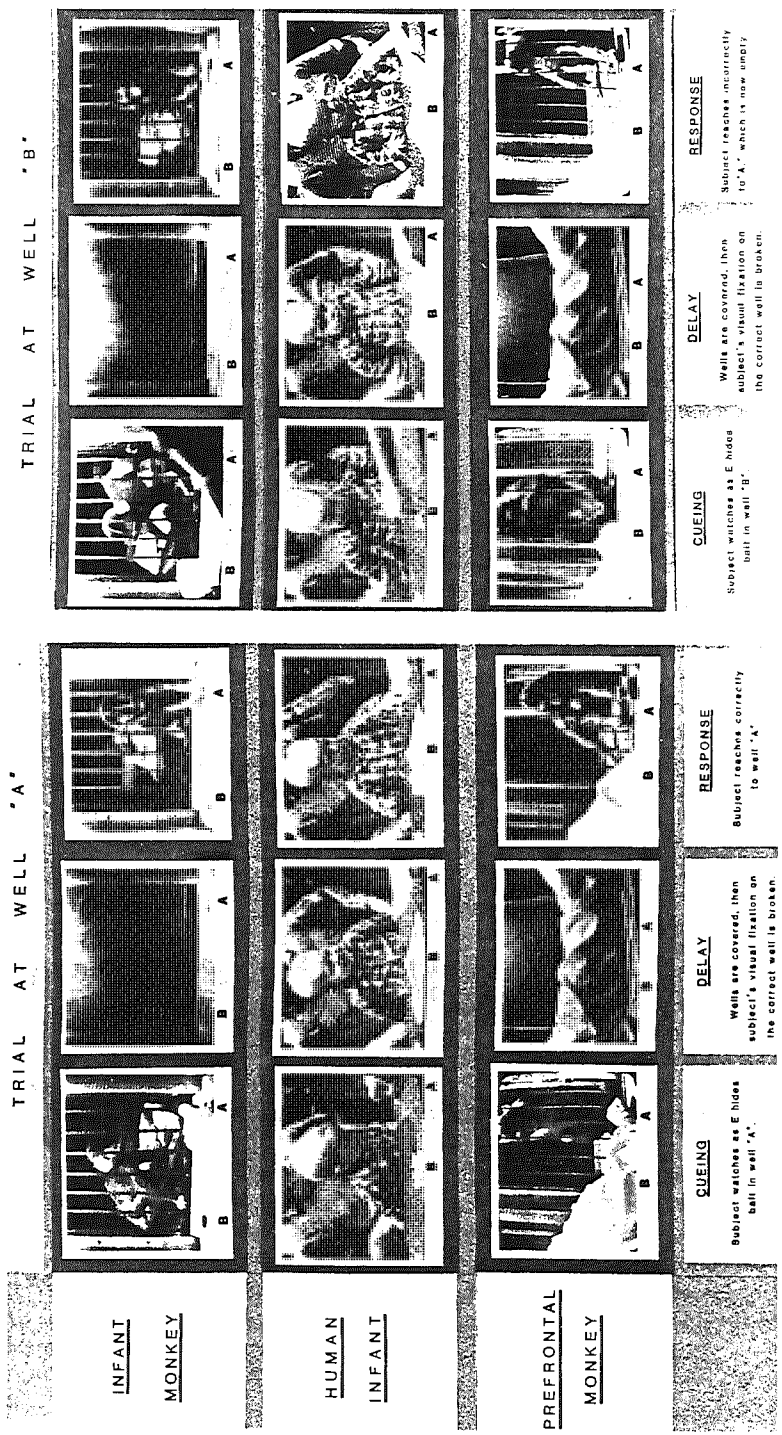

FIGURE 6. AB̄ testing with infant monkey, human infant, and adult monkey. It has been possible to use almost the same testing procedure with all groups of subjects, and the performance of human infants, infant monkeys, and adult monkeys with lesion of dorsolateral prefrontal cortex looked virtually identical.

TABLE 6. Percent Correct on the AB̄ Task by Delay and by Experimental Group

Experimental Groups	Delay (in Seconds) 2	5	10
Adult rhesus monkeys with lesions of dorsolateral prefrontal cortex			
F1	45		
F2	71	63	58
F3	67	67	64
F4	67	63	59
Mean	63	64	60
Adult rhesus monkeys with lesions of parietal cortex			
P1	97	94	92
P2	100	100	99
P3	98	99	98
Mean	98	98	96
Unoperated adult rhesus monkeys			
U1	99	98	98
U2	96	98	96
U3	99	96	97
Mean	98	97	97
Adult cynomolgus monkeys with lesions of the hippocampal formation			
H1	98	93	87
H2	100	88	86
H3	95	95	80
Mean	98	92	84
Unoperated adult cynomolgus monkeys			
C1	92	96	95
C2	99	91	91
C3	87	95	85
Mean	92	92	90
Infant rhesus monkeys with lesions of dorsolateral prefrontal cortex			
I1	81	75	65
I2	75	73	71
Mean	78	74	68
Unoperated infant rhesus monkey			
I3	97	97	97

When a prefrontally operated monkey maintained an orientation toward the correct well throughout the delay, the monkey usually reached correctly (similar to the results found with human infants and infant monkeys). This happened rarely, though, as all subjects were trained not to orient their bodies toward the correct well during the hiding or delay periods.

At delays of 15 sec, unoperated cynomolgus monkeys continued to perform well, but the performance of those with lesions of the hippocampal formation began to decline. At the 30-sec delay, the performance of the hippocampal monkeys was at roughly the same level as that of prefrontal monkeys when delays of 2–5 sec were used (see TABLE 6). Monkeys with lesions of the hippocampal formation, however, never showed the $A\overline{B}$ error pattern. At no delay did they tend to err on reversal trials (see Diamond, this volume, a).

In short, monkeys with lesions of dorsolateral prefrontal cortex failed $A\overline{B}$, showing the classic pattern of error, at the same delays (2–5 sec) as do human infants of 7½ to 9 months and infant monkeys of 1½ to 2½ months. All other experimental groups performed correctly at these delays. When able to circumvent the memory requirements of the task, however, monkeys with dorsolateral prefrontal cortex lesions performed well.

$A\overline{B}$: Infant Monkeys with Lesions of Dorsolateral Prefrontal Cortex

Lesions in the infant do not always produce the same effect as do lesions in the adult. If a neural region is late maturing, lesions of that region may produce deficits in the adult, but not in the infant (e.g., Divac, Rosvold & Szwarcbart, 1967; Goldman, 1971; 1974). It has been suggested that lower areas of the brain might mediate infants' performance on a task, even though performance of that task by adults is mediated by a later maturing area of the brain. Thus, although successful $A\overline{B}$ and delayed response performance appears to depend upon dorsolateral prefrontal cortex in the adult, this would not necessarily have to be true in the infant. For this reason, the effect of lesions of dorsolateral prefrontal cortex in infant monkeys has also been investigated.

Two of the infant rhesus monkeys who had been tested longitudinally on $A\overline{B}$ and delayed response from 1½ to 2½ months received bilateral lesions of dorsolateral prefrontal cortex at 4½ months (Diamond & Goldman-Rakic, 1986). Post-operative testing on $A\overline{B}$ began at 5½ months. A naive, unoperated infant monkey with no previous testing experience also began testing on $A\overline{B}$ at 5½ months. The prefrontal lesions were the same as those received by adult monkeys, and the prefrontal infant monkeys were tested on exactly the same $A\overline{B}$ testing procedure as were adult monkeys. Thus, they were tested for 14 days at each of 3 delays (2, 5, and 10 sec), plus 1 week of training preceding testing at each delay.

The infant monkeys with lesions of dorsolateral prefrontal cortex failed $A\overline{B}$ at all delays (2–10 sec) showing the $A\overline{B}$ error pattern at delays of 2–5 sec and random or deteriorated performance at delays of 10 sec. When they managed to orient their bodies toward the correct well throughout the delay, they reached correctly. They showed this impairment on $A\overline{B}$ despite their extensive preoperative training and their excellent $A\overline{B}$ performance with delays of 12 sec before surgery. There was no improvement in their postoperative performance on $A\overline{B}$ over the weeks of testing.

The unoperated infant monkey progressed through increasingly long delays on $A\overline{B}$ at a rapid rate. After pretraining and 1 week to slowly increase the delay from 0–2 sec, he performed perfectly at $A\overline{B}$ with a 2-sec delay from the first day of formal testing. He likewise succeeded at the 5- and 10-sec delays from the first day of testing at those delays. In contrast, the infant monkeys with lesions of dorsolateral prefrontal cortex showed no evidence of passing $A\overline{B}$ at delays of 2, 5, or 10 sec even by the end of their testing. Thus, in infant monkeys, as in adult monkeys, lesions of dorsolateral prefrontal cortex produced a profound deficit on the $A\overline{B}$ task as long as any demand whatsoever was placed on memory. If the memory demands could be circumvented by maintaining an orientation toward the correct well during the delay, infant monkeys succeeded on the task, even if dorsolateral prefrontal cortex had been removed.

Delayed Response: Infant Monkeys with Lesions of Dorsolateral Prefrontal Cortex

The same operated infant monkeys tested on $A\overline{B}$ were tested on delayed response (Diamond & Goldman-Rakic, 1986). Delayed response testing began for the prefrontal infant monkeys at 176 and 178 days of age (about 2 weeks after postoperative testing began on $A\overline{B}$) and continued until 245 and 276 days of age, respectively. The unoperated infant monkey was tested on delayed response from 200–244 days of age.

