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Comparison of human infants and rhesus monkeys on Piaget's AB task: evidence for dependence on dorsolateral prefrontal cortex

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Summary. This paper reports evidence linking dorsolateral prefrontal cortex with one of the cognitive abilities that emerge between 7.5–12 months in the human infant. The task used was Piaget's Stage IV Object Permanence Test, known as AB (pronounced "A not B"). The AB task was administered (a) to human infants who were followed longitudinally and (b) to intact and operated adult rhesus monkeys with bilateral prefrontal and parietal lesions. Human infants displayed a clear developmental progression in AB performance, i.e., the length of delay required to elicit the AB error pattern increased from 2–5 s at 7.5–9 months to over 10 s at 12 months of age. Monkeys with bilateral ablations of dorsolateral prefrontal cortex performed on the AB task as did human infants of 7.5–9 months; i.e., they showed the AB error pattern at delays of 2–5 s and chance performance at 10 s. Unoperated and parietally operated monkeys succeeded at delays of 2, 5, and 10 s; as did 12 month old human infants. AB bears a striking resemblance to Delayed Response, the classic test for dorsolateral prefrontal function in the rhesus monkey, and indeed performance on AB and Delayed Response in the same animals in the present study was fully comparable. These findings provide direct evidence that AB performance depends upon dorsolateral prefrontal cortex in rhesus monkeys and indicates that maturation of dorsolateral prefrontal cortex may underlie the developmental improvement in AB performance of human infants from 7.5–12 months of age. This improvement marks the development of the ability to hold a goal in mind in the absence of external cues, and to use that remembered goal to guide behavior despite the pull of previous reinforcement to act otherwise. This confers flexibility and freedom to choose and control what one does.

Key words: Piaget's AB task – Dorsolateral prefrontal cortex – Humans – Monkeys

Introduction

The AB task, devised by Piaget (1954 [1937]: 50–65), is one of the classic tests of human cognitive development in the first year of life. For Piaget, this task, which requires an infant to uncover a toy hidden in one of two possible locations, measured the earliest emergence of intentionality [i.e., behavior directed from the outset by a goal (e.g., get toy), requiring planning and foresight in that one or more intermediate acts (e.g., remove cover) must be executed before the goal can be obtained] and for that reason the earliest emergence of truly intelligent behavior. Intentionality is still absent at 6 months according to Piaget, but its rudiments, assessed by tasks such as AB, are clearly present by 12 months (Piaget 1952 [1936]: 210–262). Performance of human infants on AB has been studied extensively and this task has become firmly established as a reliable marker of developmental change between 7.5–12 months of age (for excellent reviews see Gratch 1975; Schuberth 1982; Harris 1986). For example, human infants of 7.5–9 months succeed at AB when there is no delay, but fail when a delay of only 1–5 s is introduced (e.g., Gratch et al. 1974; Diamond 1985). By 12 months of age, human infants succeed on AB even at delays as long as 10 s (Diamond 1985). (Below 7.5 months, human cannot be tested on AB because they will not reach for a hidden object.)

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Furthermore, when infants fail, they make the characteristic $A\overline{B}$ error, which derives its name from the observation, first made by Piaget (1954), that infants correctly find a toy at the first place it is hidden (A), but when the side of hiding is reversed to B they err. They reach back to the toy's former location, even though they have observed the hiding at B and even though they were able to succeed on the earlier trials at A.

$A\overline{B}$ is similar to several tasks that have been widely used to study cognition in macaque monkeys, such as Delayed Response and Spatial Reversal. The similarity between $A\overline{B}$ and Delayed Response is particularly striking and, for that reason, is the focus of the current paper. A trial in either task consists of (a) cueing – the subject watches as a desired object is hidden in one of two identical wells, (b) delay – the subject is required to wait a few seconds, and (c) response – the subject is allowed to uncover one of the wells, and, if correct, retrieve the desired object. For any individual trial, the procedures for $A\overline{B}$ and Delayed Response are identical.

Delayed Response performance in monkeys has been shown to depend upon dorsolateral prefrontal cortex (e.g., Jacobsen 1935, 1936; Goldman and Rosvold 1970). This link between Delayed Response and dorsolateral prefrontal cortex has been demonstrated by neuropsychological, physiological, pharmacological, and metabolic methods (for reviews see Fuster 1980; Goldman-Rakic 1987). Because such a diversity of techniques have all produced the same result, the association between Delayed Response and dorsolateral prefrontal cortex is one of the best established brain-behavior relations in the study of cortical localization.

The performance of monkeys with lesions of dorsolateral prefrontal cortex on Delayed Response is remarkably comparable to that of 7.5–9 month old human infants on $A\overline{B}$. For example, like human infants of 7.5–9 months on $A\overline{B}$, monkeys with lesions of dorsolateral prefrontal cortex succeed on Delayed Response when there is no delay, but fail when a delay of only 1–5 s is introduced (e.g., Harlow et al. 1952; Goldman et al. 1970).

The main goal of the present study was to examine the performance of adult rhesus monkeys with dorsolateral prefrontal lesions on the Piagetian $A\overline{B}$ task and to compare their performance with that of human infants on $A\overline{B}$. Given (1) the similarities between the $A\overline{B}$ and Delayed Response tasks and (2) the strong link between performance on Delayed Response and dorsolateral prefrontal cortex, we predicted that monkeys with dorsolateral prefrontal ablations would show a profound impairment on $A\overline{B}$. Furthermore, given (3) the marked similarity between the $A\overline{B}$ performance of human infants between 7.5–9 months of age and the Delayed Response performance of monkeys with lesions of dorsolateral prefrontal cortex, we predicted that monkeys with such lesions would fail $A\overline{B}$ under the same conditions and in the same ways as do human infants of 7.5–9 months.

Material and methods

Subjects

Human infants.[1] Twenty-five healthy, full-term infants (14 female, 11 male) were tested on the $A\overline{B}$ task every two weeks. All infants were located through the Boston birth records. All were from intact homes. Only four had older siblings. Most came from upper middle-class homes. Most mothers had worked at some time, but only five continued to work after the baby's birth.

When an infant could first reach for a hidden object (range = 6.5–8.5 months) $A\overline{B}$ testing began and continued through the age of 12 months.[2] Parents were informed of the general objectives of the study, were given $3 for each testing session, and received a report of the major findings.

Nonhuman primates. Ten rhesus monkeys (*Macaca mulatta*; 6 female, 4 male) were tested on the same task as the human infants. The monkeys ranged in age from 2–6 years. All were in good health. Four animals (3 female: ages 2, 4, and 4 years; 1 male: age 4) received bilateral prefrontal ablations. Two control groups were used, one operated and one unoperated: Three monkeys (1 female: age 2; 2 male: ages 2 and 6) received bilateral resection of the posterior parietal cortex. The unoperated control group consisted of 3 animals as well (2 female: both age 2; 1 male, age 3).

Parietal cortex was selected as the site for the control lesion because (1) it has been implicated in spatial processing (Pohl 1973; Mountcastle et al. 1975; Ungerleider and Brody 1977; Brody and Pribram 1978) and some theories of $A\overline{B}$ performance give prominence to the spatial component of the task (Butterworth 1975; Bremner and Bryant 1977; Bremner 1978), (2) inferior parietal cortex, like dorsolateral prefrontal cortex, is a multimodal association area (Mesulam et al. 1977; Hyvärinen 1982; Cavada and Goldman-Rakic 1986), and (3) inferior parietal cortex is sufficiently large that lesions comparable in size to those of prefrontal cortex could be made.

Two frontal, parietal, and unoperated monkeys received training on Delayed Response prior to $A\overline{B}$ testing. One frontal and one parietal animal were retested on Delayed Response postoperatively prior to $A\overline{B}$. Table 1 summarizes the sex, age, and testing histories of the animals.

Surgical procedures

The dorsolateral prefrontal lesions included cortex in both banks of the principal sulcus, the anterior bank of the arcuate sulcus, and all tissue on the dorsolateral surface rostral of the arcuate sulcus

1 The human data were collected by the first author at Harvard University in the laboratory of Jerome Kagan, as part of her dissertation in partial fulfillment of the requirements of the Ph.D.

2 Age in months = number of weeks divided by 4.33. Thus, for example, a 9 month old infant would be between 38 weeks, 6 days and 43 weeks, 2 days

Table 1.

Groups	Sex	Age (in years) at $A\overline{B}$ testing	Prior testing experience		Period between surgery and $A\overline{B}$ testing
			Pre-operative	Post-operative	
Prefrontally operated animals					
F1F[a]	F	4	Visual discrimination	None	3 weeks
F2F	F	4	DR to 120 s	Multiple wells	12 months
F3F	F	2	None	None	3 weeks
F4M	M	4	DR to 10 s	DR to 2 s	6 months
Parietally operated animals					
P1F	F	2	DR to 2 s	None	3 weeks
P2M	M	6	DR to 120 s; Multiple wells	DR to 10 s; Multiple wells	12 months
P3M	M	2	None	None	3 weeks
Unoperated animals					
U1F	F	2	DR to 10 s	—	—
U2F	F	2	None	—	—
U3M	M	3	DR to 20 s	—	—

[a] First letter of a subject's designation refers to the experimental group, e.g., F = Frontally ablated. The last letter of a subject's designation refers to the sex of the animal

(Brodmann's Areas 8, 9, and 10), similar to lesions reported in Goldman 1971.

