Abilities and Neural Mechanisms Underlying A$\bar{B}$ Performance

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DIAMOND, ADELE. *Abilities and Neural Mechanisms Underlying A$\bar{B}$ Performance.* CHILD DEVELOPMENT, 1988, **59**, 523–527. Schacter, Moscovitch, Tulving, McLachlan, and Freedman propose that infants may make the A$\bar{B}$ error because of immaturity of the memory system damaged in amnesia (e.g., the hippocampus). They contrast this with the proposal that infants may make the A$\bar{B}$ error because of immaturity of the frontal lobe system (Diamond; Diamond & Goldman-Rakic). Schacter et al.'s choice of subjects, however, did not permit a test of these 2 proposals, and characteristics of their task, such as length of delay, make comparison with infants difficult. Schacter et al. discuss sensitivity to proactive interference as a possible explanation for the A$\bar{B}$ error, but sensitivity to PI is more closely associated with frontal lobe damage than with amnesia. Schacter et al. associate perseveration with immaturity or damage to the frontal lobe; it is suggested here that this is better characterized as lack of inhibitory control. Tasks that are most likely to require frontal cortex function are those that demand both short-term memory and inhibitory control. A$\bar{B}$ is an excellent example of such a task.

In their article, "Mnemonic Precedence in Amnesic Patients: An Analogue of the A$\bar{B}$ Error in Infants?", Schacter, Moscovitch, Tulving, McLachlan, and Freedman (1986) propose that insights into why infants make the A$\bar{B}$ error can be gained by examining the errors of brain-damaged adults on similar tasks. This is an excellent strategy, but the article is misleading in some ways.

In the A$\bar{B}$ task, an infant watches as a toy is hidden in one of two identical wells, a delay of 0–10 sec is imposed, then the infant is allowed to reach. Infants of 7½–11 months usually find the toy at the first well in which it is hidden (A), but when side of hiding is reversed to B, they reach back to A (Diamond, 1985; Gratch & Landers, 1971). Hence, the name "A, not B." In the Schacter et al. tasks an object was hidden somewhere in a room rich with landmarks ("Room Search") or in one of four drawers differing in both color and location ("Container Search"). A delay of 2½ min (150 sec) filled with conversation was imposed before retrieval was permitted.

Schacter et al. found that subjects with amnesia were correct at A, but not at B. Matched controls and subjects with frontal lobe damage were correct at both. Schacter et al. thus suggest that the brain structures impaired in amnesia (temporal lobe structures, such as the hippocampus) may underlie successful A$\bar{B}$ performance in infants. They contrast this with the hypothesis that the frontal lobe may underlie successful performance on A$\bar{B}$ (Diamond, 1985, in press; Diamond & Goldman-Rakic, 1983).

Schacter et al.'s choice of subjects, however, did not permit a test of the effects of amnesia versus frontal lobe pathology, their choice of delay makes comparison with infants difficult, and their discussion of the deficits associated with amnesia and with frontal lobe damage may lead to some misunderstandings.

Choice of Subjects

All of the amnesic patients studied by Schacter et al. had signs of frontal lobe damage, as is often the case with Alzheimer's disease or aneurysms of the anterior communicating artery. It is possible to gather amnesic patients free of frontal lobe damage (e.g., patients such as NA or HM, who have had focal injuries or focal surgery). It would have been preferable to use such patients.

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Schacter et al. recognized this problem, pointing out that because their patients showed deficits characteristic of both amnesia *and* frontal lobe damage the performance of these patients might be attributable to amnesia, to frontal lobe pathology, or to a combination of the two. To try to eliminate one of these possibilities, Schacter et al. tested three patients with frontal lobe damage. Frontal cortex is a large area, however, comprising fully 25% of the cortex of the human brain. Schacter et al.'s frontal patients had damage primarily in *medial* rather than in dorsolateral frontal cortex. Yet, it is damage to the *dorsolateral* region of frontal cortex that is associated with $A\bar{B}$ errors in the rhesus monkey (Diamond & Goldman-Rakic, 1983) and with perseverative errors on the Wisconsin Card Sort (WCST) in human adults (Milner, 1964). Damage to medial portions of the frontal lobe does not produce these impairments.

Schacter et al.'s amnesic subjects failed the WCST, indicating that they may well have had damage to dorsolateral frontal cortex. In fact, they perseverated more on the WCST than did subjects with frontal damage. These results must be viewed with caution, however, because even the matched controls failed the WCST.

In short, Schacter et al. found that adults with both amnesia and pathology characteristic of dorsolateral frontal damage made $A\bar{B}$-like errors on tasks similar to $A\bar{B}$, and that adults with medial frontal damage did not. These results do not help to distinguish between interpretations of $A\bar{B}$ emphasizing dependence on the neural system implicated in amnesia and interpretations emphasizing dependence on dorsolateral frontal cortex.