The prefrontal monkey who began testing at 178 days succeeded at delayed response with a delay of 2 sec (90% in 100 trials over 3 consecutive testings), but failed to ever succeed (even on a single day) at delays of 5 or 10 sec during the 8 sessions of testing at each delay. The prefrontal monkey who began delayed response testing at 176 days succeeded at delayed response when no delay was used, but failed to ever succeed with delays of 2 or 5 sec during the 8 sessions of testing at each delay. The unoperated monkey who began delayed response testing at 200 days of age was tested with a 10-sec delay on the first day of testing as that monkey had already passed $A\overline{B}$ with a 9-sec delay by that age. The monkey succeeded at delayed response, 10-sec delay, on the first day of testing. On the next day, delay was incremented to 13 sec, and he again reached correctly. Two days later he succeeded with a 15-sec delay, and then delay was slowly incremented to 20 sec. After failing the first 2 days at 20 sec, he succeeded on the third day. Testing continued with a delay of 20 sec for 3 consecutive testings (100 trials) during which he performed at the 90% level.

Thus, performance on delayed response was comparable to performance on $A\overline{B}$. The unoperated monkey was able to pass both tasks with a delay of 20 sec by the end of testing. Although 1 infant monkey with a lesion of dorsolateral prefrontal cortex passed delayed response with a delay of 2 sec, neither infant monkey with a lesion of dorsolateral prefrontal cortex was able to succeed on delayed response at any delay above 2 sec. When no delay was used, the prefrontal infant monkeys performed well, as do prefrontal adult monkeys.

Conclusions

Dorsolateral prefrontal cortex appears to be required for successful performance of the $A\overline{B}$ and delayed response tasks because of the memory requirements of the tasks. When no delay is imposed between hiding or retrieval, or when the subject orients toward the correct well throughout the delay, animals succeed on $A\overline{B}$ and delayed response even if dorsolateral prefrontal cortex has been removed. Only

when a delay is present, no matter how brief, and orientation toward the correct well is prevented, do animals without dorsolateral prefrontal cortex fail. This appears to be true for infant, as well as adult, monkeys.

CHARACTERISTICS OF THE MEMORY REQUIRED BY THE AB̄ AND DELAYED RESPONSE TASKS AND DEPENDENT ON DORSOLATERAL PREFRONTAL CORTEX

For the remainder of the paper I would like to explore what we can deduce about the characteristics of the memory ability that seems to be assessed by the AB̄ and delayed response tasks, seems to mature between 7½ and 12 months in human infants, and seems to be dependent on dorsolateral prefrontal cortex. What do we know about this memory ability? What hypotheses about this memory ability are in need of further testing? In what ways do the characteristics of the memory ability required for AB̄ and delayed response differ from the characteristics of the memory abilities required for other tasks?

(1) Errors occur at extremely *brief delays* (2–5 sec) on AB̄ and delayed response. Human infants, infant monkeys, and infant and adult monkeys with lesions of dorsolateral prefrontal cortex fail both AB̄ and delayed response when the very shortest delay is imposed. They succeed when there is no delay and/or when they look at, or orient their bodies toward, the correct well throughout the delay.

This can be contrasted with the delays associated with performance deficits following hippocampal damage. Monkeys with lesions of the hippocampal formation succeed on AB̄ and delayed response at brief delays. They only begin to fail when longer delays of at least 15–30 sec are used. This is consistent with their performance on all tests. Even on tasks selectively sensitive to hippocampal damage (such as delayed non-matching to sample) monkeys with lesions of the hippocampal formation do not fail at delays of 2, 5, or even 10 sec (e.g., Squire & Zola-Morgan, 1983; Zola-Morgan & Squire, 1986; Zola-Morgan, Squire & Amaral, 1989; Overman, Ormsby & Mishkin, 1990). Rather delays of at least 30–60 sec are usually needed before deficits appear. For example, the monkeys with lesions of the hippocampal formation tested on AB̄ were also tested on the delayed non-matching to sample task both before and after AB̄ testing. They showed the classic deficit on this task associated with hippocampal lesions, but this deficit did not appear until delays of 60 sec or longer were used (at delays of 8 and 15 sec, their performance on delayed non-matching to sample was normal) (Diamond *et al.*, 1989).

(2) Memory of where the reward is hidden must be maintained on-line to link together the various components of a trial to guide behavior (see also Fuster, this volume). That is, *the delay is imposed within a trial* (between hiding and response), as opposed to between trials or between testing sessions. When a delay is imposed between trials or between sessions, one is typically studying whether subjects can remember an association they have already learned; in AB̄ and delayed response, subjects must bridge a temporal gap in order to establish the association. In a sense, one might consider the AB̄ and delayed response tasks to require sustained attention to represented, or remembered, information, as subjects must concentrate on keeping their attention turned to their memory of where the reward was hidden in the absence of perceptual cues.

(3) *Memory must be updated on each trial.* Subjects must pay attention to the hiding on each trial and continually update their mental record of where the reward has been hidden, as opposed to learning and remembering only one rule, which can then guide performance on all trials. The only information relevant to the reward's location is where it was hidden on this particular trial. Indeed, it would be best if subjects could stop attending to where the reward had been hidden on previous trials, wipe the slate clean, and instead concentrate on where the reward was hidden on the current trial (see also point 9 below).

Human infants and prefrontally operated monkeys perform very well on tasks where a single rule, once learned, is sufficient to correctly guide performance on all trials. Their memory for this appears to be fully intact. For example, if a landmark indicates in which of two wells the reward has been hidden, so that on each trial following the rule "reach to the well with the landmark" would lead to a correct reach, monkeys with lesions of prefrontal cortex (Pohl, 1973) and human infants (Diamond, 1983) are able to learn and remember the association between the landmark and the reward, and to use that to successfully guide their reaching.

(4) What must be remembered is where the subject last *saw* the reward hidden, as opposed to where the subject last reached and found the reward. Human infants, infant monkeys, and monkeys with lesions of dorsolateral prefrontal cortex err on the AB̄ and delayed response tasks by reaching back to where they last retrieved the reward (or toward that well when multiple wells are used [Diamond, Cruttenden & Neiderman, 1989]). This suggests they remember, at some level, where they found the reward on previous trials, even though the time between their reach on a previous trial and their reach on the present trial is considerably longer than the few second delay between hiding and retrieval within a trial. Their last correct reach within the testing session may have been several minutes before the present trial.

There is even some suggestion they may remember where they last found the reward in the previous *testing session* (2 weeks ago for human infants; 24 hours for monkeys). This can be seen in performance on the first trial of a session. Although infants generally perform very well on the first trial of a testing session, we found that performance on this trial was significantly better for infants tested only once than for the infants tested longitudinally, suggesting that the experience of retrieving the toy in the previous session may still have been exerting an influence over the infants' behavior even 2 weeks later (Diamond & Doar, 1989).

It is as if the experience of successfully retrieving the reward has more of an influence on their behavior than the sight of where the reward has just been hidden. Their memory of where they last *saw* the reward being hidden is so fragile that they reach incorrectly just 2 or 5 sec after observing the hiding. It is this memory that appears to become more robust between 7½ and 12 months in human infants and appears to depend on dorsolateral prefrontal cortex. Memory of where the reward was last retrieved does not appear to depend on dorsolateral prefrontal cortex and appears to be quite robust very early in life, certainly by 7½ months.

(5) *The information to be remembered is presented only once.* On any given trial, subjects see the reward hidden only once. This differs from situations where subjects can gradually build up a memory over several presentations. In a comment that

combines points 4 and 5, Jacobsen and Nissen (1937) wrote that the subject "is not trained to the correct response by making it . . . but instead must respond on the basis of a single unrewarded and unpunished presentation" (p. 132).

(6) Moreover, *the information to be remembered is presented only briefly* (2–5 sec) during that single presentation. After the reward is hidden, the well is quickly covered. The subject has only a few seconds to see where the reward has gone. This is in contrast to paradigms such as visual paired comparison, where the information is presented for an extended period of time (15–60 sec) (see Fagan, this volume; Diamond, this volume, b), or Baillargeon's visual habituation techniques (e.g., Baillargeon, Spelke & Wasserman, 1985). In other words, subjects slow at encoding visual information into memory might perform well on paradigms such as visual paired comparison or Baillargeon's techniques, but fail A$\overline{B}$ and delayed response.

(7) *Memory is inferred from behavior,* as neither infants nor monkeys can respond verbally.

FURTHER SPECULATIONS ON THE CHARACTERISTICS OF THE MEMORY REQUIRED BY THE A$\overline{B}$ AND DELAYED RESPONSE TASKS AND DEPENDENT ON DORSOLATERAL PREFRONTAL CORTEX

(8) The A$\overline{B}$ and delayed response tasks appear to require *explicit or declarative memory,* rather than implicit or procedural memory, even though memory is only inferred from behavior. (Explicit memory is roughly memory of which the subject is aware. Implicit memory can be demonstrated in behavior without any conscious awareness of the "memory" on the part of the person. Adults with amnesia demonstrate robust memory in conditioning paradigms, for example, even though they have no conscious recollection of having seen or performed the task before [Weiskrantz & Warrington, 1979].) It is difficult to know how to distinguish explicit from implicit memory in nonverbal subjects (see Schacter, this volume; Mandler, this volume; Rovee-Collier, this volume). Perhaps Piaget's criterion for intentionality is also appropriate for indicating when a nonverbal subject is demonstrating explicit memory. Piaget was willing to credit infants with intentionality when they could demonstrate goal-directed behavior by acting on one object as a means to the goal of acting on another object ("means–end" behavior). A$\overline{B}$ and delayed response require such an indirect response: removing a cover in order to then retrieve the reward beneath it. We suspect, although we cannot prove, that when subjects reach to the correct well, they consciously or explicitly remember that the reward was hidden there.

(9) There is a potential for *proactive interference* from previous trials during A$\overline{B}$ and delayed response testing as the same two hiding places are used throughout. Once the reward has been hidden at well A on at least one trial and at well B on at least one trial, one might consider the task to be one of *temporal order memory*: "Where was the reward hidden most recently?"