The parietal lesions included the posterior bank of the intraparietal sulcus, the posterior bank of 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). Figure 1 illustrates the intended lesion sites. As the animals are still involved in behavioral experiments; verification of lesion sites will be reported in subsequent reports.

All ablations were bilateral, symmetrical, and performed in one stage. Surgery was performed under aseptic condition using Nembutal anesthesia (40 mg/kg) administered intravenously. Fluids were administered throughout surgery and breathing and heart rate were monitored continually. Craniectomy was performed over the prefrontal or parietal areas in each hemisphere followed by opening the dura. Tissue in the target area was aspirated with a small gauge Pribram sucker. Bleeding was controlled by electrocautery and pressure. Wounds were sutured in anatomical layers with silk and polyglactin thread. Each subject was kept under close observation following surgery until full consciousness was regained, and each received 40,000 units/kg penicillin and a pain killer, 2 mg/kg pentazocine lactate. If the animal appeared to be in pain on the day following surgery, pentazocine lactate was again administered. A minimum of two weeks was allowed for postoperative recovery. Time between surgery and $A\overline{B}$ training ranged from 2 weeks to 1 year within both the prefrontal and parietal experimental groups.

Apparatus

Human infants. The $A\overline{B}$ apparatus consisted of a testing table with embedded wells. The tabletop was 87.5 cm long and 37.5 cm wide. The wells were 9.4 cm in diameter, 7.5 cm deep, and 27.5 cm apart, center to center. Light blue cotton cloths (22 × 22 cm) served as the covers. These cloths held little intrinsic interest for the infants and were easy for them to remove.

Nonhuman primates. A stationary testing tray, 67.5 cm long and 15.5 cm wide, with embedded wells was used. The wells were 2.5 cm in diameter, 1 cm deep, and 20 cm apart, center to center. Orange matboard placques (5 × 7.5 cm) served as covers.

Testing procedure

Human infants. All subjects were tested individually in the laboratory. An infant was seated on the parent's lap facing the testing table, equidistant from the wells. The experimenter was seated across the table, facing parent and child. A trial began with the experimenter holding up a bait (a toy the infant particularly liked) to catch the infant's attention. As the subject watched, the experimenter slowly hid the bait in one of two wells. Particular care was taken to insure that the subject observed this. If the infant looked away while the bait was being hidden, the infant's attention was recaptured and the hiding repeated. The experimenter then covered the two wells simultaneously.

With the covering of the wells, the delay period began. Subjects were prevented from straining, turning, or looking at a well during the delay. The parent restrained the infant's arms and torso gently but firmly from the beginning of the trial until the end of the delay period. Parents were instructed to look straight ahead during the delay and to release the infants's hands as soon as the experimenter said "okay". Visual fixation of the wells was broken by the experimenter calling to the infant during the delay and counting aloud, which caused the infant to look up. After the delay, the subject was allowed to reach.

A reach was defined as the removal of a cover. A "reach" was not scored if the subject began to reach toward a cover, but withdrew his or her hand before touching it, or touched a cover but

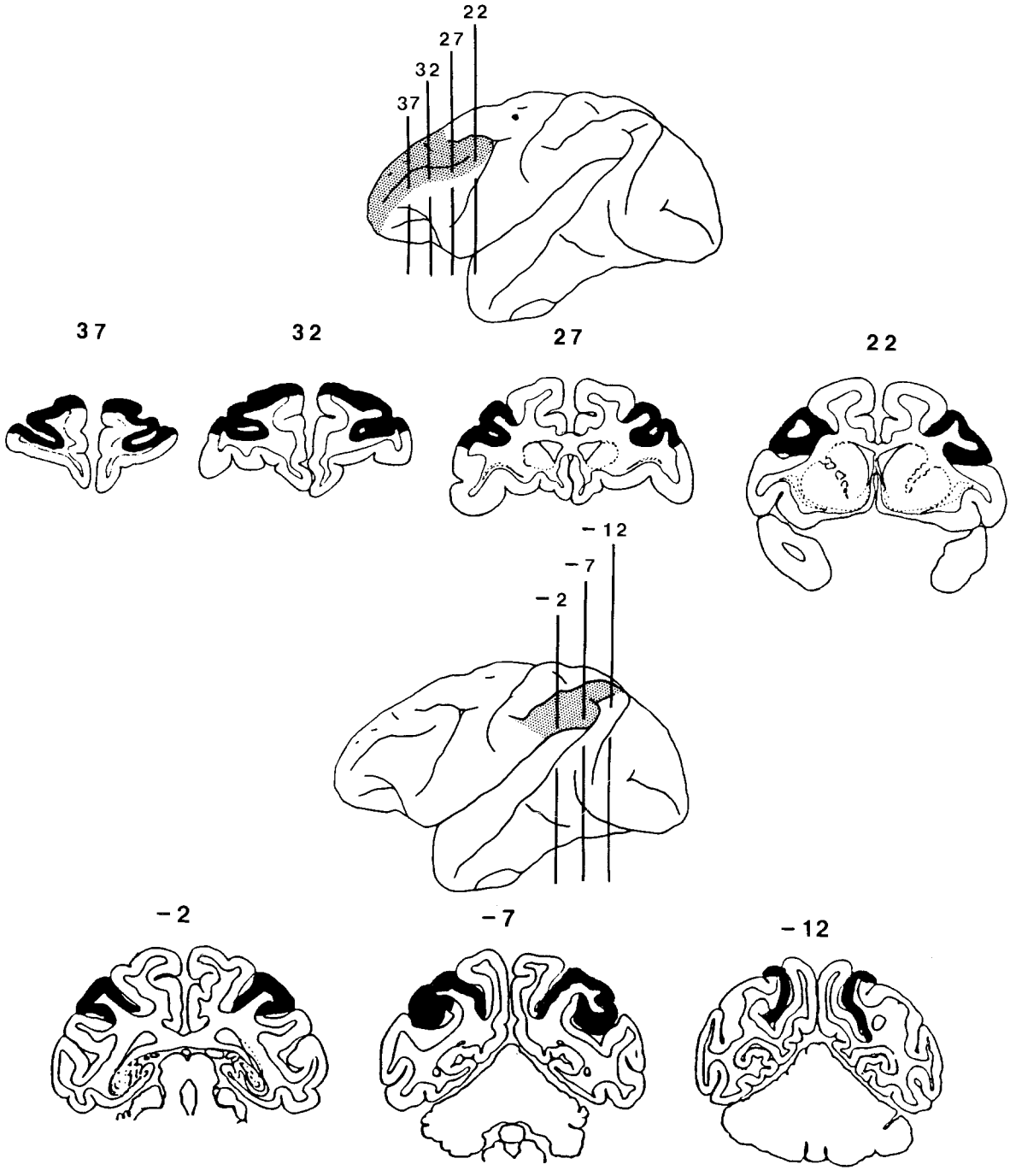

Fig. 1. Diagram of intended cortical ablations projected on the left hemisphere and in coronal sections. Dorsolateral prefrontal site is shown above and inferior parietal site below

did not remove it. If, on the other hand, the subject reached to one well, uncovered it, and then immediately reached to another well without looking into the first, the subject was credited nevertheless with reaching to the first well. A correct reach was rewarded by receipt of the bait. When a subject reached incorrectly, the experimenter directed the subject's attention to the correct well and showed the bait to the subject, but did not permit the subject to have the bait.

The bait was hidden in the same well until the subject was correct at least once ($\bar{x}$ = 2 consecutively correct responses, range = 1–3). The median number of trials per session was 15, and side of hiding was reversed 3–5 times within a session. Total number of trials and total reversals were kept as equal as possible across sessions. Because reversals were only administered following correct reaches, subjects who made numerous errors required more trials in order to receive the same number of reversals as subjects who made few errors. Hence, number of reversals and total trials could not be kept perfectly constant. One way to try to equalize total number of trials was to allow number of correct reaches prior to a reversal to vary from 1–3. It has been

demonstrated that within the range of 1–3 the number of consecutively correct reaches preceding a reversal does not affect performance on the reversal trial (Evans 1973; Butterworth 1977; Diamond 1983).

All sessions were recorded on video tape. This permitted coders to verify the infant's level of interest in the toy, the actual length of delay, whether the infant, in fact, observed the hiding, whether and how long the infant looked up during the delay, whether the infant strained, turned, or reached during the delay, etc. Coders were not informed of the experimental hypotheses and were trained to be conservative in their judgments. Average intercoder reliability was r = 0.92.

Nonhuman primates. The very same $A\overline{B}$ task described above was given to the nonhuman primates in the Wisconsin General Testing Apparatus (WGTA). Each subject was tested individually in the laboratory by the same experimenter who had tested the infants. The monkey was caged on one side of the testing table, and the experimenter sat on the opposite side, facing the subject.

