

## DEDICATION

I would like to dedicate this book, with respect and affection, to Jerome Kagan and Hens Kuypers.

Jerome Kagan was my graduate advisor at Harvard University. It was his conviction that some maturational change must be related to the behavioral advances seen in babies between 6–12 months that got me started in the line of research I am still pursuing. Jerry Kagan was one of the first developmental psychologists to appreciate that neuroscience and child development might have something to say to one another, and that such a dialogue might enrich both fields. His support, and his belief in me, made all the difference in the world in the early years of my research. It is a great pleasure to acknowledge my gratitude and affection for him.

H. G. J. M. Kuypers (1925–1989) was a pioneering neuroanatomist and behavioral neuroscientist and a lovely person. He was as comfortable, and as knowledgeable, at the level of behavior as at the level of anatomy and was equally curious about motor, perceptual, and cognitive processing. The meeting whose proceedings are presented here was one of the last scientific events Hens Kuypers attended. He will be remembered fondly by all conference participants and deeply missed by the larger neuroscience community.

—ADELE DIAMOND

## THE DEVELOPMENT AND NEURAL BASES OF HIGHER COGNITIVE FUNCTIONS

*Edited by Adele Diamond*



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## THE DEVELOPMENT AND NEURAL BASES OF HIGHER COGNITIVE FUNCTIONS<sup>a</sup>

Editor  
ADELE DIAMOND

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## Introduction

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This volume presents the papers and discussions from a meeting at Sugarloaf Conference Center in Philadelphia, Pennsylvania (May 20–24, 1989) that brought together developmental psychologists who are studying particular aspects of cognition, and neuroscientists who are studying the neural bases of those very same aspects of cognition.

One of the ways in which this gathering of people was unusual is that scientists in diverse fields *studying the same observable behaviors* were assembled. Often without realizing it, scientists in one field had been using the same experimental paradigm to study behavior as scientists in a remote field. These observable behaviors formed a bridge, enabling investigators studying development, cognition, and neurobiology to talk to one another, often for the first time. The behaviors, and the precise experimental conditions under which those behaviors occurred, provided the common denominator for translating from the language of one field to the vocabulary of others, and for relating the findings from one field to those from other fields.

Topics as diverse as spatial, temporal, and intersensory integration are covered, drawing on work from diverse species, and integrating findings from neuroanatomy, neurochemistry, neurophysiology, neuropsychology, cognitive science, and developmental psychology. Because the goal of the meeting was communication across fields, each paper is written so that people without expertise in the area can understand it. The papers contain more detail about experimental procedure and subjects' performance than typical review papers to permit readers to explore the similarities and differences across different studies in different fields in a more informed and precise manner. Following each paper is an edited transcript of the discussion of that paper at the meeting in Philadelphia. Most readers will find the ideas, hypotheses, and critiques generated in the course of those discussions of interest. Comments in the discussions are abundantly footnoted to make it easier for readers to obtain more information about the topics and findings discussed. The subject index at the back of the book is generously cross-referenced as another means of helping readers locate the information they want.

The remainder of this Introduction presents an overview of each of the seven sections of the book. Background information and definitions are provided where they might be helpful. Section 1 is an exception to the theme of "scientists studying the same observable behaviors." Instead, the first paper here outlines a developmental phenomenon: extraordinary cross-modal abilities in infants shortly after birth. The later papers outline two different neural mechanisms, either of which might underlie this phenomenon. Section 2 and thereafter present results from the developmental and neuroscientific study of the same behavioral paradigms (e.g., not

simply the development and neural bases of "memory," but the development of memory as indicated by performance on a specific task and the neural bases of memory as indicated by performance on that same task).

### SECTION 1: SENSORY INTEGRATION AND CROSS-MODAL MATCHING

From birth or shortly thereafter, as Meltzoff demonstrates, human infants are capable of detecting cross-modal equivalences and of mapping information from one sensory modality onto another. This flies in the face of long-held notions that multisensory integration is a very sophisticated ability that can be accomplished only by late-maturing association cortex, and that sensory perception initially develops independently for each modality. Only later, everyone had assumed, can children begin to integrate information from the different senses. Meltzoff contends, instead, that there is no point, even from earliest life, when the senses are isolated. The claim is that correspondences among the senses are basic to the perceptual systems themselves.

As surprising as Meltzoff's claim might seem, there is ample evidence to support it. Minutes after birth, infants will imitate facial gestures they see someone make. Here, infants use proprioceptive and kinesthetic cues from their own unseen behaviors to match a visual stimulus. Four-month-old infants can detect correspondences, or the lack thereof, between the movements of someone's mouth (visual speech) and the speech sounds they hear (auditory speech): "There is a multimodal representation of speech even in 'prelinguistic' infants who are too young to speak" (Meltzoff). Infants less than 6 months of age prefer to look at a picture that matches the nipple they are sucking rather than a picture of a differently shaped nipple, even though they were never allowed to see the nipple that was put in their mouth. Here, they seem to be able to equate tactile and visual representations of a stimulus.

To be sure, there are limits to the cross-modal abilities of newborn and very young infants. In some ways the cross-modal abilities of older children, such as those described by Rose, appear to be fundamentally different from the cross-modal abilities of infants 0 to 6 months old. Moreover, there is a plethora of evidence, not simply intuitions, that in many situations older infants fail to integrate information from different senses. Only gradually over months does this ability for intersensory integration emerge (see, e.g., Diamond, Section VII). Despite whatever limits exist to the early cross-modal skills, however, the achievements of newborn and very young infants are real. How do they do it?

#### *Superior Colliculus Might Mediate Early Multimodal Abilities*

One possibility is that the newborn infant maps sensory information from one modality onto sensory information from another modality automatically by collicular mechanisms. Older infants, and adults, who have more mature cortical areas that inhibit the colliculus, may do some of that same mapping, and more sophisticated equivalence matching, by using association cortex.

The superior colliculus is a phylogenetically ancient structure that matures very early (see FIG. 1 and FIG. 6). The layers of cells in the superior colliculus are usually discussed by dividing them into the superficial and deep layers (layers I-III and IV-VII, respectively). Cells in the superficial layers generally respond only to visual stimuli. Many of the cells in the deep layers, however, are multimodal (i.e., the same cell receives input from more than one sensory modality). That is, at the cellular level in the superior colliculus there is integration of information from different senses. Moreover, not only are there cells in the deeper layers that receive sensory input, but cells here send commands to motor output structures in the brainstem and spinal

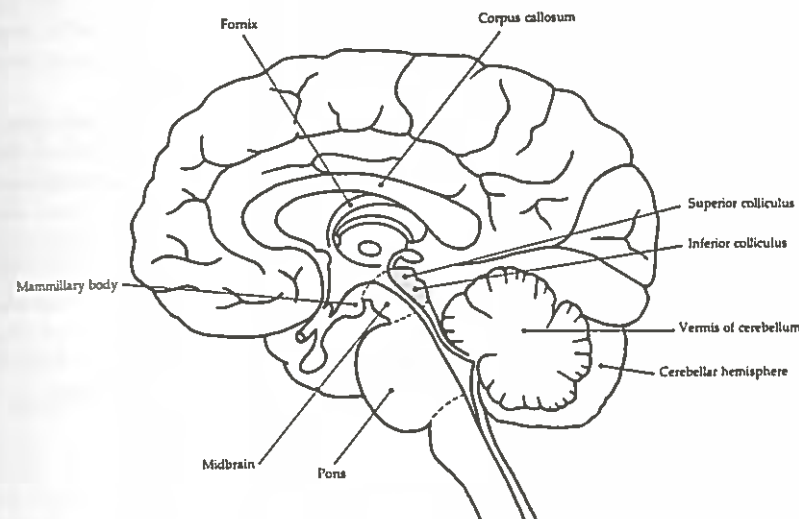


FIGURE 1. The human brain in the sagittal plane. Note that the superior colliculus is located in the roof of the midbrain or mesencephalon. The superior colliculus is involved particularly in processing, and responding to, visual stimuli; whereas the inferior colliculus is more specialized for auditory stimuli. In lower vertebrates (such as frogs) the inferior and superior colliculi are undifferentiated; they are combined into a single organ called the tectum. (Adapted from Figure 2-5 in J. B. Angevine & C. W. Cotman (1981), *Principles of Neuroanatomy*. NY: Oxford U. Press, drawn by Maureen Killackey.)

cord as well. Indeed, it may well be that individual cells in the deeper layers both receive sensory input and transmit motor commands.

Stein and Meredith have examined this multisensory integration by recording from individual cells in the superior colliculus of the cat. They report cells in the deep layers of the superior colliculus that are responsive to both visual and auditory stimuli, visual and somatosensory (e.g., tactile) stimuli, auditory and somatosensory stimuli, and even individual cells that are responsive to input from all three of these sensory modalities. The cells do not process the information from the different senses separately, but integrate this information so that how they respond to stimuli

in one modality is significantly affected (enhanced or inhibited) by stimuli in another modality. Stein and Meredith have varied the temporal intervals and spatial separations between stimuli to determine the temporal and spatial rules by which multisensory integration occurs in the superior colliculus. For example, a cell's receptive field for information in one modality is always in spatial register with that cell's receptive field for stimuli in any other modality to which it responds.

Not only might the superior colliculus be capable of subserving the multisensory integration needed for very young infants to succeed on Meltzoff's tasks, but the superior colliculus may also be capable of subserving the extremely close association between perception and action shown by infants on these tasks. A neonate perceives a facial gesture and then automatically acts to imitate it. The superior colliculus might mediate this close relationship between the perceptual system and the motor system because the multisensory cells in the superior colliculus might also be motor neurons in the sense of issuing commands to motor centers.

Plausible perhaps, but by no means proven. One of the remaining questions is: How early do cells in the superior colliculus become responsive to stimuli in more than one modality? There is a brief time lag between when kittens are first able to see and cells in superior colliculus are responsive to stimuli in one modality, and when multisensory cells are found. Do cells in the superior colliculus become responsive to multimodal stimuli early enough to mediate the multimodal abilities of a human infant minutes after birth? Another question concerns the source of the sensory inputs to these multisensory cells. If the inputs are from cortical areas, are these areas sufficiently mature early in life to furnish the kind of information the colliculus would need to subservise the multimodal abilities Meltzoff documents in infants?

#### *Exuberant Projections Might Mediate Early Multimodal Abilities*

Very early in development there are connections between the different sensory systems that later disappear. Thus, for example, axons from cells in the retina of neonatal hamsters project to "non-visual" brain areas, such as the nucleus of the thalamus specialized for somatosensory input and the inferior colliculus (which is specialized for auditory input). The nucleus of the thalamus specialized for processing visual information projects transiently to non-visual cortical areas in neonatal hamsters as well. Similarly, axons from auditory and somatosensory receptors project transiently in neonatal hamsters to areas of the thalamus specialized for processing information from other sensory modalities. Nor is this phenomenon confined to rodents. For example, neurons in many "non-visual" areas of cortex project to visual cortex in newborn kittens. These transient projections early in life outside of regions to which an area normally projects are called "exuberant projections."