Infants and prefrontally operated monkeys perform well at the first location (initial trials at well A); errors appear when the location of the reward changes (i.e., as soon as the hiding has occurred at least once at both wells). That is, errors first appear at the point where temporal order memory is first required.

There is no tendency, however, for performance to be worse during the second half of a testing session than during the first half, although interference would presumably be greater later in the session. Answering the question, "I have seen the reward hidden at both places. Where was it hidden last?" would presumably be harder after more and more trials; but the number of errors late in a session is no greater than the number of errors early in a session.

One of the prominent theories of prefrontal cortex function is that it is specialized for the memory of temporal order information (e.g., Milner *et al.,* 1985). Evidence for this viewpoint includes: When adult patients are shown a series of pictures, patients with frontal cortex damage can tell you which of 2 pictures they saw before, but not which of 2 pictures they saw most recently (Corsi cited in Milner, 1971). When asked about well-known events from the last several decades, frontal patients are impaired in recalling the order in which the events occurred, yet unimpaired in their recognition and recall of the events (Shimamura, Janowsky & Squire, in press).

(10) One might consider A$\overline{B}$ and delayed response to be tests of *spatial memory.* Typically, the hiding places differ only in left–right position (the wells and covers look identical), so the only way to keep track of where the reward has been hidden is to remember whether it was hidden on the left or on the right. Similar errors are found in human infants (Butterworth, 1976) and prefrontally operated monkeys (Fuster, 1980) if the hiding places differ only in up–down location.

One prominent theory of dorsolateral prefrontal cortex function is that it is specialized for the memory of spatial information in particular (e.g., Goldman-Rakic, 1987).[c] Evidence for this view includes: Monkeys with lesions of dorsolateral prefrontal cortex are less impaired on some nonspatial memory tasks (e.g., delayed object alternation) than they are on comparable spatial memory tasks (e.g., delayed spatial alternation) (Mishkin, Vest, Waxler & Rosvold, 1969). Monkeys with lesions of the principal sulcus (the "heart" of dorsolateral prefrontal cortex in the monkey) perform well on spatial tasks that do not require memory, but fail spatial tasks that require memory (Goldman & Rosvold, 1970). There are cells in dorsolateral prefrontal cortex that increase firing after a cue is presented and maintain that level of activity throughout the delay (i.e., they appear to serve a memory function); moreover, a subset of these cells is direction-selective, that is, they fire more if the cue was on the right or left (Fuster & Alexander, 1971; Niki, 1974; Funahashi *et al.,* 1989).

Moreover, the anatomical connections between dorsolateral prefrontal cortex and inferior parietal cortex (Brodmann's Area 7) are particularly strong (e.g., Schwartz & Goldman-Rakic, 1984). Indeed, not only are there heavy reciprocal connections between these 2 areas, but throughout diverse areas of the brain, wherever dorsolateral prefrontal cortex projects so does inferior parietal cortex, and in each case their projections interdigitate (i.e., columns of cells receiving projections from prefrontal cortex alternate with columns of cells receiving projections from parietal cortex) (e.g., Goldman-Rakic & Schwartz, 1982; Selemon & Goldman-

[c]Given the close association of frontal cortex with motor control, it may be that what has been taken as memory for spatial location of the cue is actually memory for the spatial location of where the response should be made. Present tasks do not adequately differentiate memory for cue location from response preparation.

Rakic, 1985a; 1985b). This is relevant because parietal cortex participates in the portion of the visual system specialized for the perception of motion rather than the perception of form or texture. It is conceivable that, through its connections with parietal cortex, dorsolateral prefrontal cortex might specialize in the memory of spatial information rather than memory of object features.

However, lesions of inferior parietal cortex leave A$\overline{B}$ and delayed response performance undisturbed. Subjects are evidently able to succeed at these tasks without the perceptual information processed in parietal cortex. Problematic for the spatial memory view is that infants and prefrontally operated monkeys generally perform well at the first location (well A), even though spatial memory is required here as elsewhere. Errors generally first appear only when location of the reward changes. Also, infants and prefrontal monkeys appear to be no better at remembering the location of the reward by the color of the cover than they are by spatial location alone (Diamond, 1983; Diamond & Goldman-Rakic, unpublished observations). Indeed, some of the cells in dorsolateral prefrontal cortex (and within the principal sulcus itself) that increase firing after a cue is presented and maintain that level of activity throughout the delay fire selectively depending on the *color* of the cue, just as other cells there fire selectively depending on the location of the cue (Quintana, Yajeya & Fuster, 1988; Wilson & Goldman-Rakic, 1989). In addition, most tasks diagnostic of frontal cortex damage in human adults do not appear to have a spatial component. For example, the classic test of frontal cortex function in adults is the Wisconsin Card Sort Test (e.g., Milner, 1963). Here, the sorting criterion (color, shape, or number) changes during testing, and subject must stay attentive to which criterion is currently correct. Spatial position is irrelevant to the task. Similarly, spatial position is irrelevant on the self-ordered pointing task, which requires subjects to keep track of what stimuli they have already pointed to. The spatial locations of the stimuli are scrambled after each reach. Indeed, when the stimuli are left stationary, so that the task can be solved by spatial memory, patients with frontal cortex damage perform well (Petrides & Milner, 1982). A version of the self-ordered task has recently been used with monkeys, where lesions confined only to the principal sulcus produced severe deficits in performance, even though spatial memory is irrelevant to the task (Petrides, 1988).

Most studies of A$\overline{B}$ performance in infants that have investigated memory for color have used hiding locations differing in both color and location (e.g., Bremner, 1978; Butterworth, Jarrett & Hicks, 1982; Goldfield & Dickerson, 1981). For example, the reward might be hidden under the black cover on the right, or the white cover on the left. Here, the comparison is actually between color + location versus location alone, and performance has been somewhat better in the color + location condition. We are currently preparing to investigate performance in conditions where the hiding locations are specified by either color alone or location alone. The "color alone" condition will resemble the delayed matching to sample for color task (see below) that has been used with monkeys.

Tulving and Schacter (e.g., Schacter, 1987; Tulving, 1989) have suggested that frontal cortex is critical for the memory of both space and time, specifically memory of the spatial or temporal context in which information is acquired.

(11) Memory for space or time may not be a unique ability, but may be an instance of *relational memory* in general. Spatial information (e.g., "Was the reward hidden on the right or left?") and temporal order information (e.g., "Where was the reward hidden most recently?") are inherently relational. Perhaps memory for relational information in general is dependent on dorsolateral prefrontal cortex and develops between 7½–12 months of age in human infants.

It would make sense if relational memory were more difficult than single-item memory, as the former requires remembering a relation between 2 things. Perhaps memory of information that is inherently relative, that is, that requires relating one thing to another (e.g., left, right; smaller, bigger; softer, louder; earlier, later) matures later and more slowly than memory of individual items (e.g., red, girl, circle) and requires involvement of dorsolateral prefrontal cortex.

Evidence consistent with this includes: Patients with frontal cortex damage often do well on typical delayed recall tests, but fail delayed comparison tests in which they must judge, for example, whether a color they saw earlier is the same shade as the color they see now, or whether a tone they just heard is the same pitch as the tone they hear now (Prisko, cited in Milner, 1964). Frontal patients are notoriously poor at relating 2 pieces of information together (e.g., Barbizet, 1970; Heilman & Valenstein, 1972). Grossman (1982) administered 8 visual and auditory reversal tasks (i.e., tasks which required that subjects appreciate the relation between original and transformed states) mediated by linguistic and nonlinguistic symbol systems to adults with localized brain damage. He found no domain-specific deficits; rather patients with frontal cortex damage were impaired across the board on the reversal tasks regardless of modality or content.

(12) I have argued that dorsolateral prefrontal cortex is required whenever *any information at all* must be remembered within a trial as long as the task *also* demands inhibition of a prepotent response as well (Diamond, 1985; 1988a; 1988b; 1989). That is, it may not matter whether one must remember temporal, spatial, relational, color, or object information. The critical factor may be whether the task demands *both memory and inhibition* of a dominant response. Evidence for the role of inhibition is discussed in Diamond (this volume, a). That evidence includes that fact that all memory tasks linked to dorsolateral prefrontal cortex in the monkey also impose an inhibitory demand. The *pattern* of error on the A$\overline{B}$ and delayed response tasks following dorsolateral prefrontal cortex damage, or in infants, cannot be accounted for by forgetting alone, for the delay is equal on all trials, but errors are not equally distributed across trials. Indeed, monkeys with lesions of the hippocampal formation, who have impaired memory, never show this pattern of error. This pattern of error follows what would be predicted on the basis of a deficit in inhibiting the dominant response. Moreover, when a task requires memory, but not inhibitory control, human infants perform well months before they first succeed on A$\overline{B}$ or other dorsolateral prefrontal cortex tasks.

COMPARISON WITH THE MEMORY ABILITIES REQUIRED BY OTHER PARADIGMS

Conditioning Tasks

Infants considerably younger than 7½ to 9 months are able to learn and remember an association between cue and response (see Lipsitt, this volume;

Rovee-Collier, this volume), as are prefrontal monkeys (e.g., Allen, 1943; Pohl, 1973). Conditioning has been demonstrated in infants shortly after birth and in simple organisms without frontal cortex. Once the association between cue and response has been learned, human infants of only 3 months can remember it for long periods (hours, days, and even weeks [e.g., Rovee-Collier, 1984; this volume]). Contrast this with the failure of human infants months older on the A$\overline{B}$ and delayed response tasks at delays of only a few seconds.