The only differences in procedure were: (1) food was used as the bait instead of a toy, (2) visual fixation was broken by lowering an opaque screen instead of by merely calling to the subject, (3) monkeys were not restrained from moving during the delay (although if they showed any sign of position cueing this habit was broken), and (4) two consecutively correct reaches were required before side of hiding was reversed (human infants were administered a reversal following 1–3 consecutively correct reaches).

Control animals often reached correctly on all trials. When this happens, $A\overline{B}$ becomes a double alternation task (2 trials to the left, 2 right, 2 left, etc.). To minimize the possibility that animals would treat $A\overline{B}$ as double alternation and not as a hiding problem, if an animal performed errorlessly throughout a session, on the following session we required *3* consecutively correct reaches at the same hiding place before we administered one of the reversals.

Procedure for incrementing delay

Human infants. Preliminary testing began for each human infant before that infant could uncover a hidden object with no delay. Testing on $A\overline{B}$ began with a 0 s delay as soon as that infant first uncovered a hidden object from a single hiding place.

Each infant was tested every two weeks. Delay was incremented over sessions using performance on the preceding two sessions as an initial guide. If performance on the previous session had been at or above the 90% level, then delay was increased 2–3 s on the present session. If an infant had committed the $A\overline{B}$ error at the same delay on the preceding *two* visits, delay was also increased 2–3 s. If the infant showed deteriorated performance during the last session, or if delay had been incremented for the preceding session and the infant had not performed at or above the 90% level, then the same delay as on the preceding visit was used.

Within any session, if an infant performed perfectly at the initial hiding place and on the first reversal trial, delay was increased 2–3 s[3]. If the infant still performed perfectly at the initial hiding place and first reversal trial at the longer delay, delay was incremented 2–3 s more for $A\overline{B}$ testing proper. If, on the other hand, the infant was distressed at the initial delay selected and made more than one error before side of hiding was even reversed, the delay was decreased by 2–3 s for $A\overline{B}$ testing proper.

3 It was intended that all delay changes be 2 s. However, in verifying the actual length of delay from the video tape records, we found some changes were actually 3 s. Delay changes of 2 versus 3 s were not statistically different in effect and are therefore pooled

Table 2. Typical $A\overline{B}$ testing session illustrating types of trials

Trial no.	Side of hiding	Reach	Type of trial: Repeat following correct	Reversal following correct	Repeat following error
1	L				
2			 X		
3	R	errs	 X		
4		errs			 X
5		errs	. . . . .		 X
6			. . . . .		 X
7			 X		
8	L	errs	 X		
9		errs	. . . . .		 X
10			. . . . .		 X
11			 X		
etc.					

Side of hiding = where toy is hidden. When toy is hidden in the same well as on the previous trial, this column is left blank. L = Left Well; R = Right Well

Type of trial is determined by whether side of hiding is the same as on the previous trial or not and by whether the subject was correct on the previous trial or not. repeat following correct trials = reward is hidden in same well as on previous trial, and subject was correct on previous trial

Reversal following correct trials = reward is NOT hidden in same well as on previous trial; subject was correct on previous trial

repeat following error trials = reward is hidden in same well as on previous trial; subject reached to the wrong well on previous trial

Trial 1 is not characterized by type of trial as there is no trial previous to it. In $A\overline{B}$, reversals are always administered after a correct reach, hence there are no reversal following error trials

Nonhuman primates. Each rhesus monkey was given two weeks of training to acquaint the naive animal, or reacquaint an animal after surgery and recovery period, to the WGTA, the opaque screen, and the testing procedure. After these two weeks of practice, formal $A\overline{B}$ testing at a 2 s delay began. All monkeys were tested for 14 sessions on $A\overline{B}$ at a 2 s delay.

All animals were then given one week of practice on $A\overline{B}$ at gradually longer delays to prepare for testing at a 5 s delay. One operated animal (F1F) with a prefrontal lesion died during this period. The death was accidental and unrelated to the frontal lesion. From this point on, the prefrontal group, like the two control groups, contained three subjects. All monkeys were tested for 14 sessions on $A\overline{B}$ at a 5 s delay. Another week of practice at gradually longer delays followed; then came 14 sessions of $A\overline{B}$ with a 10 s delay.

Types of trials

$A\overline{B}$ trials can be divided into three categories, illustrated in Table 2, based on (a) whether side of hiding is the same as on the previous trial or reversed and (b) whether the subject was correct on the previous trial or not. The three categories of trials are: (1) REPEAT FOLLOWING CORRECT: the subject reached correctly on the preceding trial, and the bait is again hidden in the same well; (2) REVERSAL: the subject reached correctly on the preceding trial, but the bait is now hidden in the other well; and (3) REPEAT FOLLOWING ERROR: the subject reached to the wrong well on the preceding trial, and the bait is again hidden in the same well.

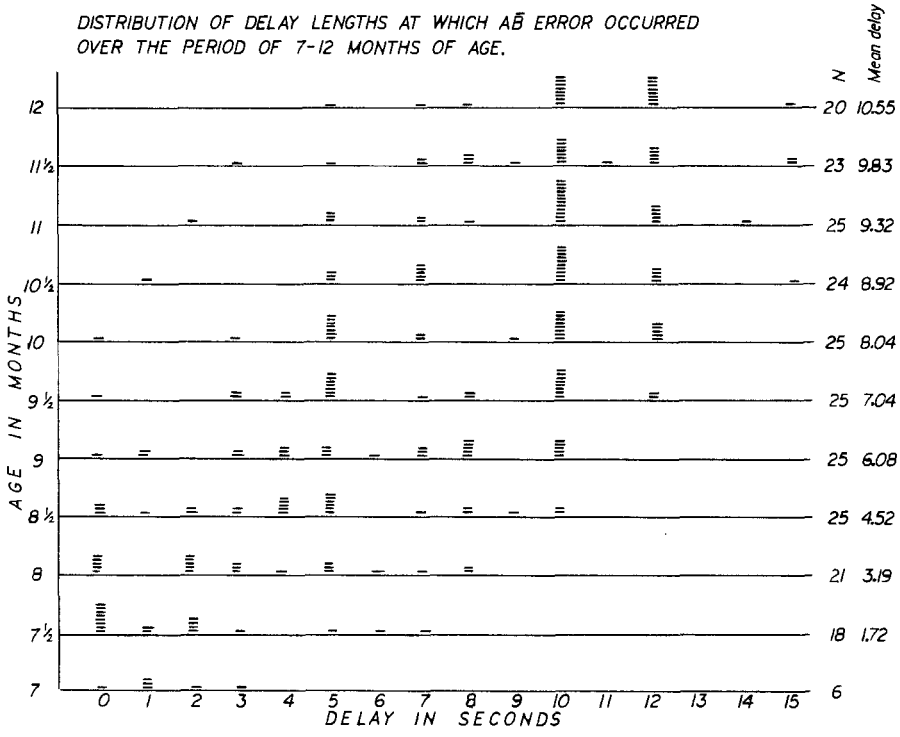

Fig. 2. Distribution of delay lengths at which AB̄ error occurred over the period of 7–12 months of age in human infants

Criteria for the AB̄ error

Only a certain pattern of performance is indicative of the AB̄ error. For example, if errors occur with equal frequency on all types of trials, the subject is reaching randomly. Only if errors occur on specific trials, in the face of otherwise accurate reaching, is the subject committing the AB̄ error.

Within a session, a subject was said to have made the AB̄ error if the following criteria were met: 1) At least one error on REVERSAL. This is the crux of the AB̄ error: when side of hiding is reversed, the subject reaches back to the previous hiding place. To reduce the likelihood that this was a chance event, one of the next two creteria had to be met as well: 2) This error was not repeated on the next trial, or 3) the subject erred on at least one more REVERSAL during the same session. Finally, because errors should be confined to REVERSALS and to the trials immediately following REVERSALS, there is a fourth criterion: Each time the subject is correct, if that trial is repeated unchanged, the subject should again be correct. If, in any given session, more than one error occurred on REPEAT FOLLOWING CORRECT trials, this performance was considered too poor to meet the criterion for the AB̄ error. Severely deteriorated performance was characterized by indications of stress and disturbance, such as long strings of errors, no reach at all on some trials, and/or much circling and agitation.

Delayed response testing

Two monkeys in the frontal, parietal, and unoperated groups received pre-operative training on Delayed Response prior to AB̄ testing. One frontal and one parietal animal were re-tested on Delayed Response post-operatively prior to AB̄. (See Table 1.) Delayed Response trials were administered exactly like AB̄ trials, except that side of hiding was varied randomly over trials and the number of trials per session was 30. Testing began at the 2 s delay and delay was incremented on the next session each time the animal passed criterion on a given session. Criterion at 2, 5, and 10 s was 90% correct over 100 consecutive trials (3 days; 40 trials on third day). Criterion at all other delays was 90% correct over 30 trials. Delay was incremented by 1 s up to 10 s, by units of 2 s from 10–20 s, and by 5 s thereafter.