If information from one modality is being projected to the thalamic and cortical areas for another modality, then sensory integration could occur there, without any need to relate the inputs coming into two different areas and without participation of association cortex. The exuberant projections in newborn infants might enable them to integrate multisensory information directly, whereas older infants and adults, who

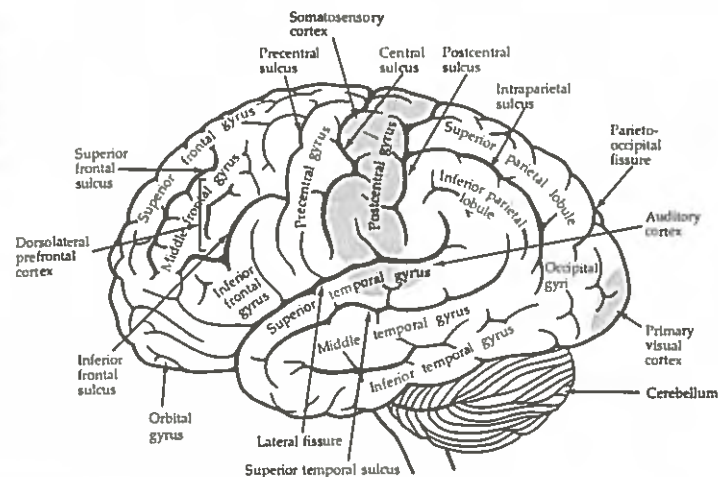


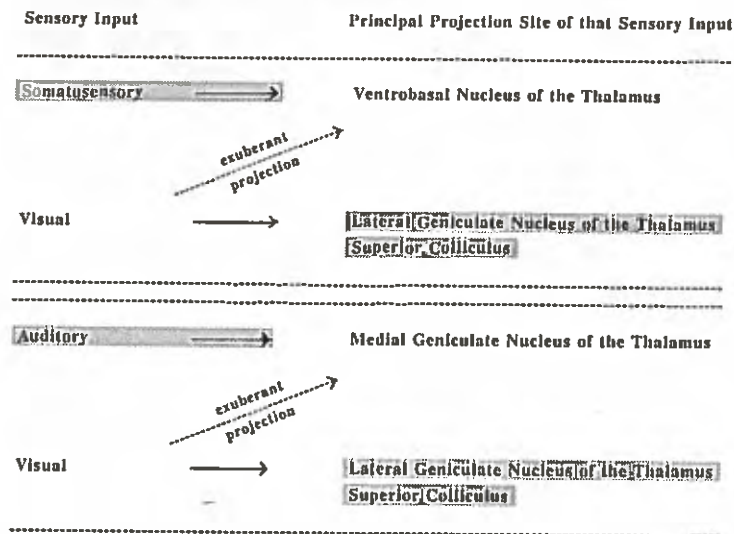
FIGURE 2. A lateral view of the left cerebral hemisphere. Note the location of auditory cortex in the superior temporal gyrus, the location of visual cortex in the occipital lobe, and the location of somatosensory cortex in the postcentral gyrus of the parietal lobe. (Adapted from Figure 2-4 in J. B. Angevine & C. W. Cotman (1981), *Principles of Neuroanatomy*. NY: Oxford U. Press, drawn by Maureen Killackey.)

no longer have these anomalous projections, might need to rely on association cortex.<sup>a</sup>

Frost has studied exuberant projections from one sensory modality to areas that do not normally receive input from that modality by surgically stabilizing these projections in the hamster. He has stabilized the retinal projection to the somatosensory thalamic nucleus (the ventrobasal nucleus) by removing the normal somatosensory inputs to this area and by ablating the normal projection sites for the retina (the visual thalamic nucleus [the lateral geniculate] and the superior colliculus) in the newborn hamster (see FIG. 3). Similarly, by removing the normal inputs to the auditory thalamic nucleus (the medial geniculate) and by removing the normal projection sites for the retina, he has surgically induced the sprouting of a novel projection from the retina to the medial geniculate nucleus in newborn hamsters (see FIG. 3). Under both of these conditions the anomalous projections are permanently maintained, making possible extended study of their properties.

<sup>a</sup>A brief tutorial on the anatomy of the visual, auditory, and somatosensory systems might be of help to some readers. Cells in the retina project via the optic tract to the visual nucleus of the thalamus (the lateral geniculate). Cells in the lateral geniculate, in turn, send axons to primary visual cortex (also called striate cortex, V1, and Area 17). Cells in both the retina and in visual cortex project to the superior colliculus. Auditory information from the cochlea eventually reaches the inferior colliculus, which relays it to the auditory nucleus of the thalamus (the medial geniculate), which in turn relays it to the auditory cortex in the superior temporal gyrus (Heschel's gyrus) (see FIG. 2). Somatosensory stimuli concern bodily sensations, such as limb position or the location, weight, or temperature of a stimulus touching the body. In rodents, the primary somatosensory nucleus of the thalamus is the ventrobasal nucleus, and it, in turn, projects to somatosensory cortex (Areas S1 and S2).

One obvious question is: Are exuberant projections functional? Clearly, they could not underlie the early multimodal abilities of infants if they could not carry information in a meaningful way. Frost's work suggests that, indeed, they are functional, at least in adults in whom Frost and his colleagues have surgically stabilized the projections. In his operated hamsters with novel retinal-ventrobasal projections, neurons in somatosensory cortex respond to visual stimuli. The presence of visually evoked responses in the somatosensory cortex of these animals suggests that the retinal-ventrobasal projection is conveying visual information. Moreover,



**FIGURE 3.** Schematic illustration of the procedure used by Frost to stabilize, and to induce, exuberant projections from the retina to non-visual areas. Top panel: He removed the normal somatosensory inputs to the somatosensory nucleus of the thalamus, and he removed the normal projection sites for the visual input from the retina. Since the somatosensory nucleus was thus deprived of its normal inputs, and the retina was deprived of its normal targets, the exuberant projection from the retina to the somatosensory thalamic nucleus was preserved. Bottom panel: Frost removed the normal auditory inputs to the auditory thalamic nucleus, and he removed the normal projection sites for the visual input from the retina. With the auditory nucleus thus deprived of its normal inputs, and the retina deprived of its normal targets, an exuberant projection from the retina to the auditory nucleus of the thalamus arose and was maintained.

the response properties of these visually responsive cells in somatosensory cortex closely resemble the response properties of neurons in primary visual cortex in normal hamsters. For example, orientation selective neurons occur with equal frequency and are equally sharply tuned in the visual cortex of normal hamsters and the somatosensory cortex of operated hamsters. Similar results appear to be true for retinal projections to classically auditory areas. Operated hamsters with novel retinal-medial geniculate projections can solve a visual pattern discrimination task that in normal animals depends on the integrity of the visual thalamus (which has

been removed in the operated animals). Subsequent removal of auditory cortex in the operated animals results in loss of the ability to succeed at this visual task.

The ability of "non-visual" areas to process visual information in meaningful ways is probably made possible by the fact that thalamic nuclei and cortical areas specialized for different sensory input perform similar transformations on the information they receive. They "use similar information processing strategies, based on similar morphological substrates" (Frost). This has a number of interesting implications that Frost addresses. He also explores why exuberant projections might arise in the first place, and what their existence means for notions of biological determinism and the role of experience.

It is plausible, perhaps, that exuberant projections between sensory areas might mediate early cross-modal abilities in the human infant, but do exuberant projections last long enough after birth under normal conditions to underlie cross-modal abilities in infants weeks or months after birth? Moreover, even if exuberant projections are functional after long stabilization how early are they functional? Are they functional early enough to subserve the achievements in a newborn infant? Exuberant projections do not exist between each area and every other area. For example, in the hamster there is no naturally occurring exuberant projection from the retina to the auditory nucleus of the thalamus. Do exuberant projections exist in humans between the requisite sensory modalities to underlie the cross-modal abilities observed in infants?

Neville has been studying what may be a case of naturally occurring stabilization and/or sprouting of an anomalous projection in humans. Her subjects are congenitally deaf individuals in whom the cochlea did not differentiate normally, leaving them profoundly, bilaterally deaf since before birth. They are otherwise free from any neurological disorder.

She has studied the performance of these deaf subjects on visual tasks and the pattern of their brain electrical activity during performance of the tasks. The electrophysiological technique she uses is called event-related potentials (ERP). Electrodes placed over various regions of the scalp record electrical activity in response to specific, precisely time-locked events. This permits fine-grained temporal analysis of the pattern of neural activity associated with particular aspects of information processing, although the source of that neural activity is less easy to determine using this technique.

Neville was found that deaf subjects respond more quickly (have faster reaction times) to motion in the periphery than do hearing subjects, even hearing subjects born to deaf parents (who, like deaf subjects, learned sign language as their first language). Moreover, Neville found that the pattern of brain electrical activity in response to visual events in the periphery has shifted in deaf subjects.

Neville speculates that this may indicate she is detecting, in the deaf subjects, visual responses from classically auditory areas (e.g., auditory cortex in the superior temporal gyrus), or enhanced visual responses from multimodal association areas (e.g., the superior temporal sulcus, which receives auditory, visual, and somatosensory input), as well as enhanced activity in posterior visual cortical areas.

Neville has also found that the ERPs of subjects (whether hearing or deaf) who acquired sign language as their first language differ in specific ways from the ERPs of those who know only a spoken language. The left hemisphere is not normally better



at motion detection than the right hemisphere, and it is sometimes worse. The left hemisphere is specialized to subserve language functions, though. Neville's work indicates that when motion is linguistically relevant (as it is for sign language) then detection of motion comes to be subserved preferentially by the left hemisphere, even on tasks where motion is not linguistically relevant.

## SECTION 2: CONDITIONING AND LEARNING

Lipsitt outlines what is known about the developmental progression of habituation and sensitization (simple non-associative learning) in human infants, and the developmental progression in the ability of human infants to acquire the classically conditioned eyeblink response. Marcus and Carew then discuss neural mechanisms underlying habituation, dishabituation, and sensitization, and Woodruff-Pak, Logan, and Thompson detail the neural basis for the classically conditioned eyeblink response, highlighting the role of the cerebellum. Keele and Ivry offer a hypothesis, and supporting evidence, for why the cerebellum is involved in acquiring, and demonstrating, the conditioned eyeblink response. Finally, Solomon, Groccia-Ellison, Levin, Blanchard, and Pendlebury discuss the role of the hippocampus in spanning a temporal gap between a warning stimulus and the noxious event following it (as in classical eyeblink conditioning using a trace paradigm).

Habituation<sup>b</sup> has been noted in human infants right after birth, and even in decerebrate human infants. Little is known, however, about how early human infants are capable of showing dishabituation or sensitization,<sup>c</sup> and nothing is known about whether these forms of simple learning appear at the same time in humans or whether one is present earlier than the other.

The work of Marcus and Carew has focused on the siphon withdrawal reflex in a simple invertebrate, the sea slug, *Aplysia*.<sup>d</sup> This work combines (1) behavioral studies of when habituation, dishabituation, and sensitization of this reflex emerge during development, with (2) study of the biophysical and biochemical mechanisms that mediate these forms of learning in this reflex, with (3) study of maturational changes at the synaptic, biophysical, and biochemical level that underlie why each of the different forms of learning in this reflex emerge when they do.