Indeed, within the A$\overline{B}$ and delayed response situations, infants and prefrontal monkeys appear to remember their last reinforced response, as they typically err by repeating it, although the delay between responses is substantially longer than the delay between hiding and response within a trial. Moreover, they can learn to associate the hidden reward with a landmark, and to use the landmark's location to guide their reaching (Pohl, 1973; Diamond, 1983).

The landmark condition requires memory, for the subject must remember the association between landmark and reward. However, the characteristics of the memory required here or in conditioning paradigms generally are very different from those typically present in A$\overline{B}$ or delayed response testing (see TABLE 7). Once the single association between landmark and reward is learned, and as long as the landmark accurately indicates the reward's location, the subject can use that to guide performance on all trials. If one's mind wanders during the hiding or one's memory lapses, that is no problem, as one need only look for the landmark when it is time to reach. Contrast this with the need to attend to the hiding on each trial and continually update the mental record of the reward's location. The association between landmark and reward is built up over many trials and involves the subject's action at the well, as opposed to merely observing a single hiding of the reward on a given trial. In addition, conditioning paradigms typically assess the subject's ability to remember an already learned association. For example, once a subject has learned that kicking will make a mobile move, will the subject remember that association between response and outcome a week later? In contrast, A$\overline{B}$ and delayed response impose the memory requirement during learning.

Delay of Reinforcement in Conditioning

When a memory demand has been imposed within a trial in a conditioning paradigm, the developmental progression looks very similar to that for A$\overline{B}$ and delayed response (Millar & Watson, 1979; Millar, this volume). Millar has studied how long a delay between response and reward infants can withstand within a trial (as opposed to how long a response once learned can be retained). He found that infants of 6 to 8 months can acquire a conditioned response if the delay between response and reinforcement is 0 sec, but not if it is 3 sec. Similarly, infants of 7½ to 8 months succeed on delayed response or A$\overline{B}$ when the delay between hiding and response is 0 sec, but not when it is 3 sec. Millar's task, like A$\overline{B}$ and delayed response, requires that memory be maintained on-line to link together the components of a trial (to relate the response to the reward in Millar's paradigm; to relate the cue to the response in A$\overline{B}$ and delayed response).

When there is a delay in the onset of reinforcement, the longer interval between response and reward provides more opportunity for the subject to make an irrelevant response. If the reward comes immediately after the correct response, the subject is more likely to know what action is being reinforced. The interference posed by extraneous actions during the delay period makes a delay of reinforcement paradigm more difficult. The need to overcome this interference may be similar to some of the requirements of the A$\overline{B}$ and delayed response tasks.

Similarly, monkeys with lesions of prefrontal cortex can make use of a cue in a conditioning paradigm if it is still present while they respond, but are impaired if there is a delay of 1 or 2 sec between cue and response (Passingham, personal communication).

On the other hand, in delay of reinforcement paradigms there is still only one rule to learn which can then guide performance on all trials. Memory does not need to be updated on each trial, and there is no problem of proactive interference from previous trials. Neither is there a spatial component. It is possible that the memory function, and the underlying neural system, required for delay of reinforcement conditioning is different from that required for A$\overline{B}$ and delayed response. It would then be only a coincidence that the developmental progression in performance on one delay of reinforcement task resembles that for A$\overline{B}$ and delayed response.

Trace Conditioning

In trace conditioning a brief delay is imposed between when the conditioned stimulus (CS) goes off and when the unconditioned stimulus (US) appears. This, too, is a paradigm requiring the integration of information over a within-trial temporal separation. Solomon, VanderSchaaf, Thompson, and Weisz (1986) have demonstrated that in rabbits, with a 0.5-sec trace interval between the tone (CS) and air puff (US), acquisition of the classically conditioned eyelid response to the tone is impaired by lesions to the hippocampus (see also Solomon, this volume), although when there is no trace interval (no delay between the offset of the tone and the onset of the airpuff), hippocampal lesions leave performance on the task unimpaired (Schmaltz & Theios, 1972; Solomon & Moore, 1975; Woodruf-Pak, Logan & Thompson, this volume).

Here is an instance where hippocampal function is required at the briefest of delays (0.5 sec). Why hippocampal function should be needed here to span such a brief delay while on other tasks hippocampal function is not required until delays reach at least 15–60 sec in length is unclear. The role of the hippocampus in trace conditioning has only been studied in the rabbit, not in primates; perhaps the hippocampus would not be required for this in primates. The role of prefrontal cortex in trace conditioning has never been studied in any species; it is possible that prefrontal cortex plays an important role here, at least in primates. However, why might A$\overline{B}$ and delayed response require prefrontal cortex involvement for brief within-trial delays, while trace conditioning requires hippocampal involvement for its brief within-trial delays? The answer probably lies in the fact that in most other respects the memory demands of trace conditioning differ from those of the A$\overline{B}$ and delayed response tasks (see TABLE 7). In trace conditioning there is only one rule to learn (a specific stimulus will be followed after a specific amount of time by a specific aversive stimulus—on all trials). Memory does not need to be updated on each trial; indeed it can be built up gradually over trials. There is no problem of proactive

TABLE 7.

Characteristics of the Type of Memory Required by the AB and Delayed Response Tasks	Other Behavioral Tasks							
	Conditioning	Delay of Reinforcement Conditioning	Trace Conditioning	Visual Paired Comparison	Delayed Non-Matching to Sample	Delayed Matching to Sample-Color	Multiple Boxes	Radial Arm Maze
(1) Errors occur at extremely *brief delays*.	no	yes	yes	no	no	yes	yes	yes
(2) The delay is imposed within a trial, as opposed to between trials or testing sessions.	no	no	no	yes	yes	yes	yes	yes
(3) Memory must be updated on each trial, as opposed to remembering only 1 rule which can guide performance on all trials.	no	no	no	yes	yes	yes	yes	yes
(4) What must be remembered is based on where the subject *saw* the reward hidden, as opposed to on the basis of reinforced acts.	no	no	no	yes	yes	yes	yes	yes
(5) The information to be remembered is presented only once.	no	no	no	yes	yes	yes	yes	yes

TABLE 7. *Continued*

Characteristics of the Type of Memory Required by the AB and Delayed Response Tasks	Other Behavioral Tasks							
	Conditioning	Delay of Reinforcement Conditioning	Trace Conditioning	Visual Paired Comparison	Delayed Non-Matching to Sample	Delayed Matching to Sample-Color	Multiple Boxes	Radial Arm Maze
(6) The information to be remembered is presented only briefly.	no	no	no	no	yes	yes	yes	yes
(7) Memory is inferred from behavior.	yes	yes	yes	yes	yes	yes	yes	yes
(8) Explicit memory is required, rather than implicit memory.	no	no ?	no ?	yes ?	yes	yes	yes	yes
(9) There is potential proactive interference. Memory of temporal order is required.	no	no	no	no	no	yes	yes	yes
(10) Memory of relative spatial location is required.	no	no	no	no	no	no	no	yes
(11) Not only memory, but also inhibition of a dominant response is required.	no	no	no ?	no	no	yes	yes	yes
Neural region required for successful performance	?	?	hippocampus	hippocampus &/or amygdala	hippocampus	frontal cortex	dorsolateral prefrontal cortex	hippocampus

interference, no need to keep the order of the trials straight, and no spatial component. The task posed by trace conditioning may be in timing when to blink, rather than in learning the association between a tone and an eventual air puff.

Visual Paired Comparison

In the visual paired comparison task, subjects look at a stimulus for a fixed familiarization period or until habituated, a delay is imposed, and then memory of the sample is tested by pairing the sample with another stimulus. Where the subject looks is recorded. Preferential looking at the new stimulus is taken as evidence of memory of the sample, since subjects prefer to look at something new rather than at the same old, boring thing (see Fagan, this volume).

A great many studies using visual habituation or paired comparison techniques have shown excellent memory over minutes, hours, and days in infants several months younger than those tested on A$\overline{\text{B}}$ or delayed response (e.g., Cohen, Gelber & Lazar, 1971; Fagan, this volume, Diamond, this volume, b; for similar results in infant monkeys see: Brickson & Bachevalier, 1984; Bachevalier, this volume). In habituation and visual paired comparison tasks, memory is built up over extended exposure to the stimulus, as opposed to the brief exposure to where the reward is hidden in A$\overline{\text{B}}$ or delayed response. Also, memory is assessed by where subjects look, rather than by where they reach. The response is simple (subjects merely look at what they are interested in) as opposed to the two-part means–end sequences required for A$\overline{\text{B}}$ and delayed response (subjects must first remove the cover, and then retrieve the reward). It is becoming commonplace to observe success months earlier when a simple looking response is required than when a means–end reaching response is required. For example, infants seem to remember that an object is behind a screen at 4–5 months when judged by their looking (Baillargeon, Spelke & Wasserman, 1985; Baillargeon, 1987) but not until 7–8 months when judged by their reaching (e.g., Wishart & Bower, 1984; see Diamond, this volume, b).

In many respects, however, the memory requirements of the visual paired comparison task appear to resemble those for A$\overline{\text{B}}$ and delayed response (see TABLE 7). For example, the delay is imposed within a trial. Memory must be updated on each trial (as a new sample must be remembered on each trial).

There is no requirement to remember temporal or spatial information, however, nor to inhibit a dominant response. Temporal order memory is not taxed because there is no problem of proactive interference, or confusion from previous trials about which object was the sample and which object is new on this particular trial, as different objects are used on every trial. Spatial information is irrelevant on the task; only what the sample looked like must be remembered. Subjects are encouraged to do what comes naturally (i.e., look at the stimulus that is most interesting to them), rather than to inhibit any strong behavioral tendency.

Visual paired comparison requires only a simple direct response (look at what interests you), and so might be thought to require implicit memory. However, hippocampal-amygdala lesions impair performance on the visual paired comparison task in both adult and infant monkeys (Brickson & Bachevalier, 1984; Bachevalier, this volume; Saunders, 1989). If the critical neural locus for the impairment on visual paired comparison is hippocampal, and not amygdalar, this would suggest that the task requires explicit memory. Therefore, it is not yet clear whether visual paired comparison is an implicit or explicit memory task.