Results

Human infants

Performance at 2 s delay. Most infants below 8–8.5 months made the AB̄ error at delays of 2 s or less. Only one infant above 11 months did so.

At the age of 7 months, 5 of the 6 infants tested (83%) committed the characteristic AB̄ error at delays of 2 s or less. (Only 6 infants could be tested on AB̄ at 7 months, as the 19 others still made no attempt to uncover a hidden object.) At 7.5 months, the average delay at which the AB̄ error occurred was 2 s (range = 0–7 s, N = 18). Most 7.5 month old infants (78%) committed the AB̄ error at delays of 2 s or less. At 8 months, the average delay for the AB̄ error was 3 s (range = 0–8 s, N = 21). About half (48%) of the infants aged 8 months committed the AB̄ error at delays of 2 s or less.

By 8.5 months, however, the average delay for the AB̄ error was 4.5 s (range = 0–10 s, N = 25), and only 20% of the infants now committed the AB̄ error at delays of 2 s or less. By 9 months, only 3 of the 25 infants still committed the AB̄ error at delays of 2 s or less. After that, one infant continued to do so through 11 months of age. By 11.5 and 12 months,

no infant committed the $A\overline{B}$ error at delays of 2 s or less (see Fig. 2).

Six infants above 11 months were tested within the same session on the delay predicted to yield the $A\overline{B}$ error and also at a delay 2–3 s *shorter* (Diamond 1985). Shorter delays ranged from 3–10 s. Order of delay presentation was counterbalanced across subjects. For every subject the results were the same: when delay was reduced 2–3 s below the delay at which the $A\overline{B}$ error occurred, errors disappeared. This evidence is important because it reveals the effect of delay within the same subject and same testing session.

Performance at 5 s delay. No infant of 7 months had passed the necessary criteria to permit testing at 5 s. Most 7.5 month old infants (83%, N = 18) committed the $A\overline{B}$ at delays below 5 s. Even at 8 months, most infants (67%, N = 21) were still making the $A\overline{B}$ error at delays than 5 s.

By 8.5 months, however, half of the infants made the $A\overline{B}$ error at delays of 5 ± 2 s, ($\overline{x} = 4.5$ s). At 9 months, the average delay for the $A\overline{B}$ error was 6 s.

By 9.5 months, however, over half the infants required delays greater than 5 s for the $A\overline{B}$ error to appear. By 11 months, only 16% of the infants made the $A\overline{B}$ error at delays of 5 s or less, and by 12 months only 1 infant was doing so.

Performance at 10 s delay. No infant below 8.5 months had passed the criteria to permit $A\overline{B}$ testing at 10 s, whereas by 12 months the average delay needed before any errors appeared was longer than 10 s. Only 3 infants were still committing the $A\overline{B}$ error at delays under 10 s by 12 months of age.

Five infants below 9 months received complete $A\overline{B}$ testing, within the same session, on the delay predicted to yield the $A\overline{B}$ error and also at a delay 2–3 s longer (Diamond 1985). The longer delays were 6–8 s. All 5 infants displayed the $A\overline{B}$ error at the shorter delay, but at delays of 6–8 s they showed *deteriorated performance*, i.e. unusually long error strings, errors even on REPEAT FOLLOWING CORRECT trials, and refusal to reach at all on some trials. Thus, even at delays less than 10 s, infants below 9 months performed more poorly than $A\overline{B}$ error standards.

Pattern of errors. Errors were not randomly distributed over trials. The following pattern of behavior was found during $A\overline{B}$ error performance: Infants reached correctly on 79% of the REPEAT FOLLOWING CORRECT trials, but they reached correctly on only 34% of the REVERSALS and an only 34% of REPEAT FOLLOWING ERRORS (see Table 3). Thus, the difference in performance between REPEAT FOLLOWING CORRECT trials and either of the other two types of trials was 45% even though procedures were identical on all trials. These differences are significant at $p = 0.0001$ (REPEAT FOLLOWING CORRECT vs. REVERSAL: $t = 12.59$, $p = 0.0001$, matched pairs comparison; REPEAT FOLLOWING CORRECT vs. REPEAT FOLLOWING ERROR: $t = 14.20$, $p = 0.0001$, matched pairs comparison).

Note that these large effects result from a change in only one variable. In REPEAT FOLLOWING CORRECT trials, the infant was correct on the preceding trial, and side of hiding is repeated. REVERSALS differ only in that side of hiding is reversed; REPEAT FOLLOWING ERROR trials differ only in that the infant erred on the preceding trial. The conditional probability of success changed markedly when either of these two variables

Table 3. Percentage of correct reaches by type of trial and sex

		Repeat following CORRECT		Reversal		Repeat following error	
Girls	Mean	79		33		29	
	Nina	75	(16)	45	(20)	37	(41)
	Sarah	92	(22)	53	(15)	27	(33)
	Jane	79	(20)	20	(20)	26	(31)
	Julia	100	(15)	32	(19)	38	(34)
	Lyndsey	62	(21)	13	(15)	36	(36)
	Jamie	80	(20)	33	(21)	13	(23)
	Rachel	89	(18)	10	(21)	20	(50)
	Erin	82	(22)	11	(18)	26	(35)
	Mariama	67	(15)	56	(18)	30	(33)
	Kate	64	(22)	36	(14)	26	(42)
	Chrissy	82	(17)	35	(17)	26	(38)
	Isabel	80	(20)	60	(20)	30	(30)
	Jennine	77	(18)	30	(20)	57	(23)
	Emily	79	(14)	29	(14)	20	(20)
Boys	Mean	79		36		40	
	Rusty	67	(18)	33	(18)	20	(35)
	Todd	100	(17)	47	(17)	33	(36)
	Tyler	89	(19)	37	(19)	33	(42)
	Brian	64	(25)	27	(15)	53	(34)
	Michael	77	(26)	20	(20)	50	(32)
	Jack	71	(14)	56	(18)	29	(21)
	Ryan	72	(18)	44	(18)	45	(40)
	James	80	(20)	13	(16)	40	(42)
	Graham	100	(12)	72	(18)	75	(20)
	Blair	85	(13)	29	(14)	43	(21)
	Bobby	80	(15)	18	(17)	30	(40)
Grand mean		79		34		34	

Number in parentheses refers to number of trials on which percentage is based

changed, even though each trial, considered in isolation, should pose the same level of difficulty as any other, as procedure and delay were constant across trials. This indicates the profound effect of the context in which a trial is embedded.

Each infant performed more poorly on REVERSALS and on REPEAT FOLLOWING ERROR trials than that same infant performed on REPEAT FOLLOWING CORRECT trials (see Table 3). It is remarkable that errors were so systematically distributed over trials and that every subject showed this same pattern. The $A\overline{B}$ error pattern occurred on 90% of the sessions for infants between 7.5–9 months of age.

General observations. Coders were able to verify from the video tape records that level of interest in the toy was high on 99% of the trials and infants looked directly at where the experimenter hid the toy on 99% of the trials. The experimenters' attempts to make each infant look up during the delay were similarly successful, but infants did not always look up throughout the *entire* delay. Parents were instructed to tightly hold their infant's arms and torso during the delay, but some infants, nevertheless, showed evidence of bodily straining or turning. Effects of visual fixation and bodily orientation were, therefore, investigated. The results show that staring at, turning, straining, or reaching toward the correct well during part, or even most, of a delay did *not* increase the likelihood of a correct reach. However, when the infant's gaze or strain was uninterrupted and *maintained throughout the entire delay*, success rate was significantly higher than on comparable trials where strain or gaze was not thus maintained (Diamond 1983). This is consistent with other reports that infants who maintained visual fixation on the correct well reached correctly, while those who shifted their gaze between the wells performed at chance levels (Gratch and Landers 1971; Cornell 1979; Goldfield and Dickerson 1981; Acredolo et al. 1984). In the present study, uninterrupted straining or staring occurred so rarely that they do not account for any of the effects reported above.

Infants tried to correct themselves when they made the $A\overline{B}$ error. Given the chance, they would reach to the second well if their first reach failed to produce the toy. The only exception to this pattern occurred at the youngest ages. Most infants were not ready for $A\overline{B}$ testing until at least 7.5 months of age, but two of our subjects could uncover a hidden object at 6.5 months and six could do so at 7 months. However, when these subjects were tested on $A\overline{B}$ at these precocious ages, they failed to correct themselves. If their reach did not immediately produce the toy, they acted as if they had forgotten why they had reached in the first place. They showed no interest in uncovering the other well nor in looking around for the toy. On occasion, this "forgetfulness" appeared before any well was uncovered: the infant would spring toward a well as soon as the parent freed his arms, but then while still in the act of reaching the infant would look up and abandon the reach, as if the infant could not remember what he started to do. At 6.5 months we saw no instance of self-correction and at 7 months it occurred after only 12% of the errors. But by 7.5–8 months most infants corrected themselves most of the time, and from 9 months on, self-correction occurred after virtually every incorrect reach when the $A\overline{B}$ error was made. During deteriorated performance, failure to correct themselves was again seen at even the oldest ages. For example, at 9 months self-correction followed 99% of the errors when the $A\overline{B}$ error occurred, but it followed only 75% of the errors during deteriorated performance.