<sup>b</sup>Habituation is the decrement in a response following repeated presentations of a once-effective stimulus. Over time, the stimulus becomes less interesting or startling, and you react to it less. For example, an adult might laugh at a joke the first time, but not on the tenth retelling. One might think of habituation as indicating recognition or memory of a stimulus.

<sup>c</sup>Dishabituation is the recovery, or facilitation, of a habituated response. For example, having ceased to laugh at the too-often-told joke, you might well laugh again if the teller surprised you and gave it a new ending, or told a different joke. One might think of dishabituation as indicating that the subject can detect the difference between the old and new stimuli, and that the immediately preceding habituation was probably not due to some non-specific cause (e.g., general fatigue) but was a specific response to the old stimulus. Sensitization refers to the facilitation of a nonhabituated response. Here, a response beginning at normal levels is increased to a super-normal level, whereas in dishabituation, a reduced response is returned to its normal levels. For example, someone who has just gotten an electric shock will show an exaggerated (i.e., sensitized) response to a mild touch.

<sup>d</sup>Those who have never seen an *Aplysia* might wish to turn to FIGURE 1 in the paper by Marcus and Carew.

The siphon is the fleshy spout of the protective covering over the respiratory chamber housing the gill in *Aplysia*. When a jet of water is delivered to the siphon, the siphon defensively contracts. It is this that is termed the "siphon withdrawal reflex." It is a long way from sea slugs to humans. The reason such a simple organism as an *Aplysia* was chosen for study by Marcus and Carew is that its neurons are large, making biophysical and biochemical analyses easier, and its neurons are few in number, making it possible to complete these analyses for all, or almost all, of the cells in the organism's central nervous system.

Marcus and Carew report that as soon as a young *Aplysia* can make the response required by the siphon withdrawal reflex (i.e., contracting the siphon), habituation of the reflex can be demonstrated—but only if the repeated presentations of the stimulus (e.g., water squirted at the siphon) occur very close together in time ( $\leq 1$  sec apart). Gradually over development, *Aplysia* can demonstrate in the habituation paradigm that they remember the earlier presentations of the stimulus over longer and longer temporal intervals.

Dishabituation and sensitization had been thought to be different expressions of the same process (facilitation of a response). For this reason it was very surprising when Carew's group found that dishabituation emerges much earlier than sensitization in *Aplysia*. Moreover, further work has shown that the cellular mechanisms mediating dishabituation and sensitization are not the same, and these, too, emerge at different ages. This body of work strongly suggests that dishabituation and sensitization may be fundamentally different processes.

Before Carew and his colleagues analyzed the development of specific neural circuits in the *Aplysia*, Cash, working in Carew's lab, counted the number of cells in each ganglion throughout the entire central nervous system of the *Aplysia* at each stage within the juvenile period of the *Aplysia*'s life. The finding that emerged was dramatic: During the 60 days preceding Stage 12 there is little change in cell number; during the 60 days comprising Stage 12 the number of cells increases 800%. Moreover, the phenomenon is system-wide; it was found in all ganglion cells throughout the *Aplysia*'s central nervous system.

This is somewhat reminiscent of another dramatic and unexpected finding: that the density of synapses increases at the same rate and over the same time period throughout sensory, motor, and association areas of primate cerebral cortex and throughout all six layers of each of these cortical areas.<sup>e</sup> This result was completely unexpected, given that traditional wisdom had long held that sensory and motor areas mature earlier than association areas and given the inside-outside pattern of cell migration, suggesting that deeper levels mature earlier than superficial ones. The findings of Rakic and colleagues indicate that, at least in some ways, the whole neocortex may be maturing more as a unit than previously thought. Together, this finding and that of Cash and Carew, suggest that the central nervous system may be more of a system in terms of at least some aspects of development than previously thought. A system-wide trigger would appear to be responsible for the widespread, simultaneous proliferation of cells in the ganglia of *Aplysia* and for the widespread,

<sup>e</sup>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.

simultaneous changes in synaptic density in disparate regions of the cerebral cortex of the rhesus monkey.

In **classical eyeblink conditioning**, the goal is to teach a subject, through repeated experience and without verbal instruction, that a neutral stimulus (e.g., a tone) signals that some irritant (e.g., a jet of air) is going to be applied to the eye at some specific interval after the onset of the neutral stimulus. Subjects show they have learned this by using the information provided by the tone to defensively blink their eye in anticipation of the airpuff at just the right time to avoid being hit in the eye by the gust of air.

An eyeblink occurs reflexively when a jet of air is directed at the eye. An airpuff, then, is an **unconditioned stimulus (US)** for an eyeblink. Subjects will blink in response to a burst of air coming at their eye without any prior training or conditioning.

The initially neutral stimulus (the tone) is called the **conditioned stimulus (CS)** because subjects need to learn that there is some reason to blink in response to it (the reason being that it is predictive of an impending air jet to their eye). The same response (blinking the eye) is called a **conditioned response** when it occurs in response to an initially neutral stimulus (the CS), and is called an **unconditioned response** when it occurs in response to a stimulus that would have elicited it anyway (the US).

Most classical conditioning of the eyeblink response is done using a delay paradigm. In **delay conditioning**, the CS (e.g., a tone) comes on first; after a specific time interval (say, 250 msec) the US (e.g., an airpuff) comes on as well; finally both terminate together. The CS precedes the US and remains on the whole time the US is present (see Solomon *et al.*, FIGURE 1b, row 1).

Lipsitt summarizes studies of classical eyelid conditioning in 10–60-day-old human infants conducted in his laboratory by Little. Even the youngest infants were capable of learning the association between tone and airpuff, but infants of all ages required a much *longer* interval between the tone and airpuff (i.e., a much longer **interstimulus interval** or **ISI**) than is true for adults—at least 3 times longer. For adults the optimal ISI is about 500 msec (0.5 sec) between tone and airpuff; infants showed no evidence of learning at ISIs shorter than 1500 msec (1.5 sec). Moreover, as Lipsitt points out, it appears to be a general principle, across different conditioning paradigms and different species, that infants require longer ISIs than do adults.<sup>f</sup> Why? The answer to that is not yet known, but it probably has to do with the speed at which infants and adults can process information.

Although infants are able to acquire the classically conditioned eyeblink response shortly after birth, both Woodruf-Pak and Solomon point out that there is a protracted developmental progression in the level at which this is learned. Thus, while infants tested in Lipsitt's lab showed significant evidence of learning, they made the conditioned response on less than 30% of the trials, whereas 6-year-olds do so on 90% of the trials. There is a decline again in adulthood (beginning in the 40s) in

<sup>f</sup>It should be noted that longer ISIs in delay conditioning paradigms do *not* entail longer delays between the offset of the CS and the onset of the US. The CS remains on until and during presentation of the CS. A longer ISI in delay conditioning means, instead, that the CS remains on for a longer time (there is a longer interval between the *onset* of the CS and the onset of the US).

the degree to which individuals show the conditioned eyeblink response; by 90 years of age the percentage of conditioned responses may be down to 40% again. Another parallel between early development and later adulthood is that subjects of 60 years of age or more appear to once again need a longer ISI between the tone and airpuff.

Woodruf-Pak, Logan, and Thompson present evidence on the neural circuit mediating the classically conditioned eyeblink response. To investigate this they have combined precise characterization of the behavioral response in the rabbit with electrical stimulation, electrical recording, and lesion techniques. They have found that cerebral cortex (indeed, any tissue above the thalamus) is not necessary for acquiring this learned response. They have also found that the **cerebellum** is necessary, and in particular, the **interpositus (or interposed) nucleus** of the cerebellum. (The location of the cerebellum in the brain is illustrated in FIGS. 1 and 2 above.)

Evidence for the role of the cerebellum in classical eyeblink conditioning includes: (1) During training, the firing pattern of cells in the cerebellum changes in parallel with behavioral changes indicating that the subject is learning the conditioned response. (2) Lesions of the interpositus nucleus abolish the learned response (blinking in response to a tone) and prevent reacquisition of this response, without affecting the unconditioned response (blinking in response to an airpuff). Since lesioned subjects can still blink, the cerebellar lesions are not having their effect at the level of the motor act; subjects are still capable of making the response. What subjects with cerebellar lesions seem to be incapable of is remembering, or using, the pre-operatively learned association to the tone, and/or of demonstrating learning of this association post-operatively. The same results are found if a light, rather than a tone, is used as the CS.

Woodruf-Pak, Logan, and Thompson hypothesize that the climbing fibers from the inferior olive provide the essential input to the cerebellum for acquiring learned responses such as the conditioned eyeblink response.<sup>g</sup>

The cerebellum is relatively immature at birth (e.g., its granule cells are still dividing and still forming parallel fibers). This is consistent with the relatively low percentage of conditioned eyeblink responses found by Lipsitt in young infants. Thompson suggests it may also be related to infants' need for a longer ISI between CS and US. Evidence is needed, however, directly relating particular maturational

<sup>g</sup>Two of the types of cells found in the outer mantle of gray matter surrounding the cerebellum (cerebellar cortex) are granule cells and Purkinje cells. Granule cells are tiny (like grains of sand) and numerous (up to 7 million per cubic centimeter in the layer below the Purkinje cells). Purkinje cells are very large, are arranged in neat rows, and have elaborate dendritic trees.

There are only two sources of input to the cerebellum, and one source of output. Both sources of input project to the deep cerebellar nuclei, including the interpositus nucleus. One source of input is called the "climbing fibers." It consists of axons from the inferior olivary nucleus (inferior olive). Each Purkinje cell receives input from only one climbing fiber, but each climbing fiber may synapse on 10 Purkinje cells. Climbing fibers also terminate on granule cells. The other source of input to the cerebellum is called the "mossy fibers." These axons originate in the spinal cord and brainstem and terminate on granule cells in cerebellar cortex. The granule cells, in turn, send out branching axons that give rise to the "parallel fibers." These parallel fibers travel in a plane perpendicular to the Purkinje cell dendrites. Each parallel fiber synapses on the dendrites of multiple Purkinje cells, and each Purkinje cell receives input from as many as 200,000 parallel fibers. Purkinje cells provide the only source of output from cerebellar cortex.

changes in the cerebellum with age-related improvements in the ability to learn the conditioned eyeblink response.

In normal aging, there is a loss of Purkinje cells in the cerebellum. This may be related to the reduced level of classical conditioning of the eyeblink response associated with aging.

Keele and Ivry hypothesize that the reason the cerebellum is needed for eyeblink conditioning is that the cerebellum plays a special role in measuring the time interval between CS and US, and in using that temporal calculation so that the eyeblink occurs at just the right moment (i.e., immediately before the air puff).

Precise timing of when the conditioned eyeblink is made is critical. If it occurs too late it fails to protect the eye from the gust of air, and if it occurs too early, the blink ends before the airpuff is presented and again it fails to protect the eye. Because classical eyeblink conditioning requires this delicate timing, Keele and Ivry argue, it requires the cerebellum. They postulate that the function of the cerebellum, at least the lateral cerebellum, may not be primarily motor, as most people have assumed, or memorial, as Thompson has suggested, but is, instead, to provide temporal computations that can be used for a variety of tasks, both perceptual and motor.