Delayed Non-Matching to Sample

The delayed non-matching to sample task is formally quite similar to visual paired comparison. In both, a sample stimulus is presented, a delay imposed, and then the subject is presented with the choice of the sample or a new stimulus. Different stimuli are used on each trial. Spatial location of the stimuli provide no clue as to which was the sample and which is new.

In delayed non-matching to sample, as opposed to visual paired comparison, the subject *reaches* to the sample and displaces it to retrieve the reward underneath. After the delay, if the subject *reaches* to the new object, he or she will be able to retrieve the reward underneath it. A reach back to the sample goes unrewarded, as nothing is hidden underneath it.

Delayed non-matching to sample has been repeatedly shown to depend on the hippocampal neural system (see review in Zola-Morgan, this volume; Diamond, this volume, b)—at delays of 15–60 sec, but not at briefer delays. Lesions to dorsolateral prefrontal cortex do not impair performance on the task at any delay (Bachevalier & Mishkin, 1986). Infants cannot succeed on delayed non-matching to sample until almost 2 years of age even with delays of only 5 sec, but the limiting factor does not appear to be the memory requirements of the task (see Diamond, this volume, b).

In most respects the memory requirements of delayed non-matching to sample are the same as those for A$\overline{\text{B}}$ and delayed response (see TABLE 7). The *only* differences are that neither temporal order memory[f] memory of spatial location, nor inhibition of a dominant response are required for delayed non-matching to sample.

Note that if dorsolateral prefrontal cortex is necessary whenever a task requires both memory *and* inhibition, then success on delayed *matching* to sample should depend on dorsolateral prefrontal cortex involvement. For here, subjects must not only remember the sample, but they must inhibit their tendency to reach to the new stimulus. The prediction depends, of course, on the use of different objects on each trial (as is done with delayed non-matching to sample—so that one object is always new, and hence preferred) and on sufficient time with the sample to begin to get bored with it. This prediction remains to be tested. Delayed matching to sample with trial-unique stimuli has yet to be administered to monkeys with lesions of dorsolateral prefrontal cortex.[g]

[f] Delayed non-matching to sample poses no problem of proactive interference, as unique stimuli are used on each trial (hence there is no need to remember which of the 2 stimuli was the sample most recently).

[g] I have just discovered an old study in which both delayed non-matching to sample and delayed matching to sample were administered to monkeys with lesions of lateral frontal cortex (Mishkin, Prockop & Rosvold, 1962). These monkeys succeeded at delayed non-matching to sample, but failed delayed matching to sample, a pattern of performance fully in accord with the stated predictions. Unoperated control monkeys succeeded at both tasks, and monkeys with lesions of infero-temporal cortex failed both tasks.

Delayed Matching to Sample for Color, Automated Apparatus

In the 1960s and 70s investigators worked with a version of delayed matching to sample that was entirely automated. Here, a center response key was lit with, say, a red light. After a delay, the key to the left was illuminated with, say, red, while the key to the right was illuminated with, say, green. The same 2 colors were used on all trials, although their left–right placement was varied randomly over trials. On half of the trials, red served as the sample, and on half of the trials green did. Subjects were rewarded for reaching to the color that matched the sample.

After the first trial, neither stimulus is novel, and so the prepotent tendency to reach to a new stimulus is irrelevant here. However, the potential for proactive interference from previous trials is present. Subjects must keep track of which response choice is correct on this particular trial (as they must in $A\overline{B}$ and delayed response, but not in delayed non-matching to sample where trial-unique stimuli are used). Indeed, delayed matching to sample for color might as well have been named "delayed response for color." The requirements and procedures for delayed matching to sample for color are identical to those for delayed response, except in delayed response subjects must keep track of whether left or right is correct and here subjects must keep track of whether red or green is correct. Thus, results here should provide an excellent test for the centrality of spatial memory. If dorsolateral prefrontal cortex is required for delayed matching to sample for color then the memory function subserved by dorsolateral prefrontal cortex would be more general than memory of relative spatial position alone. The results thus far are equivocal.

When Stamm (1969) electrically stimulated dorsolateral prefrontal cortex (Brodmann's Areas 9 and 10, including the principal sulcus), temporarily disrupting function there, monkeys failed delayed matching to sample for color. Similarly, when Fuster and Bauer (1974; Bauer & Fuster, 1976) cooled the tissue in the principal sulcus, temporarily disrupting function, monkeys failed delayed matching to sample for color with even a very brief delay (4 sec), although they succeeded with no delay or a delay of only 1 sec. As expected, their performance deteriorated further at still longer delays. These results are identical to those Fuster and Bauer obtained for delayed response (see FIG. 7). When the tissue returned to normal temperature, performance on both tasks also returned to normal. It is difficult to limit stimulation or cooling to a very circumscribed area, however, and so it is likely that the effects spread at least a little beyond the target area.

Passingham (1975) surgically lesioned dorsolateral prefrontal cortex (including the principal sulcus) in one group of monkeys, and the neighboring ventrolateral prefrontal cortex (including the inferior convexity) in another group of monkeys. He found that those with dorsolateral prefrontal cortex lesions performed well on delayed matching to sample for color. The monkeys with ventrolateral prefrontal cortex lesions failed the task even with no delay. In other words, neither area of prefrontal cortex appeared to mediate the memory requirements of the task.

Mishkin and Manning (1978) restricted their lesions either to the principal sulcus alone, or to the inferior convexity alone. The monkeys with inferior convexity lesions were impaired at the shortest delay tested (1 sec). Moreover, they were already at chance at 1 sec, so there was no room for their performance to worsen with increasing delays. Testing was not conducted without a delay. As in Passingham's

results, it would appear that the problem for monkeys with inferior convexity lesions was independent of delay (i.e., independent of the memory requirements of the task). The monkeys with lesions restricted to the principal sulcus showed a small, but reliable impairment on the delayed matching to sample for color task. Lesions restricted to the principal sulcus produce a marked impairment, however, on delayed response, where the choices differ in spatial location alone (Butters *et al.*, 1969;

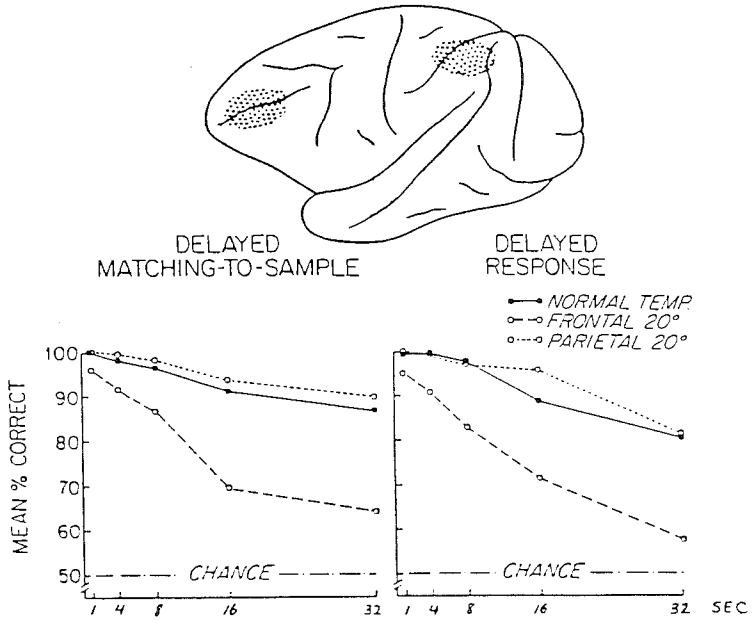

FIGURE 7. Performance of monkeys on the delayed matching to sample for color task and the delayed response task, with cortex at normal temperature (solid line), dorsolateral prefrontal cortex cooled (the larger dashes), and parietal cortex cooled (smaller dashes) (adapted from Bauer & Fuster, 1976).

Note that cooling of dorsolateral prefrontal cortex produces comparable and profound deficits on both tasks from the very shortest delay. Cooling parietal cortex, on the other hand, produces no effect on performance of either task.

Goldman & Rosvold, 1970). It would appear that dorsolateral prefrontal cortex plays some role in the memory of color, but a more central role in the memory of relative spatial location.

Multiple Boxes

The multiple boxes task (Petrides, 1988) was designed as a task appropriate for use with monkeys that would be similar to the self-ordered pointing task (Petrides & Milner, 1982) that is selectively sensitive to frontal cortex damage in human adults

(see point 10 above). In the multiple boxes task, the subject watches as a reward is placed in each of 3 boxes differing in both color and shape. The subject's task is to reach to each box one at a time, without repeating a choice. After each reach a screen is lowered for a 5-sec delay, and the boxes are scrambled. The boxes are always presented in a horizontal row, but which box is on the left, right, and center changes after each reach. Hence, memory of spatial location is irrelevant to the task. The task requires memory of which of these boxes differing in appearance the subject has already opened, and it requires inhibition because the subject is rewarded each time a new box is opened (which might strengthen the tendency to reach back to that box) and because subjects may have spontaneous preferences for particular boxes (and hence reach there first) and these preferences, too, must then be inhibited on later reaches. Petrides (1988) found that the multiple boxes task is disrupted specifically by lesions to the principal sulcus in the monkey.

Results such as these make it difficult to accept that dorsolateral prefrontal cortex is specialized solely for the memory of spatial information. Here, monkeys with lesions that are restricted solely to the principal sulcus are impaired on a task requiring no spatial location memory whatsoever. In other respects, however, the multiple boxes task poses many of the same memory demands as do $A\overline{B}$ and delayed response: The delay is brief; it is imposed within a trial; the information to be remembered is presented only once and briefly; and memory must be updated so that subjects keep track of which boxes they have already opened on this particular trial (see TABLE 7).