Length of Delay

A memory interpretation of $A\bar{B}$ need not imply involvement of the hippocampus. It could imply involvement of the frontal lobe, but the length of delay requiring frontal lobe function is shorter than that requiring hippocampal function.

Any interpretation of $A\bar{B}$ must take into account that the delay aspect of the task is critical: when there is no delay errors are rare (Diamond, 1985; Gratch & Landers, 1971). Infants perform perfectly when delay is reduced 2–3 sec below the level for the $A\bar{B}$ error (Diamond, 1985). Infants also perform well if allowed to circumvent the effects of delay by maintaining visual fixation of, or a bodily strain toward, the correct well (Cornell, 1979; Diamond, 1985; Fox, Kagan, & Weiskopf, 1979). They make fewer errors with transparent than with opaque covers and almost no errors when the toy is visible and uncovered (e.g., Butterworth, 1977). (Note that Schacter et al.'s amnesic subjects erred as often when the object was visible and uncovered [seven out of eight subjects] as they did when the object was hidden [six out of eight]. This performance is very different from that of infants and suggests that different underlying mechanisms may have been involved.)

Although a delay seems to be required for the $A\bar{B}$ error, its length is extremely brief. Infants of roughly 7½–9 months make the $A\bar{B}$ error at delays of 2–5 sec (Diamond, 1985; Gratch & Landers, 1971; Fox et al., 1979). At longer delays, for example, at 10 sec, they do not show the $A\bar{B}$ error pattern but reach randomly, failing even the trials at A (Diamond, 1985). That is, at delays over 10 sec, the $A\bar{B}$ error pattern is not found in 7½–9-month-old infants.

There is no evidence linking damage of the hippocampal system to errors at delays under 10 sec. In contrast, there is substantial evidence linking damage of the frontal lobe system to errors at delays under 10 sec on $A\bar{B}$ and on a closely related test, Delayed Response (DR).

Monkeys with lesions of dorsolateral prefrontal cortex reach correctly on $A\bar{B}$ when there is no delay. They make the $A\bar{B}$ error, however, at delays of 2–5 sec. At 10 sec they do not show the $A\bar{B}$ error, failing even the trials at A (Diamond & Goldman-Rakic, 1983, 1986). Here, as with infants, the presence of a delay is crucial, but the delay for the $A\bar{B}$ error is brief.

DR is very similar to $A\bar{B}$, and the findings parallel those with $A\bar{B}$: monkeys with lesions of dorsolateral prefrontal cortex succeed on DR when there is no delay but fail with delays of 2–5 sec (e.g., Battig, Rosvold, & Mishkin, 1960; Fuster & Alexander, 1971; Goldman & Rosvold, 1970). If allowed to maintain visual fixation of, or a bodily strain toward, the correct well during the delay, they perform perfectly (Battig et al., 1960; Fulton & Jacobsen, 1935).

Many memory tasks are sensitive to hippocampal function in the monkey, but no deficit on any task has ever been found with delays as brief as 2–5 sec following lesions of the hippocampus or hippocampus-amygdala. Indeed, delays of at least 120 sec are sometimes needed (e.g., Mishkin, 1978).

Monkeys with lesions of the hippocampus or the hippocampus plus amygdala *succeed* on DR at delays of 0–10 sec (for review see Squire & Zola-Morgan, 1983). Only when delays become longer, 15–30 sec or more, are deficits found (Zola-Morgan & Squire, 1985). The results for $A\overline{B}$ are similar. Monkeys with lesions of the hippocampus reach correctly on $A\overline{B}$ at delays of 2–10 sec. At 15 sec their performance starts to decline and at 30 sec they make a significant number of errors. They never show the $A\overline{B}$ error pattern, where errors are confined to only certain types of trials (reversals and repeat trials following errors) (Diamond, Zola-Morgan, & Squire, 1987).

It is important, therefore, that Schacter et al. used a delay of 150 sec. Short delays are often too easy for adults, even adults with severe brain damage; hence one can see why a long delay was used. Schacter et al. noted that their delay was long but did not elaborate on why this might be a problem. The long delay makes interpretation difficult. It could change the task from one dependent upon the frontal lobe to one dependent on the hippocampus.