This hypothesis is a radical departure from the traditional view of the functions of the cerebellum, which emphasizes the cerebellum's role as part of the motor system. Yet, Keele and Ivry's hypothesis is consistent with much of the available data. For example, it is consistent with findings that the cerebellum is also required for other conditioning tasks involving discrete, precisely timed protective responses (such as the conditioned leg flexion response to avoid shock), but not for classical conditioning of responses that are not precisely timed (such as heart rate slowing in anticipation of shock). It is also consistent with findings that accuracy in timing motor responses is correlated across different motor effectors (e.g., fingers and feet) and that these motoric measures of timing are also correlated with accuracy in perceptual judgments of the length of temporal intervals in auditory tasks—suggesting that a common process may underlie timing regardless of task or response system.

Keele and Ivry report evidence that patients with damage to the cerebellum are impaired in the regular timing of motor tasks. This is not surprising, and is fully consistent with the traditional view of the cerebellum's role in motor functions. More striking, however, is the evidence Keele and Ivry present that cerebellar patients are impaired on a *non-motor* task (perception of duration) that requires precise timing, but not on a non-motor task (perception of loudness) where timing is irrelevant.

Finally, as Keele and Ivry point out, the exquisite lattice structure and striking anatomical regularity of the cerebellum is consistent, at least on the surface, with the precise timing functions they postulate. Recall the parallel rows of Purkinje cells and the rows of parallel fibers traveling in planes perpendicular to the Purkinje cell dendrites described in footnote *g*. A message conveyed by a single parallel fiber is related to successive Purkinje cells at precise, minutely different temporal intervals.

Solomon and his colleagues have carried out some of the seminal experiments on the role of the hippocampus in classical eyeblink conditioning. It had been known for some time that cells in the hippocampus of the rabbit markedly increase their rate of firing during classical eyeblink conditioning. These learning-induced changes in unit activity in the hippocampus precede and accurately predict subsequent behavioral learning performance. Because of the close correlation between rate of cell firing in

the hippocampus and improved performance on the task, it had been generally assumed that the hippocampus was necessary for the learning that occurred.

Imagine the surprise, then, when Solomon and Moore demonstrated that, if you bilaterally remove the hippocampus, rabbits still acquire the classically conditioned eyeblink response, and at the normal rate—even though they have no hippocampus. This illustrates an important lesson: Increased activity in a neural region, even when that is closely tied to learning and performance on a task, does not necessarily mean that the neural region is involved in mediating that learning or performance. It may indicate, for example, that the neural area is monitoring what is happening, although not directly participating.

Solomon and his colleagues went on to show that the hippocampus is necessary for classical eyeblink conditioning if a trace paradigm is used, rather than the commonly used delay paradigm. In trace conditioning, the CS and US do *not* overlap, as they do in delay conditioning. Instead, the CS is presented, followed by a trace interval (of say, 0.5 sec) during which no stimuli are presented; finally the US appears (see Solomon *et al.*, Figure 1b, row 2). Thus, the hippocampus appears to be important for bridging the temporal gap when the CS and US are not temporally contiguous (as in trace conditioning), although the hippocampus is not needed when the CS and US overlap (as in delay conditioning). It is not yet clear, however, why the hippocampus is important for trace conditioning. One possibility is that it performs a memory function. Another possibility is that it helps subjects to inhibit responding during the long period between CS onset and US onset.

Solomon and his colleagues, and Woodruff-Pak and her colleagues, have also looked at changes in eyeblink conditioning with aging in rabbits and humans, in humans with Alzheimer's disease, and in young rabbits with induced neuropathology resembling that seen in normal aging and in disorders of aging.

In normal aging, human adults show a progressively lower level of conditioning and require progressively longer ISI intervals between the CS and US. However, *all* normal adults, at all ages, show clear evidence of some degree of learning of the conditioned eyeblink response. This is not true for those diagnosed as probably having senile dementia of the Alzheimer's type.<sup>h</sup>

Now, although the hippocampus is not necessary for learning the conditioned eyeblink response, an abnormally functioning hippocampus interferes with this learning. That is, while classical eyeblink conditioning proceeds normally if the hippocampus is missing, it is impaired if the hippocampus is present but malfunctioning. Alzheimer's disease appears to be accompanied by profound alterations in hippocampal functioning. This suggests that the abnormally functioning hippocampus of Alzheimer's patients may interfere with their acquisition of the conditioned eyeblink response, although the motor aspects of their eyeblink reflex are intact.

An important question, asked by several participants at the meeting during the discussions, is: What happens to classical eyeblink conditioning in patients with amnesia, who presumably have damage to the hippocampal neural circuit? The few studies that have investigated classical conditioning in amnesic patients (although

<sup>h</sup>A definitive diagnosis of Alzheimer's disease can only be made after death, when the brain is examined. As Nadel points out, however, just as behavior is not fully definitive on its own, neither is anatomy. The brains of Down syndrome patients look much like the brains of Alzheimer's patients, although Down syndrome patients are not demented.

not of the eyeblink response) have found it to be normal. It may be that in amnesia the hippocampus is essentially not working at all (in which case classical eyeblink conditioning using a delay paradigm should be unaffected), whereas in Alzheimer's disease, the hippocampus is still working but sending out garbled messages (in which case delayed conditioning should be impaired, as it is in Alzheimer's patients). If this analysis is correct, then amnesic patients should acquire the conditioned eyeblink response normally when a delay paradigm is used, but be unable to acquire the response when a trace paradigm is used. This remains to be investigated. Perhaps, also, persons with mild amnesia, who have *better* memory than persons with profound amnesia, might perform *worse* on delay conditioning than those with profound amnesia. This would be expected if mild amnesia means a partially, inadequately functioning hippocampal neural circuit, while profound amnesia means a shutdown in that circuit altogether. Studies of questions such as these might yield important insights into the nature of the hippocampal impairment in amnesia.

### SECTION 3: WORKING MEMORY: SPANNING WITHIN-TRIAL TEMPORAL DELAYS

In this section, Millar, Diamond, and Fuster focus on the memory required to link together the elements in a cue-response-reward episode when a delay is imposed between two of those elements. If a cue specifies which response is correct, but subjects are only allowed to respond after the cue has disappeared, subjects must hold the memory of that cue on-line in order to know what response to make. Similarly, if subjects receive feedback concerning their response only some time after that response has been made, they must remember what that response was in order to learn the relationship between the feedback and their response.

This requires memory during the learning process to link together the parts, to learn an association. It is to be distinguished from memory of an already learned association. In the latter case the delay is imposed, not between elements within a trial, but between trials or between testing sessions. In the first case the question is, "Over how large a temporal gap can the subject still integrate the various parts of a trial?" In the latter case the question is, "Over how large a temporal gap can an association, once learned, be remembered?"

Diamond describes the developmental progression in the performance of human infants and infant monkeys on the  $A\bar{B}$  and delayed response tasks, and the neural system required for successful performance on these same two tasks, as indicated by lesion studies in infant and adult monkeys. In the  $A\bar{B}$  and delayed response tasks, a delay is imposed between the cue and the response. The subject watches as a reward is hidden, and then after a delay is allowed to search for the reward.  $A\bar{B}$  and delayed response are essentially the same task; two names exist primarily because of the historical isolation of human infancy researchers (who call the task " $A\bar{B}$ ," pronounced "A not B") and neuroscientists (who call the task "delayed response").

The youngest infants who can be tested on the  $A\bar{B}$  and delayed response tasks cannot succeed if there is any delay at all between when the reward is hidden and when they are allowed to reach. They usually succeed, however, if allowed to reach right away. Gradually as they grow older, infants become able to withstand longer

and longer delays on these two tasks. Thus, at 7½ months they fail when a delay of only 2 sec is imposed and at 8 months they fail with delays of 3 sec; by 12 months they succeed on  $A\bar{B}$  and delayed response with delays of up to 10 sec. The same developmental progression seen in human infants between 7½–12 months is seen in infant monkeys on both the  $A\bar{B}$  and delayed response tasks between 1½–4 months of age.

The most prominent member of the neural circuit required for success on the  $A\bar{B}$  and delayed response tasks is dorsolateral prefrontal cortex. Frontal cortex comprises the entire cortical expanse in front of the central sulcus (see FIGS. 2 and 4). Prefrontal cortex includes all of frontal cortex in front of the precentral sulcus (see FIG. 2; the remaining area of frontal cortex, between the precentral and central sulci, is motor cortex). Prefrontal cortex is an association area; its functions are primarily

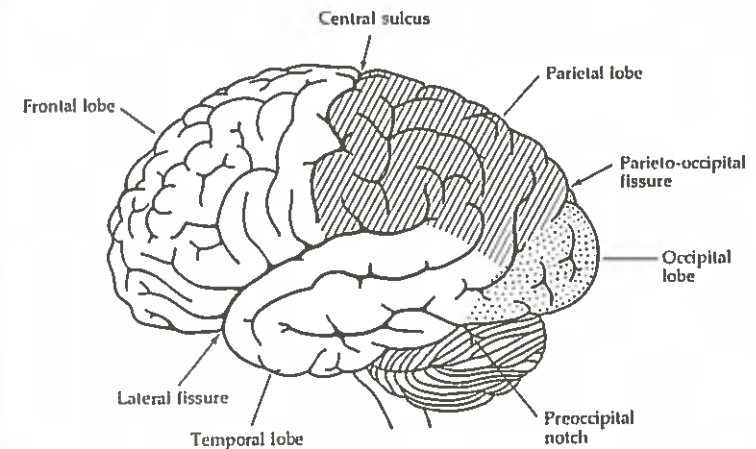


FIGURE 4. The cerebral lobes indicated on the lateral surface of the left hemisphere. The frontal lobe and temporal lobe are shown in white; the parietal lobe is shown striped; the occipital lobe is the stippled area. (Reprinted from Figure 13-1A in J. B. Angevine & C. W. Cotman (1981), *Principles of Neuroanatomy*. NY: Oxford U. Press, drawn by Maureen Killackey.)

integrative, neither exclusively sensory nor motor. Dorsolateral prefrontal cortex occupies roughly the middle frontal gyrus (see FIG. 2), from the superior to the inferior frontal sulci.<sup>4</sup>

Adult monkeys with lesions of dorsolateral prefrontal cortex succeed on  $A\bar{B}$  and delayed response if there is no delay, but fail both tasks with delays as brief as 2–5 sec, as do human infants of 7½–9 months and infant monkeys of 1½–2½ months. Across a host of parametric variations in the two tasks, the performance of monkeys with lesions of dorsolateral prefrontal cortex mirrors that of 7½–9-month-old human infants and 1½–2½-month-old infant monkeys. Lesions to inferior parietal cortex or to the hippocampal formation do not produce these effects on  $A\bar{B}$  or delayed

<sup>4</sup>RAJKOWSKA, G. & P. S. GOLDMAN-RAKIC. 1990. Cytoarchitectonic remapping of areas 9 and 46 in the human prefrontal cortex. Abstracts of the Society for Neuroscience (Part II) 16: 1093.

response performance! In considering these results, it is important to remember two points: (1) Even if dorsolateral prefrontal cortex is undergoing important maturational changes between 7½–12 months in humans and 1½–4 months in monkeys (as these results suggest), dorsolateral prefrontal cortex is not fully mature by the end of these periods. It continues to mature long past infancy, and probably does not reach full maturity until at least puberty. (2) Dorsolateral prefrontal cortex does not function in isolation. It is part of a system of interconnected neural areas that function together.