Multiple boxes requires one to remember which responses one has already made, and the appearance of the boxes one has already chosen. In a related study, Passingham (1985) baited each of 25 stationary foodwells. As in the multiple boxes task, the monkeys were to retrieve the rewards one at a time in any order they chose, the goal being to reach to all wells without repeating a choice. Here, however, the wells were identical and stationary (i.e., they were distinguishable only by spatial location). Performance on this task, as on multiple boxes, was significantly impaired by lesions of dorsolateral prefrontal cortex, even though this task required memory of spatial location while multiple boxes require memory of the appearance of the boxes, but not their locations. Both tasks require memory of the responses one has already made.

Radial Arm Maze

The radial arm maze, designed by Olton (1978; Olton, Collison & Werz, 1977; Olton & Samuelson, 1976) to study spatial memory in the rat, bears a marked resemblance to the multiple boxes and Passingham tasks, although they were developed independently. Here, each of the 8 arms of the maze are baited, and the rat is to retrieve all the rewards, in any order desired, the goal being to retrieve all the rewards without re-entering any given arm. The memory requirements of this task would seem to be the same as those imposed by Passingham's task just described. One might, therefore, have expected that success on this task would depend on dorsolateral prefrontal cortex involvement. Instead, however, this task has been firmly and repeatedly linked to hippocampal function in the rat (e.g., Olton, Walter & Gage, 1978; Olton & Papas, 1979).

Similarly, Sherry and Vaccarino (1989), who are interested in the hoarding behavior of birds, allowed black-capped chickadees to store food at various possible cache sites. After a long delay (3 hours), the birds were allowed to try to find the food they had stored. Birds with lesions of the hippocampus were severely impaired in finding all their food caches. Like prefrontal monkeys on the multiple boxes and Passingham tasks and hippocampal rats on the radial arm maze, they erred by making more revisiting errors.

Why is it that dorsolateral prefrontal cortex appears to be required for success on multiple boxes and the Passingham tasks in monkeys, while the hippocampus appears to be required for success on the radial arm maze in rats and the Sherry tasks in birds, given that the requirements of all these tasks seem to be so similar? One possibility is that the requirements of the former and latter tasks differ in some critical way. For example, the former tasks impose an extremely brief delay, whereas the latter tasks impose longer delays. The former tasks require only a reaching response; the latter tasks require a locomoter response in a larger spatial environment. Another possibility is that both dorsolateral prefrontal cortex and the hippocampus are required for all four of these tasks. Certainly there needs to be more investigation of hippocampal involvement in performance of multiple boxes and the Passingham tasks, and more investigation of frontal cortex involvement in performance of the radial arm maze and the Sherry tasks.

There is another intriguing possibility, however. The functions subserved by the hippocampus may change as one moves up the phylogenetic scale and more cortex is available to subserve higher cognitive functions. The hippocampal neural system may subserve some of the memory functions in simpler organisms that are subserved by the dorsolateral prefrontal cortex system in more complex organisms, with more fully developed cortices. It has been suggested (e.g., Goldman & Rosvold, 1972) that lower areas of the brain may subserve functions early in development that are taken over by higher areas of the brain when those higher areas finally mature. The present hypothesis extends this thinking to phylogenetic development as well. If the functions of the hippocampus are not the same in primates as in lower animals, then conclusions about hippocampal function in humans based on work with simpler organisms must be made only with great caution.

In conclusion, there is much evidence that dorsolateral prefrontal cortex subserves some memory function. Certain parameters of this function appear clear. When dorsolateral prefrontal cortex involvement is required, it is needed from the moment a delay is imposed. That is, tasks that require dorsolateral prefrontal cortex involvement show this dependence at the very shortest delay intervals. Also, tasks dependent on dorsolateral prefrontal cortex present the to-be-remembered information only once and briefly and require that the memory buffer be constantly updated and purged of old information that could proactively interfere with memory of new information. Whether dorsolateral prefrontal cortex performs these tasks for all to-be-remembered information or only for spatial or relational information is not yet clear. Also unclear are the fundamental differences between the memory functions subserved by dorsolateral prefrontal cortex and the hippocampus. One difference appears to be in time frame: Dorsolateral prefrontal cortex is required even at the shortest delays (e.g., 2–5 sec); the hippocampus is required at longer delays (e.g., 30–60 sec). The work on trace conditioning and the radial arm maze, however,

suggest that even this difference may not be absolute. With more work investigating the critical requirements of the various tasks, and looking at the hippocampal and frontal contributions to performance of the same tasks we should acquire a fuller understanding of how memory is changing during the first year of life and the roles of the various neural circuits in these memory abilities.

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DISCUSSION

L. P. ACREDOLO (*University of California, Davis, CA*): My question concerns the distinction between landmarks that were well associated with the reward versus those that were much more temporary. You report that the infants tested cross-sectionally (i.e., only once) did not seem to adopt the blue cover as a landmark indicating the toy's location. I was curious about the changes over age. How old were the cross-sectional subjects? Were they at all at a point in transition where the older infants were capable, within that one session, of using the blue cover as a marker?

A. DIAMOND (*University of Pennsylvania, Philadelphia, PA*): I didn't look at it over age, just at 9 months.

ACREDOLO: Just 9-month-olds. Well, that would make sense then.

N. FOX (*University of Maryland, College Park, MD*): Adele, can you talk a little bit about the change *before* they can solve the task? In other words, at the point in time where they are failing the 0 delay to the point in time where they are able to solve it with 0 delay. I guess my question is, Do you see any memory component involved?

DIAMOND: No, I don't think that improved memory can account for going from not being able to uncover a hidden object to being able to uncover it.

FOX: Either at a single hiding place or with 2 hiding wells?

DIAMOND: Yes.

R. THOMPSON (*University of Southern California, Los Angeles, CA*): I have always been, and still am, a little confused about the difference between hippocampal lesions and delayed non-matching to sample and dorsolateral prefrontal cortex lesions of the principal sulcus and the delayed matching to sample and delayed response tasks. Could you elaborate? I think an awful lot has been made out of a very small difference.

DIAMOND: When a task depends on dorsolateral prefrontal cortex, it is sensitive to disruption at the very shortest delays (e.g., 2–5 sec) and, in my opinion, requires both memory and inhibition. Delayed response requires both memory (of where the reward was hidden) and inhibition (of the tendency to repeat a rewarded, reinforced response); and in human and monkey infants, and in infant and adult monkeys with lesions of dorsolateral prefrontal cortex, performance on the task is disrupted at delays of 2–5 sec.

On the other hand, when a task depends on the hippocampus, hippocampal lesions only impair performance at delays longer than 15, 30, or 60 sec (there is no deficit at the shortest delays), and only memory is required (not inhibition as well). Delayed non-matching to sample with trial-unique stimuli requires only memory (of the sample), and it is impaired by lesions to the hippocampus, but not by lesions to dorsolateral prefrontal cortex.

Delayed *matching* to sample with trial-unique stimuli, unlike delayed non-matching, requires both memory (of the sample) *and* inhibition (of the natural preference for novel stimuli). It is for this reason that I think delayed matching to

sample should depend on dorsolateral prefrontal cortex and should be sensitive to disruption at the very shortest delays. (See TABLE 1 in the discussion of Fuster's paper.) This is true only when trial-unique stimuli are used, however, because when the same stimuli are used repeatedly over trials (as is done when the automated apparatus is used) no stimulus is novel.

With repeated stimuli you no longer have the novelty preference working for you in delayed non-matching to sample nor working against you in delayed matching to sample, since both stimuli are familiar. You now have a different problem, though; the problem of keeping straight which stimulus was the sample on this particular trial. Brenda Milner's work suggests that the ability to retain temporal order in memory depends on frontal cortex, but it is not yet clear how that maps onto the various subdivisions within frontal cortex.

One similarity between all these tasks and between the functions of dorsolateral prefrontal cortex and the hippocampus is in the general type of memory. It is not memory of an already learned association, it is not memory of a rule, and it is not memory built up slowly over trials. Rather, it is memory within a trial, during learning, to bridge a gap between the cue (hiding in the case of delayed response, sample stimulus in the case of delayed matching and non-matching to sample) and the response, and it is memory that must be updated on each trial.

P. TALLAL (*Rutgers University, Newark, NJ*): You say that you think delayed non-matching to sample is a straightforward memory task, and you also mention that the duration of exposure might be important. Have you actually studied the progression longitudinally of the duration of exposure? And would that help you to understand whether or not it is actually a problem of getting the information in to begin with versus remembering it?

DIAMOND: Well, all these things I am going to be addressing tomorrow. I am going to be talking about delayed non-matching to sample with trial-unique stimuli and will be talking about length of exposure.

A. SHIMAMURA (*University of California, Berkeley, CA*): Just a comment and a question. As you know, your data matches our data with prefrontal patients very closely in terms of the patients not really having a memory deficit, per se, but they do fail certain tasks that require inhibiting dominant responses. So, in terms of the adult data on frontal patients and the hippocampal lesion data with amnesic patients, it fits perfectly. A different point, in terms of the finding that the child or the monkey can self-correct, it seems that they only have two choices in those situations. One question is, if you had 3 wells there, do you think that they would then again self-correct?