Nonhuman primates

Performance at 2 s delay. Monkeys with prefrontal lesions committed the $A\overline{B}$ error at the 2 s delay. Their behavior looked remarkably similar to that of human infants (see Fig. 3). No animal with parietal lesions and no intact animal committed the $A\overline{B}$ error at the 2 s delay.

Prefrontal animals performed on average at the 63% level, while parietal and unoperated animals performed at the 98% level with no animal in the parietal or unoperated groups performing below 96% correct over all trials (see Table 4). The difference in percent correct for animals with prefrontal lesions and the percent correct for either control group was significant at $p = 0.0001$ ($t = 18.77$ prefrontal vs. parietal; $t = 17.80$ prefrontal vs. unoperated; $N = 3$ for all groups). There was no difference in performance between unoperated monkeys and those who had received parietal lesions.

Performance at 5 s delay. All monkeys with prefrontal lesions committed the $A\overline{B}$ error at the 5 s delay as well, while all monkeys with parietal lesions and all unoperated controls succeeded at this delay.

Prefrontal animals performed on average at the 64% level across all trials. Parietal animals performed at the 98% level, unoperated animals at the 97% level, with no parietal or intact animal performing below 94% correct. The difference between the performance of the animals with prefrontal ablations and the performance of either control group was significant ($t = 18.94$, $p = 0.0001$ prefrontal vs.

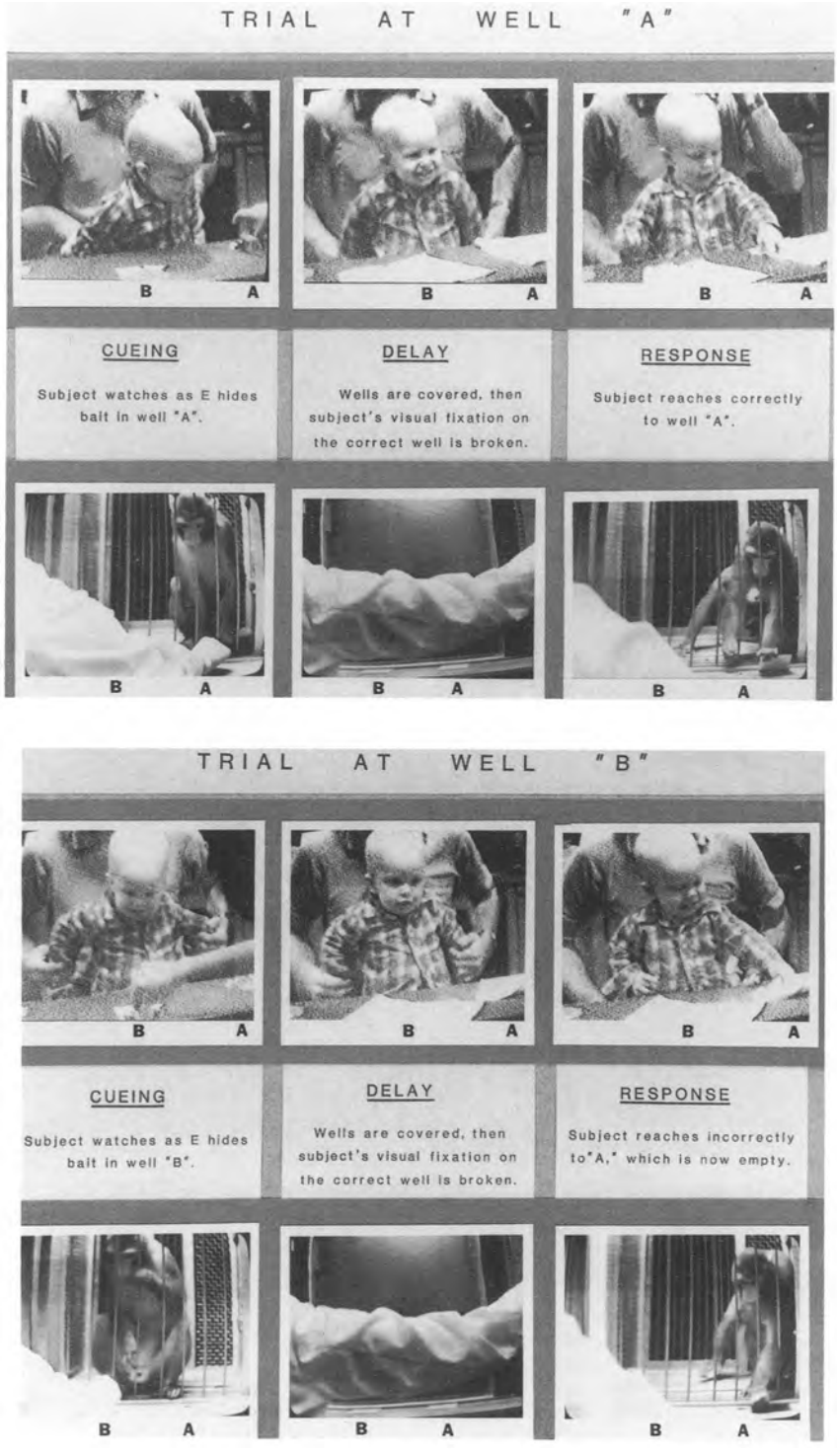

Fig. 3. Comparison of performance of human infant and prefrontally operated rhesus monkey on $A\overline{B}$. Both succeed on the trial at A, but when the bait is then hidden at B, they both reach back to A, even though they both observed the hiding

parietal; t = 23.86, $p = 0.0006$ prefrontal vs. unoperated, logarithmic x arc sine transformation for unequal variances). There was no significant difference in performance between unoperated monkeys and those who had received parietal lesions.

Performance at 10 s delay. Like infants below 9 months, the performance of prefrontal monkeys deteriorated at delays of 10 s so that the criterion for the $A\overline{B}$ error was not met. Their performance even on REPEAT FOLLOWING CORRECT trials was

Table 4. Percent correct by type of trial and delay for each subject in each experimental group

Experimental groups	2 s				5 s				10 s			
	All trials	Repeat following correct	Reversal	Repeat following error	All trials	Repeat following correct	Reversal	Repeat following error	All trials	Repeat following correct	Reversal	Repeat following error
Prefrontal												
F1F	45[a] (311)[b]	76 (96)	32 (59)	37 (172)								
F2F	71 (223)	88 (84)	56 (61)	58 (65)	63 (279)	87 (93)	46 (67)	47 (104)	58 (320)	54 (107)	57 (63)	50(133)
F3F	67 (258)	84 (86)	39 (64)	67 (86)	67 (235)	84 (96)	59 (66)	67 (77)	64 (294)	63 (107)	52 (63)	69(106)
F4M	67 (249)	81 (93)	56 (63)	60 (81)	63 (273)	80 (97)	49 (63)	46 (100)	59 (317)	60 (104)	47 (62)	51(130)
Mean	63 (260)	82 (90)	46 (62)	56 (101)	64 (262)	84 (95)	51 (65)	53 (94)	60 (310)	59 (106)	52 (63)	57(123)
Unoperated												
U1F	99 (178)	100 (94)	99 (69)	100 (1)*	98 (181)	99 (93)	96 (70)	100 (4)*	98 (183)	99 (95)	97 (70)	75 (4)*
U2F	96 (187)	98 (94)	93 (71)	88 (8)*	98 (183)	98 (95)	97 (70)	100 (4)*	96 (185)	96 (94)	99 (70)	86 (7)*
U3M	99 (177)	100 (92)	99 (70)	100 (3)*	96 (181)	98 (93)	96 (70)	83 (6)*	97 (183)	98 (93)	96 (70)	100 (5)*
Mean	98 (181)	99 (93)	97 (70)	96 (4)*	97 (182)	98 (94)	96 (70)	94 (5)*	97 (183)	98 (94)	97 (70)	87 (5)*
Parietally												
P1F	97 (185)	98 (94)	99 (71)	100 (5)*	94 (183)	96 (90)	93 (69)	80 (10)	92 (193)	95 (96)	89 (70)	88(16)
P2M	100 (172)	100 (91)	100 (68)	— (0)*	100 (180)	100 (96)	100 (70)	— (0)*	99 (182)	99 (97)	100 (70)	100 (1)*
P3M	98 (175)	99 (87)	97 (70)	83 (6)*	99 (185)	99 (99)	100 (70)	100 (2)*	98 (185)	99 (97)	96 (70)	100 (4)*
Mean	98 (177)	99 (91)	99 (70)	94 (4)*	98 (183)	98 (95)	98 (70)	93 (4)*	96 (187)	98 (97)	95 (70)	96 (7)*

* Data based on less than 10 trials does not yield a reliable percentage

Note that the average percent correct score for all the trials includes performance on the first trial of each daily session, but performance on this trial does not contribute to the score for any of the three trial types

[a], % correct; [b], no. of trials

near chance; average performance across all trials was 60%. Parietal and unoperated monkeys continued to reach correctly (parietal animals: $\bar{x}$ = 96%; intact animals: $\bar{x}$ = 97%). The difference between the performance of prefrontal monkeys and the performance of the other two groups was significant [t = 16.34, p = 0.001, for prefrontal vs. parietal; t = 20.18, p = 0.0001 for prefrontal vs. unoperated (arc sine transformation for unequal variances)].