Much of the work demonstrating the importance of dorsolateral prefrontal cortex for success on the delayed response task, and especially for bridging within-trial delays on the task, was conducted by Fuster and his colleagues using (a) localized cooling, where a neural area is temporarily inactivated and the monkey can serve as its own control, and (b) single cell electrical recording, where patterns of neuronal activity in intact animals can be related to performance. For example, Fuster (and later other investigators as well) found cells in dorsolateral prefrontal cortex that increase firing when the reward is hidden and maintain that elevated level of activity throughout the delay. Moreover, they found a direct relation between this firing pattern and monkeys' performance on the task; on trials where such prefrontal activity is not seen, subjects do not succeed in finding the reward. Fuster has gone on to find similar results with several other tasks that impose a delay between cue and response.

Fuster proposes that dorsolateral prefrontal cortex is required not only for memory, but for planning, because he views memory and planning as flip sides of the same coin: Memory is looking backward in time; while planning is looking forward. In addition, Fuster relates the cognitive functions of prefrontal cortex to the motor functions of the more posterior portions of frontal cortex: "Prefrontal memory is the memory *in and for action*. . . . [T]he sensory, perceptual, and mnemonic properties of prefrontal cortex are defined by the behavioral actions they subserve. All this is in harmony with the notion that the frontal lobe, as a whole, is primarily dedicated to the control of movement and motor behavior. Prefrontal cortex provides the short-term cognitive support that such control sometimes requires."

Millar describes the developmental progression in human infants' performance on an operant conditioning task where the reinforcement, instead of occurring immediately after a subject's response, is delayed a few seconds. Here, the delay is imposed between the subject's response and the reward, rather than between the cue and the subject's response. The required response in Millar's task was a vigorous arm movement. The reward was a brief colored light display. The youngest infants tested (6-month-olds) succeeded when there was no delay between their response and the reward, but even the oldest infants (8 months) failed to learn the relationship between response and reward when these were separated by a 3-sec delay.

<sup>1</sup>Parietal cortex extends from the central sulcus back to the parieto-occipital fissure (see FIGS. 2 and 4). It is divided into superior and inferior portions (see FIG. 2). The hippocampal formation is deep within the temporal lobe (see FIG. 4) and includes the hippocampus proper, dentate gyrus, and surrounding tissue such as much of the parahippocampal gyrus and entorhinal cortex (see FIG. 5 below).

Millar's delay of reinforcement paradigm, on the one hand, and  $A\bar{B}$  and delayed response, on the other, all impose a delay within the cue–response–reward sequence in a trial, and human infants of 8 months succeed on all three tasks when the delay is 0 sec, but not when it is 3 sec. Perhaps all three tasks require similar cognitive abilities and are dependent on the same neural system. However, both Millar and Diamond point out differences between the delay of reinforcement and  $A\bar{B}$ /delayed response tasks that may be at least as important as their similarities. And, both Millar and Diamond note that trace conditioning, too, involves a delay between the elements within a trial (between the offset of the US and the onset of the CS)—yet trace conditioning appears to require involvement of the hippocampus, while  $A\bar{B}$  and delayed response require dorsolateral prefrontal cortex.

What are the critical features of a task that cause it to require involvement of dorsolateral prefrontal cortex? What are the critical features of a task that cause it to require hippocampal involvement? How do the abilities dependent on dorsolateral prefrontal cortex differ from those dependent on the hippocampus, and how do each of these differ from abilities dependent on still other areas of the brain? What kinds of tasks require neither dorsolateral prefrontal cortex nor the hippocampus? Sections 3, 4, and 5 address this set of questions (see especially Table 7 in Diamond [Section 3] and Table 1 in Zola-Morgan & Squire [Section 4]). Section 3 focuses on the type of memory that appears to depend on dorsolateral prefrontal cortex and related structures. Section 4 focuses on the type of memory that depends on the hippocampus and related structures.

#### *Memory Requirements Shared by Tasks Dependent on Dorsolateral Prefrontal Cortex or the Hippocampus*

To be sure, there is considerable similarity between tests sensitive to damage of dorsolateral prefrontal cortex and tests sensitive to hippocampal damage. For example, tasks requiring either dorsolateral prefrontal cortex or the hippocampus for successful performance tend to present the to-be-remembered information only once; new information must be remembered on each trial. Tasks dependent on other neural systems tend to allow subjects to learn a rule over repeated presentations over many trials; once the rule is learned it can guide performance across all trials. Consider the  $A\bar{B}$  task, which depends on the dorsolateral prefrontal neural circuit, and the delayed non-matching to sample task [described below in Section 4], which depends on the hippocampal neural circuit.  $A\bar{B}$  involves a rule ("reach where the reward was last hidden") and so does delayed non-matching to sample ("reach to the object that does not match the sample shown earlier"), however, on neither task is the rule sufficient to guide correct performance. On  $A\bar{B}$  the subject must also remember, on each trial, where the reward was hidden. On delayed non-matching to sample the subject must also remember, on each trial, what the sample looked like. A task like spatial or pattern discrimination, on the other hand, requires *only* that subjects remember the rule (e.g., "reach left" or "reach to the square"). Conditioning tasks that require only the learning of one rule (e.g., "kick your left leg to make

mobile move" as in Rovee-Collier's task) generally do not require prefrontal or hippocampal involvement.

The to-be-remembered information is usually presented only briefly in tasks dependent on dorsolateral prefrontal cortex or the hippocampus. Subjects see where the reward is hidden on an  $A\bar{B}$  or delayed response trial, or see what the sample is on a delayed non-matching to sample trial, for only about 2–5 sec. In contrast, on looking tasks such as those used by Baillargeon (see Diamond, Section 4) the to-be-remembered information is present, or repeatedly presented, over periods of 20–30 sec or more.

Tasks dependent on dorsolateral prefrontal cortex or the hippocampus usually impose the memory requirement (that is, the delay) within a trial (e.g.,  $A\bar{B}$  or delayed response; trace conditioning or delayed non-matching to sample), as opposed to between trials or between testing sessions. Deferred imitation [see Section 5 below] studied by Meltzoff and by Mandler, shares certain characteristics with tasks dependent on dorsolateral prefrontal cortex or the hippocampus: The to-be-remembered information is presented only once and briefly. Subjects are not given the opportunity to learn this slowly over trials, and their memory might not be tested until a day or a week later. However, deferred imitation tasks do not impose the delay between the elements of the sequence to be remembered, in contrast to  $A\bar{B}$  or delayed response. For example, when Meltzoff touches his head to a panel causing a light to come on, that effect happens immediately, not after a 3-sec delay. Similarly, when Mandler or a colleague hits a lever thereby launching the object that was resting on the opposite end of the lever, the effect is immediate, not delayed. Human infants succeed on deferred imitation tasks relatively early in life, over rather long delays; it is unlikely that these tasks require dorsolateral prefrontal cortex involvement.

The memory functions of dorsolateral prefrontal cortex or the hippocampus are not limited to any particular modality; i.e., they are supramodal. Tasks performed in the haptic modality produce the same results here as do tasks performed in visual modality.

Finally, tasks dependent on dorsolateral prefrontal cortex or the hippocampus involve explicit testing, where subjects receive feedback on each trial and where it is possible for them to be wrong. In contrast, tests of implicit memory [see Section 4 below] where subjects are asked to respond with the first thing that comes to mind (instead of being asked to respond with items they previously studied) do not appear to require hippocampal or prefrontal involvement. Similarly, looking measures (such as those used by Baillargeon [cited in Diamond, Section 4]) simply allow infants to look at what interests them; the infant is not given feedback that this or that response was wrong.<sup>k</sup>

Must *all* of these task characteristics be present for a given task to require dorsolateral prefrontal cortex or the hippocampus? If not, which of these characteristics are the critical one(s)? The answers to these questions are not yet known. It is, true, however, that one or more of these characteristics is true of many tasks that do

<sup>k</sup>It is commonplace to find that subjects perform better, and succeed at younger ages, when they are not aware that they are being, or will be, formally tested, see, for example:

NEWMAN, L. S. 1990. Intentional and unintentional memory in young children: Remembering vs. playing. *Journal of Experimental Child Psychology* 50: 243–258.

not require dorsolateral prefrontal cortex or the hippocampus. For example, it is certainly true that subjects receive explicit feedback in conditioning tasks that require no prefrontal or hippocampal involvement.

#### *Characteristics That Appear to Distinguish Tasks Dependent on Dorsolateral Prefrontal Cortex from Tasks Dependent on the Hippocampus*

Deficits from lesions or inactivation of prefrontal cortex are evident at the very briefest delays (e.g., 1 or 2 sec). Deficits from lesions or inactivation of the hippocampus, however, are usually not evident unless longer delays (e.g., 30–60 sec or more) are used. Thus, tasks with only very brief delays do not generally require involvement of the hippocampus. (Trace conditioning appears to be an exception here.)

Tasks dependent on dorsolateral prefrontal cortex often use the same stimuli repeatedly over trials and hence pose potential problems of proactive interference. Subjects will fail such tasks if they have difficulty with temporal order discrimination (e.g., "Where was the reward hidden on *this* trial, as opposed to on the last trial or earlier trials?"). Milner has highlighted the importance of prefrontal cortex for the memory of temporal order. Patients with frontal cortex damage alone or in combination with amnesia generally show deficits in temporal order memory and in resistance to proactive interference; amnesic patients without frontal lobe pathology tend not to show these deficits. Tasks dependent on the hippocampus, such as delayed non-matching to sample, usually do not pose problems of proactive interference because a given stimulus is used on only one trial (see, however, Diamond's discussion of the radial arm maze [Diamond, Section 3]). Fagan points out that evidence of proactive interference is also evidence of intact memory in some sense, as the earlier information must be remembered at some level for it to interfere. However, it is also evidence of either disorganized memory (losing track of the order in which the information was encountered) or of weak immediate or explicit memory (so that the memory of older information, or of information tied to previous actions, is as strong or stronger than the memory of the information most recently presented).

The information that must be remembered in tasks dependent on dorsolateral prefrontal cortex is often spatial location (e.g., left or right position) rather than the color or shape of a stimulus. Goldman-Rakic has suggested that a region within dorsolateral prefrontal cortex may be required specifically for memory of spatial information, but not for memory of other things. The hippocampus, on the other hand, seems to be equally necessary for the memory of anything—color, shape, or position.

Diamond suggests that dorsolateral prefrontal cortex may also be required for the memory of anything (color, shape, or position), as long as the task also requires inhibition of a strong response tendency. For example, in  $A\bar{B}$  and delayed response, subjects may have difficulty inhibiting the response that was previously rewarded. Delayed non-matching to sample, on the other hand, which depends on hippocampal involvement but not dorsolateral prefrontal cortex, requires no inhibition; indeed, it capitalizes on subjects' inherent inclination to reach to something new rather than to something they have seen before.