DIAMOND: Well, that is why I used 7 wells in my multiple wells study as opposed to fewer, which is what most people have used. One question with A$\overline{\text{B}}$ was, if errors are due to poor memory, then infants should reach randomly around the correct well. If errors are due, in part, to difficulty inhibiting the tendency to reach back to A, however, then infants should reach to the A-side of B, rather than equally to both sides of B. It would not be necessary here that infants reach back specifically to A itself. Unfortunately, most studies of multiple wells have placed A and B at the endpoints. If B is an endpoint, the only way you can err is by reaching toward A, so most of the work on multiple wells had not addressed whether infants are being pulled to reach toward A or are just reaching randomly. With 7 wells we were able to have neither A nor B at an endpoint, and to have 2 wells between A and B, and 2 wells to the far side of B away from A. What we found is the errors are predominantly toward the A-side of B, rather than evenly distributed around B, which fits the notion that the problem is inhibiting going back toward A. If there are a lot of wells and they are farther apart, infants don't always go back *precisely* to A, but they reach back in that direction.[h]

Studies using 3 wells that have looked at self-correction after an incorrect reach have found that by and large infants are quite accurate on their second reach.[i]

FOX: Adele, you have tested both infant monkeys and infant humans. In your infant human data you showed the standard error bars, and there is quite a bit of variability in response and in performance. Is there the same degree of variability present in infant monkeys?

DIAMOND: Well, one of the things I didn't realize when I started working with monkeys is there are two differences between the work that is done with humans and the work that is done with monkeys. On the one hand, the people who work with animals are appalled by how few trials infancy researchers use. In A$\overline{\text{B}}$, most people use 5 trials. I use a big 15 trials. When they are studying animals, however, they use thousands of trials. But as an infancy researcher, I was used to having 20, 40, 100 subjects in a study, whereas researchers working with monkeys typically have 3 animals per group. Infancy researchers then look aghast and ask, "How can you draw any conclusions from an *N* of 3?"

So, my long-winded answer to you is I had 3 infant monkeys and there was some variability among them, but you can't tell very much about individual differences from an *N* of 3. If I had had an *N* of 25, as I did with the human infants, I think you would find fairly large error bars. Everything is happening over a much shorter time period in infant monkeys. If you adjust your axes to be in days, rather than weeks, I think you would get the same kinds of error bars for infant monkeys as you do for human infants.

FOX: One other follow-up question to that. You showed curves for delayed response and for A$\overline{\text{B}}$. Have you done a study with the same infants in both procedures?

DIAMOND: For infant monkeys yes, but not for human infants. I have not tested the same babies on both tests longitudinally. But here there were different infants, from different cities and different labs, with different testers, and the progressions on the two tasks are almost identical. In a way, it is *more* striking than if the results came from the same infants in the same lab.

J. COHEN (*Carnegie-Mellon University, Pittsburgh, PA*): It seems to me if you think that what's going on in dorsolateral prefrontal cortex has to do with interference, then you could test this using a classical interference paradigm, or some analogue thereof, like the Stroop test. I wonder if anybody has done work like this with monkeys—where you train them on one pair of associations more than on another, and where the stimuli have two different dimensions. One dimension could be

[h] DIAMOND, A., L. CRUTTENDEN & D. NEIDERMAN. 1989. Why have studies found better performance with multiple wells than with only two wells on A$\overline{\text{B}}$ Society for Research in Child Development Abstracts **6**.

[i] WEBB, R. A., B. MASSAN & I. NADOLNY. 1972. Information and strategy in the young child's search for hidden objects. Child Development **43**: 91–104.

trained more heavily than the other. Then the monkeys could be presented with the two-dimensional stimuli with some cues as to which dimension they should be attending to, and one could look at the effect. When the cue tells you to attend to the less well-trained dimension, you have to suppress a response based on the more heavily trained dimension. You would predict big Stroop effects.

DIAMOND: If you had a big preference, you would predict big Stroop effects.

COHEN: Right. I wondered has anyone done anything like this in animals?

DIAMOND: No. Does anyone else know of anything like that? Perhaps old studies of reversal learning have looked at something like that.

D. SCHACTER (*University of Arizona, Tucson, AZ*): I agree with Art Shimamura, that there is generally a nice fit between your results and the results on adult frontal patients. But I still don't see entirely how, for example, you need this *extra*-memory factor. If we think not of item memory, not of the same kind of memory that stems from the amnesic syndrome, but temporal, contextual memory, why do you need something else, let's say, to account for the classic Milner recency data where the deficit in the frontal patients is selectively remembering which of two items came first?

DIAMOND: Well, I don't have a great answer, but I think that it's keeping a relation in memory as opposed to keeping one isolated item in memory, so that monkeys or patients with frontal cortex damage have no problem remembering "did I see this before or didn't I see this before?" What they have trouble with is keeping track of the sequence in memory, keeping track of the *relations* between things.

SCHACTER: That is another kind of memory. That is not going outside the domain of memory.

DIAMOND: Right. That is why it is not a great answer.

SCHACTER: Oh, it *is* a great answer, it's just not the answer *you* want. It's the answer I want. You have made me very happy.

DIAMOND: But then there are things like the Stroop test, or making an eye movement *away* from a cue, which are very sensitive to frontal cortex damage but do not seem to require memory at all, or my object retrieval task. These tasks all require inhibition, though.

J. DELOACHE (*University of Illinois, Urbana, IL*): A couple of questions. Does the $A\overline{B}$ error depend on the subject actually reaching and retrieving an object on the first trial?

DIAMOND: Only one study has looked at that. That was in Gratch's lab and I think Evans was the person who did it.[j] If infants watch the experimenter retrieve the toy from well A (but are not allowed to reach), when the toy is hidden at B, infants are as likely to reach back to A (i.e., as likely to make the $A\overline{B}$ error) as when they have actually reached to A themselves on previous trials.

DELOACHE: The second question: I'm just curious. Both you and Linda Acredolo have been talking about if the baby can watch the relevant location during the intervals, and they do, then the baby performs much better. What would performance actually look like? Are they virtually always right if you allow them to orient toward the correct well during the delay?

[j] EVANS, W. F. & G. GRATCH. 1972. The stage IV error in Piaget's theory of object concept development: Difficulties in object conceptualization or spatial localization? Child Development **43:** 682–688.

DIAMOND: That is complicated. If they look at the correct well throughout the delay, yes, they are always right. But, if the question you are asking is, "If you allow them to look at the correct well throughout the delay are they always right?" The answer is no, they are not always right, because if you allow them to look they don't always continue to look, and if they don't continue to look then they are not always right. Simply allowing them to look at the correct well does not guarantee that they will do so, or will continue to do so without looking away.

DELOACHE: Would there be individual differences among babies in this?

DIAMOND: It is more a matter of age differences.

ACREDOLO: We found a correlation between locomotion, the length of time they have been locomoting, and their tendency to watch in the $A\overline{B}$ test. So you do find individual differences, and in our research it is related to locomotor ability.

TALLAL: In these tasks you are talking about memory, and you are teaching them something (i.e., rewarding them at A), but you did mention that infants and monkeys also have a natural preference for novelty, and you have suggested that prefrontal cortex should play a major role when inhibition is required of a learned or of a "natural" tendency. I was wondering about—sort of making a leap here—some of the work by Mark Johnson in Cambridge and in London where he has found the natural preference, let's say in chicks, for looking and running towards conspecifics, and a natural preference in babies within 10 min of looking towards face-like objects or presentations.[k] Would a lesion of dorsolateral prefrontal cortex interfere with something like this that is a predisposition rather than learned?

DIAMOND: Well, it certainly should. I think that is exactly what is happening on the object retrieval task (my transparent barrier task), where the natural preference is to reach for the visible object, reach straight for it. It is not something we teach them. It is something they come to the situation innately prepared to do. Success here requires inhibiting that tendency and instead taking the circuitous route that will get you through the opening. Dorsolateral prefrontal cortex lesions totally disrupt performance on this task. Frontal monkeys persist in trying to reach straight for the visible object—so do human infants.

TALLAL: How about something *really* innate like face preference?

DIAMOND: Well, I haven't looked at face preference. I have been trying to get Gabriel Horn, who is the person Mark [Johnson] initially did this with, to look at chicks because he wants to argue that what they are looking at is basically the analogue of prefrontal cortex. I think he probably will do this at some point, and Mark and I have been talking too. I think it is a very good idea to take the situations Mark has been looking at and see if you need prefrontal cortex to inhibit the strong preferences he has shown.

R. CASE (*Stanford University, Stanford, CA*): We have phenomena with older children that look a lot like this, in the sense that you have to establish a response first and then have some memory and inhibitory requirements. These things come in a lot later and are invariably damaged or interfered with in adult patients who have

[k] JOHNSON, M. H. 1988. Memories of mother. New Scientist **18:** 60–62.

G. HORN & M. H. JOHNSON. 1989. Memory systems in the chick: Dissociations and neuronal analysis. Neuropsychologia **27:** 1–22.

JOHNSON, M. H. 1989. Information processing and storage during filial imprinting. *In* Kin Recognition. P. G. Hopper, Ed. Cambridge University Press. Cambridge, England.

damage to the frontal lobes. My question is, if this is in place by 8, 9, or 12 months, what further maturation is bringing in these later things? In answer to Dan [Schacter's] question, if you look at the Wisconsin Card Sort Test, all frontal patients do absolutely terribly on that, and their errors are always ones like those found on the A$\bar{B}$ task; they are always going for the previous response.

DIAMOND: That is a very important question. What I am trying to say can easily be misinterpreted. Although I am trying to argue that there is a maturation in dorsolateral prefrontal cortex between 8–12 months in human infants, that does not mean dorsolateral prefrontal cortex is fully mature by 12 months. We know it is *not* fully mature by 12 months. It is probably not fully mature until puberty. So, even if something important is changing in prefrontal cortex during infancy, it is not that prefrontal cortex is fully mature by the end of infancy; those changes keep continuing. Kathi Boyer and I have looked at changes in preschool children on prefrontal tests. We have looked at a simplified version of the Wisconsin Card Sort, for example, where you see changes from 3–5 years.[l] If you use the adult version of the Wisconsin Card Sort, children cannot do that until they are 10 or 11 years old.[m] These abilities keep developing. If you complicate the test, you are going to see developmental progressions in performance of the test at later ages. Even if I am correct that important maturational changes occur in prefrontal cortex during infancy, prefrontal cortex keeps maturing long past infancy.