It is unlikely that the performance of the prefrontal animals reflects a transient deficit of insufficient practice to permit mastery of the task. All animals were given two weeks of practice before formal testing on $A\overline{B}$ at 2 s. All intact and parietal animals reached correctly at the 2 s delay well before this practice period ended. Over the 14 days of formal testing on $A\overline{B}$ at 5 and 10 s, there was no evidence of improvement in the performance of prefrontal animals (as shown by trend analysis or by matched pairs t-tests of the first sessions vs. the last 4 sessions or the first 7 sessions vs. the last 7). At the 2 s delay, prefrontal subjects improved slightly during the first half of testing (linear regression coefficient = 0.02, p = 0.09), but showed no further improvement at all over sessions 8–14.

Pattern of errors. The pattern of behavior observed during $A\overline{B}$ performance by prefrontally ablated animals was similar in all respects to that observed in human infants. Prefrontal monkeys, like human infants, self-corrected if given the chance, and did not err if they maintained a bodily orientation toward the correct well throughout the delay.

As with human infants, the errors of prefrontal animals were not randomly distributed over trials at delays of 2 and 5 s. These animals tended to be correct if the bait was again hidden where they had just reached correctly. They tended to err on reversal trials and to repeat this error over several trials (see Fig. 4). The following pattern of behavior was observed in prefrontally operated animals with delays of 2–5 s: They reached correctly on 83% of the REPEAT FOLLOWING CORRECT trials, but were correct on only 49% of the REVERSALS and only 55% of the REPEAT FOLLOWING ERROR trials. Just as with human infants, performance on REPEAT FOLLOWING CORRECT trials was significantly better than on any other type of trial (REPEAT FOLLOWING CORRECT vs. REVERSAL: t = 12.22, p = 0.007, matched pairs comparison; REPEAT FOLLOWING CORRECT vs. REPEAT FOLLOWING ERROR: t = 4.76, p = 0.04, matched pairs comparison). Indeed, there was no overlap in scores; no prefrontal monkey performed as well on REVERSALS or on REPEAT FOLLOWING ERROR trials as any prefrontal

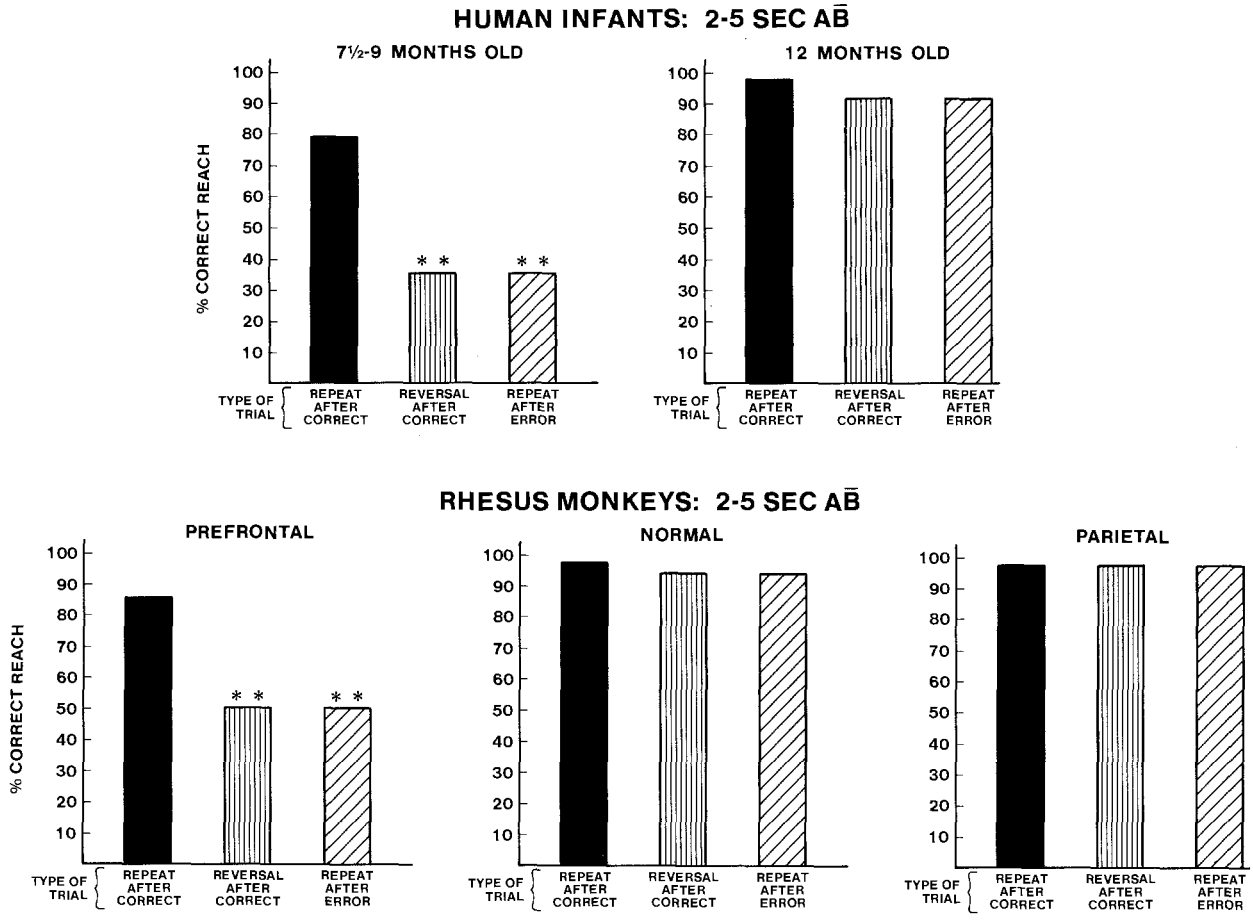

Fig. 4. Pattern of performance on AB̄ by type of trial for human infants and rhesus monkeys

monkey performed on REPEAT FOLLOWING CORRECT trials.

In contrast to the performance of 7.5–9 month old human infants and prefrontal animals, animals in both control groups reached correctly across all types of trials. They showed no significant difference in performance by type of trial (see Fig. 4).

The AB̄ error occurred on most sessions at delays of 2 and 5 s for the prefrontally operated monkeys (64% of the sessions), with performance better on 16% of the sessions and worse on 20%. Control animals, on the other hand, rarely made the AB̄ error (4% of the sessions), showing accurate performance on over 95% of the sessions.

With a 10 s delay, prefrontal animals, like human infants 7.5–9 months, showed *deteriorated performance*. They were upset and showed long perseverative error strings, sometimes refusing to reach at all or making no attempt to self-correct if they were wrong. Human infants indicated their distress by fussing; prefrontal monkeys did so by agitated circling. They performed at chance across all categories of trials (see Table 4). Performance did not differ by type of trial as is characteristic of the AB̄ error, where performance on REPEAT FOLLOWING CORRECT trials is at a high level. The AB̄ error occurred on only 29% of the sessions at the 10 s delay for prefrontal monkeys; most of their sessions at 10 s were characterized by deteriorated performance.

Control animals performed well on all types of trials even at the 10 s delay. No significant difference in performance by type of trial was found at any delay for either parietal or unoperated subjects. Their performance remained accurate on 87% of the sessions at the 10 s delay.

Comparison of AB̄ and delayed response performance

Two animals in each experimental group had training on Delayed Response prior to AB̄ testing. One animal in each lesion group had additional training on Delayed Response post-operatively prior to AB̄ testing.

Table 5.

Groups	Pre-operative testing on Delayed Response	Post-operative testing on Delayed Response	Pre-operative testing on $A\overline{B}$	Post-operative testing on $A\overline{B}$
Prefrontally operated monkeys				
F2F	tested on 2–120 s passed 2–120 s	—	—	fails even 2 s
F4M	tested on 2– 10 s passed 2– 10 s	fails even 2 s	—	fails even 2 s
Parietally operated monkeys				
P1F	tested on 2 s passed 2 s	—	—	tested on 2–10 s passed 2–10 s
P2M	tested on 2–120 s passed 2–120 s	tested on 2–10 s passed 2–10 s	—	tested on 2–10 s passed 2–10 s
Unoperated monkeys				
U1F	tested on 2–10 s passed 2–10 s	—	tested on 2–10 s passed 2–10 s	—
U3M	tested on 2–10 s passed 2–10 s	—	tested on 2–10 s passed 2–10 s	—

One animal in the frontal group, F2F, was tested on Delayed Response with delays from 2–120 s pre-operatively, and had reached criterion of 90% correct at all delays. After surgery, F2F failed Delayed Response at even a 2 s delay. Another frontal animal, F4M, was tested on Delayed Response at delays of 2–10 s pre-operatively and had reached criterion at all delays. After prefrontal ablation, however, F4M failed to pass Delayed Response at even a 2 s delay. This animal had reached stable 90% performance on Delayed Response (2 s delay) by testing day 4 prior to surgery, but failed to pass Delayed Response (2 s delay) even after 12 days of testing after surgery. This is consistent with the post-operative performance of F4M on $A\overline{B}$. This animal also failed to pass the task even at the 2 s level (see Table 5).