#### SECTION 4: IMPLICIT AND EXPLICIT MEMORY I: THE VISUAL PAIRED COMPARISON AND DELAYED NON-MATCHING TO SAMPLE TASKS

**Explicit memory** is memory of which you are aware. You know you know this. It has been called by many other names as well, such as "declarative memory," "conscious memory," or "symbolic representation" (see Table 1 in the discussion of the Roediger *et al.* paper). **Implicit memory**, on the other hand, is memory that can be demonstrated, but of which you are not aware. For example, the second time something is presented you might learn it faster even though you have no conscious recollection of the first presentation. Here, the change in the rate at which the material is learned is taken as evidence that at some level the first presentation is still "remembered." Other names that have been suggested for implicit memory are "procedural memory," "unconscious memory," "enactive representation," or "habit" (see Table 1 in the discussion of the Roediger *et al.* paper). Note that evidence of implicit memory is always based on an inference (because if subjects could tell you they remembered, the memory would be "explicit") and any change in behavior on the basis of past experience, such as learning or priming, is considered an appropriate basis on which to make this inference.

Is there any behavioral evidence, however, that could serve as a basis for inferring the presence of *explicit* memory? In particular, how can one know a memory is explicit in organisms who do not speak, such as babies or animals? What criteria might be used to determine the existence of explicit memory in such subjects? Or, is memory that must be inferred from behavior necessarily implicit? Is explicit memory inextricably tied to language so that an organism lacking language is incapable of explicit memory? Perhaps non-verbal organisms are capable of explicit memory but we will never know because of the fundamental inability to assess explicit memory in such subjects. That is, one might argue that we cannot know that subjects are aware that they remember unless they tell us; non-verbal subjects cannot tell us; hence this is fundamentally unknowable in subjects who lack language. They may have the ability for explicit memory; we just have no way of telling.

There is wide disagreement on the answers to these questions, and this book presents a lively discussion of the issues by the contributors. For example, Roediger suggests, "The term declarative memory seems a misnomer when applied to animals who (in our experience) are not prone to making declarations. . . . Indeed, both tests tapping animals' 'procedural memory' and their 'declarative memory' seem to us to rely completely on procedures." On the other hand, Meltzoff is not only willing to credit animals and infants with the capacity for explicit memory, but he speculates that this ability may be present even in newborns. "There may *never* be a time that the human infant is confined to a purely habit/procedural mode." Fagan and Rovee-Collier suggest that asking whether an animal's or infant's behavior indicates implicit or explicit memory may not be an interesting, productive, or answerable question. For example, in discussion Fagan said, "I don't know if it buys us anything to get so hung up on all of these terms, like recall, consciousness, imitation, and so forth. What we have is a set of paradigms that show us what sort of information the infant can encode, at particular ages, under certain conditions, at what speeds. . . ." But Mandler countered: "I disagree for the following reason: I don't know how to

generalize from one paradigm to another to make my predictions and to make my explanations unless I have a theory of the processes that underlie it."

The book's contributors also suggest criteria one might use to determine the existence of explicit memory from behavioral measures. One set of criteria concerns whether the behavior displays the characteristics of, and is sensitive to the parameters affecting, known instances of explicit or implicit memory. For example, Roediger points out that, in studies of memory in adults, repetition appears to have profound effects on explicit memory, but not on implicit memory: "If you expose a stimulus once versus several times, and multiple exposures have no effect on performance, then at least by analogy to adult findings, the test is more likely to be implicit. The number of repetitions has a profound effect on virtually all explicit memory tests for adults. By comparison, its effect on implicit tests is minimal." Yet, applying such criteria can be tricky for, as Bachevalier points out, in studies of memory in animals, repeated presentation has seemed more important for *implicit* memory. Information that we assume is remembered implicitly is often acquired gradually over multiple presentations, whereas information that we assume is remembered explicitly is often acquired after a single presentation.

Another approach to determining whether a behavior indicates implicit or explicit memory is to determine the neural bases for known instances of explicit or implicit memory. Then, behaviors dependent on one of these neural systems, but not on others, might be considered indicative of the kind of memory subserved by that neural system. For example, **amnesia** is characterized by a severe impairment in explicit memory but preserved implicit memory. Amnesic patients may not consciously remember having ever seen or done a task before, but their performance on the task nevertheless shows a normal learning curve of progressive improvement. Damage to the **hippocampus** and closely related structures in the medial temporal lobe (such as **entorhinal cortex** and the **parahippocampal gyrus**; see FIG. 5) appears to be one of the principal causes of amnesia. Performance on the **delayed non-matching to sample task** is impaired in monkeys with lesions of the hippocampus and closely related structures, and performance on this task is impaired in human adults with amnesia. This has led many to conclude that the delayed non-matching to sample task is a measure of explicit memory, even though the task measures memory through action and requires no verbal report.

In delayed non-matching to sample, a sample object is presented, which the subject displaces to receive a reward. A delay is imposed. Then, the sample object is presented again along with a new object; now the reward is hidden under the new (non-matching) object. Subjects are supposed to displace the new object and are rewarded for doing so by being allowed to retrieve the reward. The procedure is repeated over several trials with different stimuli on each trial ("trial-unique stimuli"). Memory duration is studied by varying the delay between the sample and paired presentations.

In their chapter, Zola-Morgan and Squire review results where exactly the same tasks have been administered to monkeys and to amnesic patients, to similar patient groups in different labs, and to patients with different forms of amnesia in the same lab. This has provided the basis for precise, quantitative comparisons of performance across many laboratories and subject populations. The most well-studied task used with both monkeys and amnesic patients is the delayed non-matching to sample task.

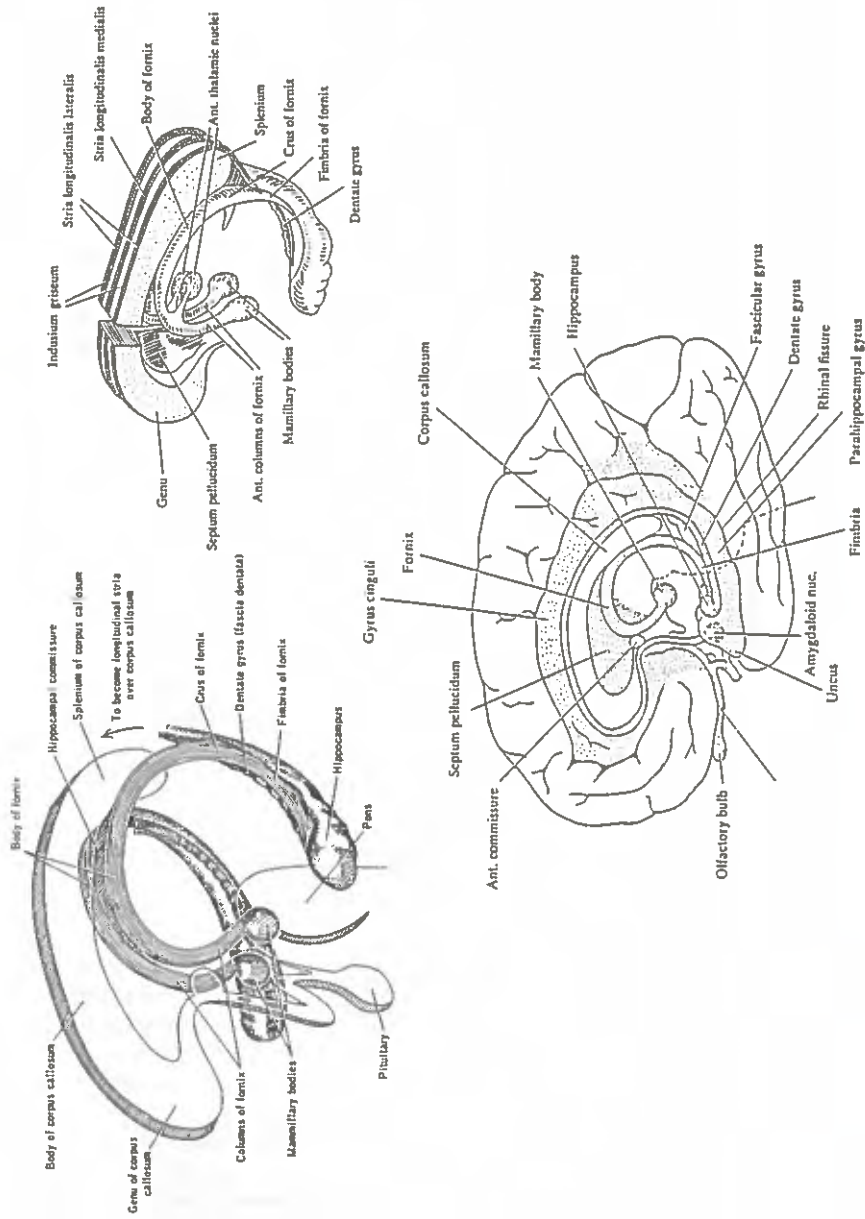


FIGURE 5. Views of the hippocampal system showing component structures, fiber tracts, and the location of the system inside the brain, as viewed from the medial surface of the left hemisphere. (Above left reprinted from figure 2-19 in E. L. House & B. Pansky (1967), *A Functional Approach to Neuroanatomy*, NY: McGraw-Hill Book Co. Reprinted from a figure on p. 345 (above right) and adapted from a figure on p. 339 (below) of B. Pansky (1980), *Review of Neuroscience*, NY: MacMillan. All three figures drawn by Ben Pansky.)

It has been known for several years that monkeys with lesions to the hippocampal system are severely impaired on delayed non-matching to sample. Now, performance on this task by monkeys with lesions to precisely localized sites within the hippocampal neural circuit is being used to determine the critical component structures within this system, the neural system that appears to underlie explicit memory. Advances in imaging techniques are also improving our ability to determine the sites of structural brain damage in patients with amnesia.

Both amnesic patients and monkeys with damage to the hippocampal neural circuit are able to succeed on the delayed non-matching to sample task at delays of 5 sec or so; at longer delays (such 1 min or 10 min) they fail. In this respect the performance of amnesic patients and hippocampally operated monkeys are comparable. However, the match is not perfect. For example, hippocampally operated monkeys generally display a more severe deficit on the task, in absolute terms and compared to controls, than do amnesic patients.

The careful delineation of the characteristics of amnesia provided by Zola-Morgan and Squire is exactly the kind of information required to evaluate whether, and to what extent, monkeys with lesions of the hippocampal neural circuit constitute a good animal model of amnesia. The behavior of the monkeys can then be assessed on each of the diverse dimensions for which information is available concerning amnesia. For example, one of the characteristics of the memory loss in amnesia is that, although material learned long ago is remembered well, information encountered since the onset of the amnesia is often forgotten after a few minutes ("anterograde amnesia"). Moreover, information acquired long before the onset of the amnesia is remembered better than information acquired closer to the time of the amnesia's onset. This is called "temporally graded retrograde amnesia." Zola-Morgan is finding that, not only do monkeys with lesions of the hippocampal neural system fail delayed non-matching to sample while succeeding on tests of implicit memory, but they show the same temporally graded retrograde memory loss characteristic of amnesic patients.