CASE: Also, the Thatcher sort of data suggests that with maturation you are getting connections from the frontal lobes backwards and sometimes other things are coming in.[n] Is it maturation *in* dorsolateral prefrontal cortex or maturation of connections between there and other areas?

DIAMOND: There are two answers to that. First, when I say "dorsolateral prefrontal cortex," I mean the "dorsolateral prefrontal cortical system." It doesn't make sense to talk about an isolated structure in the brain, so I am talking about connections. For example, one possible change in the prefrontal cortical system during infancy might be in the maturation of the prefrontal projection to the superior colliculus. Another answer to your question is that I tend to talk about dorsolateral prefrontal cortex as maturing during this period as if it is the only thing that is maturing, or as if that is where most of the action is. However, there is suggestive evidence that I may be completely wrong there, that neocortical areas may be more of a unit than we had been thinking, not simply the prefrontal cortex system and the parietal system, and so forth, but that the whole neocortex may be maturing more as a unit than previously thought. Certainly this is suggested by the work of John-Pierre Bourgeois and Pasko Rakic, where they looked at synaptic density and were greatly surprised to find changes across all neocortical areas (including visual cortex and dorsolateral prefrontal cortex) in synaptic density during exactly the same age

[l] DIAMOND, A. & K. BOYER. 1989. A version of the Wisconsin Card Sort Test for use with preschool children, and an exploration of their sources of error. Journal of Clinical and Experimental Neuropsychology. **11:** 83.

[m] CHELUNE, G. J. & R. A. BAER. 1986. Developmental norms for the Wisconsin Card Sorting Test. Journal of Clinical & Experimental Neuropsychology **8:** 219–228.

[n] For example, THATCHER, R. W., R. A. WALKER & S. GIUDICE. 1987. Human cerebral hemispheres develop at different rates and ages. Science **236:** 1110–1112.

period.[o] Emilie Marcus and Tom Carew, in their work in *Aplysia,* have found changes across diverse domains all occurring over the same time period (see Marcus & Carew, this volume). It may be that the brain is much more of a system in terms of development than I had been thinking it was.

J. FAGAN (*Case Western Reserve University, Cleveland, OH*): When you study a particular task like A$\bar{B}$, lesion a part of the brain, and show that performance on the task is interrupted, you make a general statement about the effect of the lesion on memory. It is not memory in general, however. It is memory for a particular kind of information. We tend to forget what kind of information we are talking about. Are you talking about memory for position, basically?

DIAMOND: Well, I want to argue that, although this task requires memory of position, I don't think the memory functions that are dependent on dorsolateral prefrontal cortex or that are maturing between 8–12 months are specific to position.

FAGAN: This is my concern. That is, were other tests done to look at the disruption of memory for form, color, patterns, and so forth? Obviously your babies were attending to color, and they had long-term memory for it because they went to the blue cover even on their next visit two weeks later, so they had formed a good long-term memory for at least one other kind of information. In focusing on a task, you tend to get away from focusing on what kind of information they are encoding. For example, I am very concerned with the nature of intelligence. Now, position doesn't say much to me about intelligence, because on the standard two-choice discrimination test later in life, a retarded child will do better on position discrimination than a normal child, because it is higher in the hierarchy of being responded to. It is an old error factor in Harlow's work. He tried to get rid of position preferences, which were often the first way monkeys tried to solve a task.

DIAMOND: I will respond to the questions and points you raise in order, Joe. First, have we looked at memory for color, form, etcetera? The reason I would like to use delayed matching to sample with trial-unique objects is to address that—because there the choices would differ in color and shape, but positional information would be irrelevant. Delayed response for color is essentially delayed matching to sample with the same two colors used on all trials (e.g., as Joaquin Fuster and others do it in the automated apparatus). This is impaired by lesions to prefrontal cortex, but it is not yet clear, at least to me, what the critical site within prefrontal cortex is. A$\bar{B}$ has been tested with choices that differ in position alone, or that differ in both position and color but never with choices that differ in color alone. We hope to do that in the next year or two. You are exactly right that one needs to look at different tests, or different variants of the same test, to understand the characteristics of the memory ability dependent on dorsolateral prefrontal cortex and developing between 8–12 months of age.

Second, it is true that infants have a very good long-term memory that the blue cover is associated with the toy, as you mentioned. Infants also have a very good long-term memory of position, that is, on their next visit two weeks later they often reach back to the well where they found the toy on the previous session. Memory built up by repeated association is different, however, from memory of something you

[o] RAKIC, P., J-P. BOURGEOIS, N. ZECEVIC, M. F. ECKENHOFF & P. S. GOLDMAN-RAKIC. 1986. Concurrent overproduction of synapses in diverse regions of the primate cerebral cortex. Science **232:** 232–235.

have observed only once. Memory built up by repeated association is robust and long-lasting even in quite young infants and is not sensitive to damage of either frontal cortex or the hippocampus. In fact, the A$\overline{B}$ task pits these two kinds of memories against one another—memory of past associations between reaching to A and finding the toy and memory that where you just saw the toy hidden now was well B.

Finally, what you say about position preferences and Harlow's work fits exactly with what I am saying about delayed response and A$\overline{B}$. Infants and prefrontal monkeys are perfect at the first well, at the first position. It is when they have to *reverse* a position preference that you get errors. The initial preference, going to a position, is easy to establish. The problem comes in when you say, "Ok, it's the other position that is correct now."

Pat Goldman-Rakic would say that dorsolateral prefrontal cortex is specialized for the memory of spatial location or position. She would answer your question differently than I. I think it does not matter what kind of information must be remembered as long as *both* memory and inhibition of a predisposition are required.

L. NADEL (*University of Arizona, Tucson, AZ*): You say that dorsolateral prefrontal cortex is important for both memory and inhibition. Why should those two functions be conjoined, be subserved by the same neural system? What do they have in common?

DIAMOND: That is an excellent question. Unfortunately, I do not have an excellent answer. I have always been troubled by postulating these two seemingly unrelated abilities. I wish I could think of some overarching ability of which they are both instances. Or, I wish I could show how one is simply derivative of the other. It is unparsimonious to postulate two abilities. The data, however, seem to keep pointing in that direction. I don't know why memory and inhibition should be dependent on the same neural circuit, except that the data keep indicating that that is, in fact, the case.

Note, though, that since dorsolateral prefrontal cortex is required at such very brief delays (e.g., 2–5 sec), one might better conceive of the function we have been calling "memory" as "sustaining attention" instead. Now, sustaining attention requires inhibition as much as does not repeating a rewarded response, for one must resist distraction in order to keep one's attention focused. Hence, perhaps we are looking at two aspects of the same ability, the ability to exercise inhibitory control.

NADEL: I have another point that elaborates on something Joe [Fagan] was saying. I think that one of the messages of yesterday's session is that the kinds of features that infants respond to are not necessarily the same kinds of things that adults respond to when they are dealing with objects in the world or whatever. A lot of the tasks that people are talking about involve simply importing notions about what is being responded to from work with adults. This may actually be rather mistaken; infants may not be responding to those same things. The tasks are the same, but the question remains, Are the infants actually attending, and responding, to the same features that adults would be attending and responding to?

DIAMOND: What task in particular, Lynn?

NADEL: Any of the tasks that you are dealing with.

DIAMOND: Well, tasks like A$\overline{B}$ were not imported from adults or even from monkeys.

NADEL: But, the concepts that we use for describing those tasks are the concepts that are essentially adult concepts. They are devised by adult experimenters, and we talk about them in ways that are familiar to us. But the question remains, what is the infant dealing with essentially?

DIAMOND: Right. That is an important point.

Also, one of the points that gets lost when you start to divide things up as I have done by presenting the work in two different talks, is that babies of 7 months can remember an awful lot longer than 2–5 sec. I am not at all stating that 2–5 sec is the memory span of an infant. I think part of why 7-month-olds fail at 2–5 sec on A$\overline{B}$ has to do with the particular kind of memory required here, but a lot of it has to do with additional requirements of the task above memory (e.g., inhibition).

NADEL: Let me also add a point about the novelty experiments and the prediction about the delayed matching to sample task and the effect of lesions on performance of that task. Novelty preference is a relative thing, so you would probably be forced to predict that even though you would get deficits on delayed matching to sample with lesions of dorsolateral prefrontal cortex, if we gave the monkeys a really exciting reward, that would switch back to being a straight, non-novelty preference situation and the deficit should go away.

DIAMOND: Right. On A$\overline{B}$, for example, infants can withstand a longer delay for a greatly preferred reward. But, on A$\overline{B}$ they see what the reward is and where it is going, whereas on delayed matching to sample they do not. But certainly if you did delayed matching to sample with objects where the familiar one is more preferred than the novel one, monkeys with lesions of dorsolateral prefrontal cortex should show no deficit whatsoever there.

NADEL: It should be manipulable with that.

DIAMOND: Yes.

ACREDOLO: If you had varied the distance between wells A and B for your infants, would you have found a different slope, still increasing, but different perhaps?

DIAMOND: I did not vary the spatial separation between A and B. But, yes, I agree that it is quite likely the slope would be different for different distances between A and B. Actually, I don't think the slopes themselves would be different, rather their intercepts would be different. I did a little study in which we used food rather than toys, and infants did much better with the food. These were well-fed, middle-class infants. When we used food they could withstand delays 4 or 5 sec longer than they could when we hid a toy, even a toy that by all of our measures they were very interested in. But, it was not a difference of night and day. The infants were better when we used food, but they weren't perfect.