Proactive Interference

Schacter et al. (1986, p. 822) suggest that sensitivity to proactive interference may be one of the reasons for the $A\overline{B}$ error, and that sensitivity to proactive interference is characteristic of amnesics. While sensitivity to proactive interference can account for many of the findings with $A\overline{B}$, it is only the subgroup of amnesics who have pronounced frontal lobe signs, for example, Korsakoff amnesics, who are sensitive to proactive interference (Moscovitch, 1982; Squire, 1982). (Studies cited by Schacter et al., such as Kinsbourne & Winocur, 1980, and Winocur & Weiskrantz, 1976, included only Korsakoff amnesics.) Indeed, Moscovitch (1982) has elegantly shown that amnesics without frontal symptoms show normal release from proactive interference, whereas frontal patients without amnesia are abnormally sensitive to proactive interference. Thus, sensitivity to proactive interference is a frontal sign, found in patients with frontal lobe damage whether they are amnesic or not, but not found in amnesic patients without frontal lobe damage.

Perseveration

In distinguishing their interpretation from that linking $A\overline{B}$ to the frontal lobe, Schacter et al. state, "[Diamond and Goldman-Rakic (1983)] proposed that the $A\overline{B}$ error is attributable to perseverative tendencies associated with poorly developed frontal lobes in infants" (1986, p. 820). Although Diamond and Goldman-Rakic propose that the $A\overline{B}$ error in infants is attributable to poorly developed frontal lobes, they have argued *against* perseveration as an interpretation of frontal lobe pathology. Diamond has proposed that success on $A\overline{B}$ requires two abilities: (*a*) the ability to span a temporal separation (i.e., memory or attention), and (*b*) inhibition of the prepotent response (Diamond, 1985, in press; Diamond & Goldman-Rakic, 1985). A deficit in inhibitory control can be manifest as perseveration, but perseveration is the result, not the cause. Indeed, when the prepotent response is different from the response the subject has been making, lack of inhibitory control is manifest as a *failure* to perseverate.

The following example may help clarify this distinction: Infants of 7–8 months, who are making the $A\overline{B}$ error, fail to perseverate on a transparent-barrier task. Here, reaching straight to a visible goal is the predominant response. When they see a toy through the open front of the clear box they successfully retrieve it. After three such trials, if the box is moved forward 1 inch, and the toy back ½ inch (so the toy is now visible through the box top), infants reach to the closed top, despite three consecutive success experiences at the front (Diamond, 1981). Here, perseveration would lead to success, but infants fail to perseverate because they are unable to inhibit the predominant pull of the visible goal.

The proposal that success on $A\overline{B}$ requires both memory and inhibitory control can explain findings not readily accounted for by interpretations of $A\overline{B}$ that emphasize memory alone. (1) Some infants err on trials at B with transparent covers, and a few err when there are no covers at all. Here, memory is not taxed, but inhibitory control is. It is consistent with the memory/inhibitory control explanation that such errors occur, and also that such errors are less frequent than those that occur when both abilities are taxed. (2) Errors are not distributed equally across trials although delay is held constant. Infants and prefrontally operated monkeys are correct when inhibitory control is not required, as on the first hiding or when the hiding is repeated where they just reached correctly on the previous trial. They err on reversals and on repeat trials following errors. Since delay is the same across trials, a factor other than memory (e.g., inhibitory control) is needed to account for differential performance across types of trials.

(3) The results with multiple wells are often taken as evidence for a memory interpretation because infants do not reach back specifically to A, as they would if there were something special about A. However, a memory interpretation should predict errors randomly distributed around B, and that is not always found. The memory/inhibitory control interpretation predicts that errors should always be in the direction of A, rather than on the side of B away from A, because infants are thought to have difficulty fighting the tendency to reach back to A; this is what is found. (The tendency to reach back to A should deflect reaches toward A. The reach need not be specifically at A, or even nearer to A than to B, but the reach should never be deflected to the side of B away from A.) In one experiment with multiple wells, infants were allowed to reach to either side of B (Cummings & Bjork, 1983). They used six wells; A was well 2 and B was well 5. No infant reached to well 6 on the reversal trial (the well on the side of B away from A), although 65% of the infants erred. All infants erred by reaching to the side of B toward A. It is hoped that future studies will explore this further with more wells on the side of B away from A and with the wells arranged in a semicircle rather than in a straight line to offset the preference to reach toward the midline.

Frontal cortex and the hippocampus are interconnected, but their functions are dissociable. Dorsolateral frontal lesions in the monkey produce errors on $A\bar{B}$ at 2–5 sec but do *not* impair performance on tasks that require memory but not also inhibition, such as Delayed Non-Match to Sample. Lesions of the monkey hippocampus do *not* produce errors on $A\bar{B}$ at 2–5 sec, but at longer delays they produce errors on both Delayed Non-Match to Sample and $A\bar{B}$, although they never produce the $A\bar{B}$ error pattern. That pattern seems to reflect problems in both memory and inhibitory control.

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