Overman summarizes the developmental progression in human infants' performance on the delayed non-matching to sample task using a longitudinal testing procedure approximating as closely as possible the procedure used in testing monkeys. For example, the children were tested every weekday for many weeks, as is typically done in testing monkeys. Diamond summarizes the developmental progression in human infants' performance of the delayed non-matching to sample task using a cross-sectional design approximating as closely as possible the procedure used with adult amnesic patients. Both Overman and Diamond report that infants do not begin to perform well on the task until about 20-21 months of age even with delays as brief as 5-10 sec. These results highlight the remarkably small role of practice in improving performance on this task, and hence the large role presumably played by maturational factors. Infants tested for the first time on delayed non-matching to sample at 21 months perform at levels comparable to infants of the same age who had received daily testing for months. Indeed, Overman and his colleagues found that the age at which longitudinal testing began (between 12 and 20 months) had virtually no effect on when mastery of delayed non-matching to sample was achieved.

Bachevalier summarizes the developmental progression in infant monkeys' performance on the delayed non-matching to sample task, as assessed by the same



longitudinal testing procedure used by Zola-Morgan and others with adult monkeys and used by Overman with human infants. Success on delayed non-matching to sample appears quite late in infant monkeys, just as it does in human infants, even at delays as brief as 5–10 sec. In addition, Bachevalier also reviews the results of herself and colleagues on the neural basis for delayed non-matching to sample performance in infant and adult monkeys. In infant monkeys, as in adult monkeys, success on the task appears to require involvement of the hippocampus.

Neonatal lesions of inferotemporal cortical area TE, however, do not impair delayed non-matching to sample performance in infant or adult monkeys, even though such lesions performed in adulthood *do* impair performance on the task. It appears that the sparing of function following neonatal lesions of area TE is made possible, at least in part, by the preservation and expansion of **exuberant projections** from neighboring area TEO to the amygdala and hippocampus. In adults, the amygdala and hippocampus project to TEO, but TEO does not project back. In neonates these projections are reciprocal, however, at least through the first month of life in the monkey. In monkeys with neonatal lesions of TE, these projections from TEO appear to continue to be reciprocal throughout life—and these exuberant TEO projections appear to be *functional*, as performance on delayed non-matching to sample falls if the projections are removed. Lesions of area TEO produce no effect on delayed non-matching to sample performance in normal adult monkeys. However, such lesions do impair performance in adult monkeys who had received lesions of area TE as neonates.

It turns out that the delayed non-matching to sample task, which was developed primarily for work with adult monkeys, is very similar in general form to the **visual paired comparison task**, which was developed primarily for work with human infants. In both tasks, a sample is presented, a delay imposed, and then the sample is presented again along with a new stimulus. What the experimenter is interested in is which stimulus will the subject choose—the sample or the new, non-matching stimulus? Since infants and monkeys are inherently curious and have a natural preference for novelty, memory of the sample is usually inferred from consistent choice of the new stimulus.

In visual paired comparison testing, subjects are allowed to look at the sample until they get bored (i.e., reach a habituation criterion) or for a set period of time (e.g., 20 or 60 sec). When the sample and new stimulus are presented together, the experimenter records how long the subject looks at each. Choice of the new stimulus is indicated by looking longer at the new stimulus than at the now familiar sample.

Fagan outlines the developmental progression of human infants' performance on the visual paired comparison task he pioneered. He begins by discussing methodological issues that were addressed in developing the visual paired comparison technique. For example, he provides evidence that visual fixations can be reliably recorded from infants with good agreement among observers. Then he considers parameters that influence performance on the task, such as age of the subject, nature of the stimuli, initial familiarization time with the sample, and the length of delay between sample presentation ("familiarization" or "study") and the paired presentation of the sample and new stimulus ("test").

By comparing infants equated for conceptual age and for postnatal age, Fagan and his colleagues have been able to begin to explore the roles of maturation and

experience in the development of the abilities required on the visual paired comparison task. Concerning the nature of the stimuli, Fagan and his colleagues have shown, for example, that infants may be more likely to demonstrate recognition of a stimulus' (such as a person's face) if they are shown multiple views of the stimulus during familiarization, rather than seeing the stimulus from only a single perspective. This is true even if the sample is displayed from that single perspective when paired with a new stimulus during the test phase.

The visual paired comparison task can be used to assess infants' speed of encoding by varying the length of familiarization time with the sample. Or, the task can be used to study infants' recognition memory by varying the delay between familiarization and test. Finally, by varying the stimuli paired with the sample on test, the visual paired comparison task has been used to assess infants' ability to detect differences and invariances among stimuli (e.g., Is a stimulus viewed from a different angle the same stimulus? Is a photograph of an object a new stimulus or is it equivalent to the actual object presented during familiarization?).

Rose and her colleagues have been able to show that what was thought to be a qualitative shift during infancy from preference for familiarity to preference for novelty is actually a quantitative improvement in speed of encoding. Younger infants need more time to encode the sample stimulus. A given presentation time is likely to be too brief for them to have fully encoded the sample; hence they will prefer to continue to look at the sample when it is presented again during the test phase. If presentation time is increased, even very young infants will look preferentially to the novel stimulus during test. Moreover, if the presentation is extremely brief, even older infants will prefer to continue to look at the familiar sample during test.

Finally, Fagan suggests that visual paired comparison performance in infancy may predict later intelligence. Throughout much of the chapter, he discusses the visual paired comparison task as a measure of recognition memory, but when he discusses it as a measure of intelligence (especially in the discussion) he speaks of it primarily as a measure of speed of encoding. Indeed, infants with Down syndrome are impaired on the task, but the difference between their performance and that of controls largely disappears if study time is increased. Nadel and Diamond sound cautionary notes, though, about the use of intelligence tests. Nadel reminds us that any yardstick of intelligence measures only a subset of the range of cognitive abilities. All of us would be the worse if only certain abilities were encouraged but not others, that is, if any one yardstick were adopted as *the* measure of intelligence. Diamond reminds us of work in social psychology, such as in labeling theory. If a test indicates a child is "dumb" (even if the test results are incorrect), that child will often come to consider himself dumb and perform accordingly. Similarly, adults will believe the child is dumb, and will treat him or her accordingly. The cost to the individual, and to society as a whole, of such mislabeling can be great indeed.

To complement Fagan's summary of the developmental progression of human infants on the visual paired comparison task, Bachevalier outlines the developmental progression of infant monkeys' performance on the task, and the neural system required for success on the visual paired comparison task in infant and adult

<sup>1</sup>Remember that recognition of the sample is demonstrated in the visual paired comparison task by preferential looking at the new stimulus (e.g., the new face).

monkeys. Preferential looking to the novel stimulus (demonstrating memory of the sample) is shown very early on the visual paired comparison task in infant monkeys, just as it is in human infants. In contrast, memory of the sample is not demonstrated in either monkeys or humans on the delayed non-matching to sample task until quite late. Perhaps the differences between the visual paired comparison and delayed non-matching to sample tasks are more important than their similarities. On the other hand, however, lesions to the hippocampus + amygdala impair performance on both tasks in both infant and adult monkeys.

On the basis of the requirements of the task alone, one might predict that visual paired comparison is a measure of implicit memory "... in the sense that we're not explicitly asking subjects to go to one or the other, it's just a preference measure, sort of like studying adaptation level or priming" (Shimamura). Subjects don't know they are being tested and receive no feedback about what they should do or what response is right or wrong. On the other hand, the visual paired comparison task appears to be generally similar to delayed non-matching to sample, which is thought to measure explicit memory based on lesion studies and work with amnesic patients. Indeed, performance on the visual paired comparison task is disrupted by lesions thought to disrupt explicit memory (lesions of the hippocampus + amygdala).

It may turn out that visual paired comparison requires only implicit memory, and that lesions to the hippocampal memory system excluding the amygdala will not impair performance on the task as they do delayed non-matching to sample performance. In other words, the hippocampus may be more important for performance of delayed non-matching to sample, but the amygdala may be more important for visual paired comparison performance. Patients with amnesia may, perhaps, succeed (i.e., look preferentially to the new stimuli) when tested on visual paired comparison. Or, it may be that both delayed non-matching to sample and visual paired comparison are measures of explicit memory and that delayed non-matching to sample is not mastered until so much later because of requirements of the task other than its memory requirements. Indeed, while it had been thought that the hippocampus was a late-maturing structure, Diamond and Bachevalier cite anatomical and behavioral evidence suggesting that it matures quite early in primates.

What, then, might be the late-maturing ability required for success on delayed non-matching to sample but not on visual paired comparison? Mishkin has suggested, and Overman and Bachevalier agree, that this may be the ability to abstract the quality of novelty from constantly varying stimuli and to learn the rule that an external reward is consistently associated with this intangible quality. "Perhaps [infants] can readily learn to associate responses with particular qualities as required in discrimination learning tasks but not with the object's abstract qualities" (Bachevalier). Overman calls this "novelty abstraction" and "learning of the novelty-reward rule."

Perhaps this is the critical ability, but infants seem to respond well at a very early age to the abstract quality of novelty on the visual paired comparison task. Also, telling infants the rule so that they do not have to deduce it, or leaving the written rule in front of amnesic patients so they can refer to it as needed, seems to have little effect on performance.

Diamond considers these possibilities and others in her chapter. For example, resistance to interference is another plausible candidate for the critical late-maturing

ability. In the visual paired comparison task nothing intervenes between sample and test, but in the delayed non-matching to sample task subjects receive a reward after displacing the sample. In her work on the  $A\bar{B}$  task with multiple wells (Diamond, Section 3), Diamond showed the powerful effect interference can have on infants' performance; perhaps infants would succeed on delayed non-matching to sample at a much younger age if they received no reward after the sample was presented. Or, speed of encoding might be a critical ability here. The sample is typically presented much longer during visual paired comparison testing (often 20–30 sec) than during delayed non-matching to sample testing (typically 2–5 sec). If the sample were presented for a longer period, perhaps infants would succeed on delayed non-matching to sample at a much younger age.

The work on the neural basis and developmental progression of performance on the delayed non-matching to sample task provides a valuable lesson. It has been convincingly shown, by work with adult monkeys and adult amnesic patients, that this task requires the memory functions dependent on the hippocampus and related structures. It has also been shown that neither children nor monkeys can succeed on this task until relatively late. A natural conclusion, and one that many initially drew from this set of results, is that the hippocampal system and the memory functions it subserves mature relatively late. However, most would now agree that this conclusion is not correct. Instead, the hippocampus appears to mature relatively early. Another ability on which delayed non-matching to sample depends evidently matures late. This illustrates the importance of using caution in generating conclusions about development from work with adults, the importance of converging lines of work in anatomy and behavior in the same population, and the importance of fine-grained task analysis. For example, while it is true that both adults with hippocampal damage and infants fail the delayed non-matching to sample task, analysis of the parameters affecting their performance indicates that they appear to fail for different reasons. For example, the delayed non-matching to sample performance of adults with hippocampal damage is sensitive to length of delay: They perform well at very brief delays; their performance worsens as the delay increases. Infants, however, show a flat performance  $\times$  delay function: Those who fail do so at the very briefest delay; those who succeed do so across a wide range of delays.