One parietal animal, P1F, was tested on Delayed Response with a 2 s delay prior to surgery and passed criterion. Another parietal animal, P2M was tested on Delayed Response with delays on 2–120 s pre-operatively and reached criterion at all delays. This same animal was tested after parietal ablation on Delayed Response with delays of 2–10 s and passed criterion at all delays. When P2M was first tested on Delayed Response (2 s delay) prior to surgery, he did not consistently perform at the 90% level until testing day 7. After surgery, P2M performed Delayed Response (2 s delay) at the 90% level by the second day of testing. This is consistent with P2M's post-operative performance on $A\overline{B}$, which was successful at delays of 2–10 s.

One unoperated animal, U3M, was tested on Delayed Response with delays up to 20 s prior to $A\overline{B}$ testing. Another intact animal, U1F, was tested on Delayed Response with delays up to 10 s prior to $A\overline{B}$ testing. Both animals passed Delayed Response at all delays. Similarly, they passed $A\overline{B}$ at all delays.

Thus, all subjects performed at high levels on preoperative Delayed Response testing. This indicates that the animals chosen for prefrontal ablation were comparable in ability to the control animals as measured by performance on Delayed Response prior to surgery. Prefrontally operated subjects succeeded on pre-operative Delayed Response testing at delays as long or longer than the delays at which they later failed post-operatively.

Discussion

The present study had two main goals: (1) to determine whether performance on Piaget's $A\overline{B}$ task depends upon dorsolateral prefrontal cortex in rhesus monkeys, and (2) to compare the performance of human infants on $A\overline{B}$ with that of operated and intact rhesus monkeys. We reasoned that these comparisons would shed light on the possibility that

$A\overline{B}$ performance in humans depends upon prefrontal cortex and that maturation of this region underlies the improved performance on $A\overline{B}$ seen between 7.5–12 months of age in human infants.

$A\overline{B}$, like Delayed Response, depends upon prefrontal cortex in the rhesus monkey

The present study provides the first evidence that $A\overline{B}$ performance depends upon the integrity of dorsolateral prefrontal cortex and requires the same abilities and underlying neural mechanisms as does Delayed Response. In the present study, we found that lesions of dorsolateral prefrontal cortex severely disrupted performance of $A\overline{B}$. Equally large lesions of parietal cortex did not produce this effect.

We had predicted that $A\overline{B}$ performance would depend upon dorsolateral prefrontal cortex given $A\overline{B}$'s marked similarity to Delayed Response, and Delayed Response's well established reliance on dorsolateral prefrontal cortex. Within-trial procedures are identical for Delayed Response and $A\overline{B}$. The only difference between the two tasks is that the site of hiding varies randomly in Delayed Response and regularly on $A\overline{B}$, dependent upon the subject's response.

Results were entirely comparable for the two tasks. The prefrontal animal tested on Delayed Response and $A\overline{B}$ failed both at delays of 2–10 s, even though that same animal had passed Delayed Response pre-operatively at delays ten times as long. The parietal and unoperated animals tested on both Delayed Response and $A\overline{B}$ performed above the 90% level on both at delays of 2–10 s.

Further confirmation that Delayed Response and $A\overline{B}$ require the same abilities comes from findings that (a) human infants show the same developmental progression over the same ages on Delayed Response as they do on $A\overline{B}$ (Diamond and Doar, in prep.), and (b) infant monkeys exhibit the same developmental progression over the same ages on $A\overline{B}$ as they do on Delayed Response (Diamond and Goldman-Rakic 1986).

The present results for Delayed Response are fully consistent with those in previous studies. When parietal animals have been tested on Delayed Response in the past, no deficit has ever been observed (Jacobsen 1936; Meyer et al. 1951; Harlow et al. 1952). Monkeys with lesions of dorsolateral prefrontal cortex, on the other hand, have repeatedly been shown to fail Delayed Response at delays of even 2 s (Harlow et al. 1952; Battig et al. 1960; Goldman et al. 1970; Fuster and Alexander 1971). Moreover, they fail even if they have had pre-operative training on Delayed Response (e.g., Gross and Weiskrantz 1962), just as we found. Finally, our observations that prefrontally operated animals succeed if they are allowed to keep looking at, or orienting their body toward, the correct well during the delay are in accord with previous reports (Fulton and Jacobsen 1935; Battig et al. 1960; Kojima et al. 1982; Miles and Blomquist 1960; Pinsker and French 1967).

Indeed, the dependence of Delayed Response on dorsolateral prefrontal cortex has been demonstrated in scores of lesion studies (for reviews see Fuster 1980; Goldman-Rakic 1987), and further confirmed by a variety of techniques which temporarily and reversibly interrupt prefrontal function: localized cooling (Fuster and Alexander 1970; Bauer and Fuster 1976; Alexander and Goldman 1977), localized electrical stimulation (Weiskrantz et al. 1962; Stamm 1969; Stamm and Rosen 1969), and pharmacological manipulations (Arnsten and Goldman-Rakic 1985; Brozoski et al. 1979). Delayed Response has also been shown to depend upon dorsolateral prefrontal cortex by techniques which assess patterns of functioning in the intact brain: electrophysiological recording (e.g., Fuster and Alexander 1971; Fuster 1973; Niki 1974; Niki and Watanabe 1976; Kojima and Goldman-Rakic 1982) and 2-deoxyglucose metabolic mapping (Bugbee and Goldman-Rakic 1981).

Moreover, this dependence of Delayed Response on dorsolateral prefrontal cortex is selective. Other tasks, such as Visual Discrimination, which require associative learning are not affected by lesions of dorsolateral prefrontal cortex (Goldman 1971; Harlow and Dagnon 1943; Jacobsen 1935, 1936; Passingham 1972).

Developmental progression on $A\overline{B}$ in human infants

The results with human infants showed that the $A\overline{B}$ error occurs at delays of 2–5 s at 7.5–9 months, and at delays greater than 10 s after one year. These results accord well with the findings of others. Longitudinal studies of $A\overline{B}$ performance in human infants have also been conducted by Gratch and Landers (1971) and Fox et al. (1979). Both studies found that infants of 8 months made the $A\overline{B}$ error at a delay of 3 s, exactly as reported here. Also in agreement is the study by Millar and Watson (1979) which demonstrated that infants of 6–8 months could acquire a conditioned response if the delay between response and reinforcement was 0 s, but not if it were 3 s. This corresponds closely to the finding that infants of 8 months will succeed on $A\overline{B}$ if the delay between hiding and response is 0 s, but not if the delay is 3 s.

The progression of $A\overline{B}$ performance with age is not attributable to practice, as results from cross-sectional studies attest. For example, infants tested for the first time at 9 months make the $A\overline{B}$ error at delays of 3–7 s (Evans and Gratch 1972; Gratch et al. 1974; Butterworth 1977), just as the 9 month olds did who were repeatedly tested here. The same developmental progression has been found across social class lines (e.g., lower class: Gratch and Landers 1971; upper middle class: Diamond 1985). This commonality across children with diverse experiences is consistent with the importance of a maturational component to improved performance on $A\overline{B}$ with age.

Results for prefrontal monkeys on $A\overline{B}$ and Delayed Response are similar to those for human infants

The $A\overline{B}$ performance of prefrontally operated monkeys was comparable in every way to the $A\overline{B}$ performance of 7.5–9 month old human infants. Prefrontal monkeys and 7.5–9 month old human infants made the $A\overline{B}$ error at delays of 2–5 s. Both groups performed at high levels on REPEAT FOLLOWING CORRECT trials while performing at low levels on REVERSALS and REPEAT FOLLOWING ERROR trials, even though the delay was the same on all trials. Both groups tried to self-correct following an error. The performance of prefrontal monkeys and 7.5–9 month old human infants deteriorated further when the delay was increased to 10 s, where they ceased to perform well even on REPEAT FOLLOWING CORRECT trials.

There have been no other studies of $A\overline{B}$ performance in the adult rhesus monkey, intact or lesioned, with which to compare our results. The closest approximation was a study by Anderson et al. (1976) who explicitly tried to assess Piagetian Object Permanence in unoperated and frontally operated adult rhesus monkeys. Their task differed from $A\overline{B}$, however, in that they used a random sequence of hiding (like Delayed Response, not $A\overline{B}$), three hiding places rather than two, inverted wicker baskets rather than wells, and no delay. They found that prefrontally lesioned animals showed a significant impairment on their task, but that this deficit was not lasting. Given the absence of delay or distraction, recovery is not surprising.

The performance of prefrontally operated animals on Delayed Response is comparable in all respects to the performance of 7.5–9 month old human infants on $A\overline{B}$. Human infants succeed at $A\overline{B}$ and monkeys with dorsolateral prefrontal cortex lesions succeed at Delayed Response when there is no delay (*infants*: Harris 1973; Gratch et al. 1974; Diamond 1983. *monkeys:* Harlow et al. 1952; Battig et al. 1960; Goldman et al. 1970; Fuster and Alexander 1971). Human infants below 9 months fail $A\overline{B}$ and prefrontally ablated monkeys fail Delayed Response when a delay is introduced, even if it is as brief as 1–5 s (*infants:* Evans 1973; Gratch et al. 1974; Diamond 1985. *monkeys*: Harlow et al. 1952; Battig et al. 1960; Goldman et al. 1970; Fuster and Alexander 1971). This is true whether the hiding places differ in left-right position (*infants*: Gratch and Landers 1971; Diamond 1985. *monkeys*: Harlow et al. 1952; Goldman et al. 1970) or up-down location (*infants*: Butterworth 1976. *monkeys*: Fuster 1980).

Human infants of 7.5–9 months on $A\overline{B}$ and prefrontally operated monkeys or chimpanzees on Delayed Response succeed when they are allowed to keep looking at, or orienting their body toward, the correct well throughout the delay (*infants*: Cornell 1979; Fox et al. 1979; Diamond 1985. *non-human primates:* Fulton and Jacobsen 1935; Battig et al. 1960; Miles and Blomquist 1960; Pinsker and French 1967). However, if a distractor is introduced, or if the subject spontaneously shifts orientation, then performance falls to chance levels, even if the disturbance is only momentary (with *infants*: Diamond 1985. With *non-human primates*: Fulton and Jacobsen 1935).

Effects of brain injury on $A\overline{B}$ and Delayed Response in humans

Human adults with damage confined to dorsolateral prefrontal cortex have never been tested on $A\overline{B}$. However, amnesic adults with frontal lobe signs have been tested on tasks modelled after $A\overline{B}$ (Schacter et al. 1986)[4]. These patients made extensive perseverative errors on the Wisconsin Card Sort, indicating that they may well have had damage of dorsolateral prefrontal cortex (Milner 1964). Like human infants and monkeys with dorsolateral prefrontal lesions, these patients were as likely to err when the object was uncovered as when it was covered. Human infants make very few errors when there are no covers (Butterworth 1977). These latter errors may have been due to the fact that the patients had both amnesia and frontal lobe pathology. Schacter et al.

4 It should be mentioned that the tasks used by Schacter et al. differed from $A\overline{B}$ in some potentially important ways. The delay was 150 s, rather than 0–10 s. Infants or monkeys are tested on $A\overline{B}$ at a testing table containing two identical embedded wells in a laboratory room without distractions. In Schacter et al.'s "Room Search" task the object was hidden somewhere in a room rich in objects and landmarks. The "Container Search" task took place in the same room and the object was hidden in one of four drawers, each drawer of a different color

also tested adults with damage to medial frontal cortex. They succeeded on the A$\overline{B}$-like tasks and perseverated less on the Wisconsin Card Sort. This is consistent with *dorsolateral* prefrontal cortex as the critical neural locus for A$\overline{B}$ and Wisconsin Card Sort performance.

Human adults with frontal cortex damage have been tested on Delayed Response (Freedman and Oscar-Berman 1986). Patients with frontal damage, confirmed by CT scans, performed more poorly on Delayed Response than amnesic patients or alcoholic controls. Performance on Delayed Response was correlated with performance on the Wisconsin Card Sort, as it should be if both are measures of dorsolateral prefrontal function. It should be noted that the amnesic subjects used here had Korsakoff's syndrome or aneurysms of the anterior communicating artery, conditions associated with frontal lobe signs – yet these patients performed quite well on Delayed Response. This contrasts with the results of Schacter et al. (1986) who found that amnesics with frontal lobe signs failed their A$\overline{B}$-like tasks. The difference in results may be due to the difference in length of delay. Schacter et al. used a 150 s delay, while Freedman and Oscar-Berman used delays of "0"[5], 10, 30, and 60 s, summing the results over all delays. Damage of the temporal lobe system implicated in amnesia is associated with deficits at long delays but not at short ones (see Squire and Zola-Morgan 1983; Diamond 1988). Thus, the 150 s delay used by Schacter et al. may have taxed the fragile memory of the amnesics, while the shorter delays used by Freedman and Oscar-Berman may not have taxed their memory.

Abilities which depend upon dorsolateral prefrontal cortex required for success on A$\overline{B}$ and Delayed Response

Spanning a temporal separation. A$\overline{B}$ and Delayed Response require the subject to relate two temporally separated events: cue and response. When there is no delay between hiding and retrieval even 7.5–9 month old human infants and prefrontally operated monkeys succeed. However, when a delay is introduced, even as brief as 2–5 s, 7.5–9 month olds and prefrontal monkeys fail. Indeed, within a single session, infants reach correctly during half the session and make the A$\overline{B}$ error during the other half if the delay is simply changed by 2–3 s, holding everything else constant (Diamond 1985). Clearly, the delay aspect of the task is crucial.

Human infants and prefrontally operated monkeys perform well if allowed to circumvent the effects of delay by maintaining visual fixation of, or bodily strain toward, the correct well. Similarly, if a visible cue consistently indicates the correct choice, prefrontal monkeys succeed even when side of hiding is reversed (Pohl 1973) and 7.5–9 month olds succeed on reversals even at long delays (Diamond 1983). Here, the subject does not need to keep track of where the bait has been hidden on each trial and hold that in short-term memory. The subject need only look for the visible landmark.

The work with a visible cue or landmark also helps to clarify the aspect of memory required by A$\overline{B}$ and Delayed Response, for memory is required in the landmark condition as well. The subject must remember the association between the landmark and the correct well. Once this association is learned, however, that single piece of information can guide reaching on all trials. Human infants of 7.5–9 months are capable of remembering this type of learned or conditioned association over periods much longer than 2–5 s, indeed over days and weeks (Fagen and Rovee-Collier 1982; Rovee-Collier 1986). Human infants of this age, however, cannot learn the conditioned association if the delay between response and reinforcement is 3 s (Millar and Watson 1979), just as they do not reach correctly when the delay between hiding and response is 2–5 s in A$\overline{B}$ or Delayed Response. It should be emphasized that the delays needed to produce errors on A$\overline{B}$ or Delayed Response in 7.5–9 month olds or prefrontal monkeys are very brief. Long-term memory is not required to span a separation of 2–5 s; short-term working memory is involved (see Goldman-Rakic, 1987 for further discussion of this issue).

Inhibiting the prepotent response. In A$\overline{B}$, the bait is first hidden at A and the subject is rewarded for reaching there. Thus, the tendency to reach to A is strengthened. This conditioned tendency or "habit" to reach to A must be inhibited when the bait is hidden at B, if the subject is to reach correctly.

A mechanism such as this is needed to account for the pattern of errors shown on A$\overline{B}$ by human infants and prefrontal monkeys, whose performance varies systematically by type of trial. Delay is held constant across all trials, yet errors are *not* equally likely on all trials. Human infants and prefrontally operated monkeys err on REVERSALS and on REPEAT FOLLOWING ERROR trials. They reach correctly, though, when inhibitory control is not required, i.e., on REPEAT FOLLOWING CORRECT trials.

5 After the covering of the wells, a curtain was quickly lowered and rasied between the wells and the subject. Thus, the "0" s delay was probably at least 1–2 s long

Indeed, infants sometimes reach back to A even when they appear to know the toy's location. This is consistent with difficulty inhibiting the habitual response, and suggests the problem is not simply forgeting. For example, errors sometimes occur when the toy is *visible* at B, as when the covers are transparent, and occasionally when there is no cover at all (Harris 1974; Butterworth 1977; Neilson 1982; Bremner and Knowles 1984). Even when the toy is hidden, human infants and prefrontal monkeys immediately correct themselves if their initial reach is incorrect. Often, infants will uncover A, *not look in*, then reach immediately to B and retrieve the toy (Diamond 1985). It is as if they know the toy is at B even though they reach first to A. Most telling, infants occasionally look directly at B as their hand reaches to A. They uncover A while they are looking at B. If visual fixation were the dependent measure, the infants would be scored as correct on such trials (Diamond, in press).

This latter behavior is reminiscent of that seen with the Wisconsin Card Sort, the classic test for frontal lobe function in human adults: After having been rewarded for sorting the cards by one criterion, patients with damage to the frontal lobe have difficulty sorting the cards by a new rule. However, these patients can sometimes tell you the new rule as they continue to sort the cards incorrectly. Indeed, they sometimes say as they are sorting the cards by the old criterion, "This is wrong, and this is wrong..." (Luria and Homskaya 1964; Milner 1964; Nauta 1971). Infants cannot tell you verbally, but looking at A even as they reach to B may be the non-verbal equivalent. The ability to resist the strongest response of the moment endows humans and other higher organisms with extraordinary flexibility and freedom to choose and control their actions. It gives us the option of not being solely creatures of habit.

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