The use of the same task with different subject groups, which is generally so well regarded, was the basis for much disagreement among the participants at the meeting reported in this book (see discussion following the paper by Overman). Diamond and DeLoache asserted that often a task must be modified so that it is appropriate for a different subject population. Nadel, Frost, and Overman argued strongly, however, for the need to use *exactly* the same task with all populations: "The simple logical point is that if you do something different you know you are doing something different. If you do something the same, you at least have the possibility that the things you are doing really are the same" (Nadel). Both positions have an important point. On the one hand, you would not want to use monkey chow as the reward for human subjects, nor would you want to test American children's understanding of science by questions written in French. Otherwise, the test would be measuring knowledge of science among French children, but measuring knowledge of French among American children. On the other hand, any modification to a test might change the test in some critical way so that it is now sensitive to different

variables. One can never know a priori which modifications will be innocuous and which modifications will be critical. One can, however, try to put this to empirical test.

#### SECTION 5: IMPLICIT AND EXPLICIT MEMORY II: PRIMING, REACTIVATION, AND RECALL

Section 5 contains 2 sets of sharply contrasting viewpoints and approaches. While **Mandler** is concerned with the development of consciousness and explicit memory, **Rovee-Collier** is concerned with a functional analysis of memory as shown through behavior, without regard to whether the memory is conscious or not. Although the tasks they use could not be more different, each sees her own task as a measure of cued recall. **Schacter** presents evidence that implicit and explicit memory are fundamentally different, and dissociable, forms of memory, and indeed that there may be multiple, dissociable memory systems for different types of implicit memory. However, **Roediger**, **Rajaram**, and **Srinivas** then present evidence that implicit and explicit memory may *not* depend on fundamentally different memory systems; indeed there may be only one memory system.

**Mandler** tackles the question of how one might provide evidence of explicit memory in infants. First, she considers classical conditioning, operant conditioning, and looking where a stimulus is expected before it appears (visual anticipation). She concludes that while learning in each of these tasks indicates memory, that memory need not be conscious or explicit. **Deferred imitation**, however, meets Mandler's criteria of a task that requires explicit memory. Here, the memory cannot be built up gradually since it is assessed after only one or very few presentations; no overt motor response is made at the time of acquisition which might later be unconsciously repeated; the delay (24 hours–2 weeks) is sufficiently long that the correct response cannot simply be read off a temporary, perceptual record. **Meltzoff**, who pioneered the use of this technique, describes in his chapter how 9-month-old infants, shown a single action just once and not allowed to practice it, can reproduce that action 24 hours later when brought back to the laboratory.

**Mandler** extends this technique to longer action sequences, longer delays, and older children (13–20 months old). She reports studies by herself and colleagues on infants' imitation of action sequences 2–3 actions in length modeled by the experimenter ("infants' cued recall of event sequences"). The cues are the props the experimenter used in the demonstration. Recall is tested after delays of a few seconds ("immediate imitation") and 2 weeks ("deferred imitation"). Although children execute the actions on the immediate recall test, **Mandler** argues that such experience on a single trial could not serve as the basis for establishing a conditioned response; explicit memory is still required for success on the deferred recall test. Some of the sequences are familiar to the children; others are novel but causally related; still others are novel and arbitrarily ordered, yet meaningful in the sense that the items were related to one another.

In the discussion following **Mandler's** paper, the meeting participants hotly debated whether **Mandler** and **Rovee-Collier** are studying similar or fundamentally different types of memory. Are there any circumstances under which conditioning or

visual anticipation might require explicit memory? Does deferred imitation necessarily require explicit memory? Are explicit and implicit memory fundamentally different? If they are, one approach to determining which type of memory is required by a task is to apply the first set of criteria mentioned above; that is, determine whether infants' behavior on a task displays the characteristics of, and is sensitive to parameters affecting, known instances of explicit or implicit memory. In this regard, **Mandler** calls for specifying explicitly the requirements for demonstrating conscious memory in infants, such as 1-trial learning, perhaps.

**Nakamura** suggests investigating the performance of amnesic patients on deferred imitation as a way to begin to empirically address whether the task requires implicit or explicit memory. Others suggest looking at the performance of monkeys with lesions of the hippocampus and closely related structures, or the performance of monkeys with lesions of dorsolateral prefrontal cortex (since that is thought by some to subserve memory of temporal order, which might be required to remember the sequence in which actions were performed). This illustrates the second set of criteria mentioned above: "Determine the neural bases for known instances of explicit or implicit memory. Then, behaviors dependent on one of these neural systems, but not on others, might be considered indicative of the kind of memory subserved by that neural system." Most meeting participants agreed that task analyses alone, in the absence of information on the neural basis for successful performance, would never be sufficient to determine whether deferred imitation, in fact, requires explicit memory.

Investigation of the neural basis without analysis of the task requirements, however, also ends up being unilluminating. Both avenues must be pursued. Explicit memory is still insufficiently understood to enable us to predict with accuracy how amnesic patients or hippocampally operated monkeys will perform on a given test. Typically, explicit memory is operationally defined as the memory ability that amnesic patients and monkeys with lesions of the hippocampus lack. Similarly, a test is typically said to require implicit memory if these two subject populations succeed. Ultimately, however, "defining these systems in terms of whether a particular task is hippocampally dependent or not just doesn't seem to get you anywhere. . . . It just gets you a class of tasks that are affected by hippocampal lesions. That tells you nothing about what the hippocampus is doing unless you know what the tasks are measuring. So there has got to be some principled understanding of what the task requirements are. . . . What is there in common amongst [a set of tasks]? What kinds of information are being accessed and utilized?" (**Nadel**, in the discussion of **Bachevalier's** paper).

**Rovee-Collier** uses an operant conditioning paradigm with **conjugate reinforcement** to study memory in infants, and to derive precise answers to the question of what kinds of information are being accessed and utilized. A ribbon is tied to the infant's ankle and to a mobile overhead, allowing the infant to control the frequency and intensity of the mobile's movements. The reward is the movement of the mobile. The response is rate of kicking. The cue is the sight of the mobile. Infants are trained with their leg connected to the mobile; they are tested before and after the training portion of each session with the ribbon disconnected from the mobile. Infants as young as 2 or 3 months learn quickly in this paradigm (doubling or tripling their rate

of kicking within a few minutes), and they continue to remember what they have learned for quite a long time.

After a long enough interval has elapsed so that infants no longer show evidence of retention in their behavior, Rovee-Collier and her colleagues present a brief reminder consisting of some element(s) from training (such as the distinctive crib bumper used during training, or the mobile disconnected from the infant, but moving at the same rate as that at which the infant had moved it during the final minutes of training). The presentation of the reminder is called a "reactivation treatment." Rovee-Collier and her colleagues have systematically varied the delay (from minutes to weeks) between training and test, training and reactivation, and reactivation and test; degree of similarity between the mobile at training, reactivation, and test; and degree of similarity of the context in which training, reactivation, and test are conducted, to yield an impressive body of work. In her chapter, Rovee-Collier discusses general principles of learning and memory that have emerged from this work.

For example, just seeing the reminder for 2-3 min can reinstate the infant's rate of kicking to its full post-training level, even though, judging by the infant's behavior, the training had been completely forgotten before the reminder. This indicates that the memory was still present at some level, even after all evidence of memory had disappeared from behavior.

Rovee-Collier and her colleagues have found that if the training occurred in a novel context, exposure to that context alone can reactivate a forgotten memory. However, if the mobile is presented in a different context than that used for training, infants act as if they don't remember the mobile. This indicates that infants must encode contextual information as part of their memory of the training episode. Indeed, results are similar whether a change is made in the proximal context (e.g., changing the crib or the liner in the crib) or distal context (e.g., using the original crib and crib-liner but in a different room). Rovee-Collier suggests that "young infants learn *what happens in what place* long before they are able to locomote from one place to another. . . ."

It is interesting to consider the process happening inside an infant after exposure to a reminder. Memory (of the association between their kicking and the mobile moving) is recovered very slowly. Infants of 6 months show no evidence of remembering this a half hour after the reminder, although 4 hours later they do. Three-month-old infants show no evidence of retention until 8 hours after seeing a reminder. Infants tested one day after a reminder respond equivalently to the original mobile or a novel one. Three days later, when they remember better, they discriminate between the original and novel mobiles. Thus, once evidence of memory has disappeared from behavior, the specifics are retrieved more slowly than the general outlines. This is the mirror-image of the decay function following training, where initially only the familiar mobile elicits the conditioned response, but later any mobile will do. Note that this suggests that the delay interval at which memory is tested will affect one's conclusions about what infants encode and remember, since the contents of newly acquired memories and of reactivated old memories change over time. They may also change over age. There is some evidence that the decay function for 6-month-olds is different from that just described for 2- and 3-month-olds. There may never be a time when "any mobile will do" for infants of 6 months; as long as they show any evidence

of learning, they discriminate between the training mobile and other mobiles. This suggests that important changes in memory processes may occur between 3 and 6 months of age.

The most common way that explicit memory is studied in adults is by tests of **recall** or **recognition**. (For example, subjects are asked if they can recall the words that were previously presented, or if they can recognize them from a list containing both new and previously presented words; see TABLE 1.) The most common way that

TABLE 1. Examples of Tests of Explicit Memory

#### I. VERBAL TASKS

##### Free Recall

Subjects are asked to remember something (e.g., a previously studied word or past event) without the aid of any cue.

##### Semantic Cued Recall

Subjects are given a cue to help them remember. The cue is a word related in meaning to the one they are asked to recall, e.g., "venom" as the cue for "hemlock."

##### Graphemic Cued Recall

Subjects are given a cue to help them remember. The cue is a word that looks and/or sounds like the one they are asked to recall: e.g., "hamhock" as the cue for "hemlock." Subjects are instructed to recall the word that most looks or sounds like the cue from the list of words they studied earlier.

##### Paired-Associate Learning

Subjects are given the first word of an arbitrary pair of words and are explicitly asked to recall, from the previously presented word pairs, the word that goes with the one presented: e.g., ship - castle. Memory is directly shown by subjects' greater tendency to produce previously studied words and to produce them to the appropriate cue word.

##### Recognition

Subjects are asked to remember (e.g., previously studied words or past events) and are given more than one choice, one of which is the correct answer. Subjects indicate they remember by selecting the correct choice from the alternatives offered.

#### II. NON-VERBAL TASKS

##### Delayed Non-Matching to Sample

Subjects displace a sample object to receive a reward. After a delay, subjects are given the choice of displacing the sample object or a new object that does not match the sample. Only choice of the non-matching object is rewarded. New stimuli are used on every trial ("trial-unique objects"). Recognition memory of the sample is indicated by subjects' consistent choice of the novel, non-matching object.

implicit memory is studied in adults is by tests of **priming**. Here, subjects are instructed to give the first response that comes to mind; no reference is made to the previously presented material, and it is hoped that subjects will not realize that the previously presented material is relevant to the test. On a priming test a subject might be asked, for example, to complete a word stem or word fragment with the first

TABLE 2. Examples of Tests of Implicit Memory

I. PRIMING TASKS	
IA. Lexical Priming	
<b>Word Stem Completion</b>	Subjects are asked to complete a word from a stem (usually 3 letters): e.g., c a s _ _ _ for castle. Memory is inferred from a higher rate of success and/or shorter time to completion for stems of previously presented words than for stems of words not previously presented, and/or from an enhanced tendency to complete these stems with previously studied words. Comparison groups are those who studied no words or different ones (e.g., casket or cashew).
<b>Word Fragment Completion</b>	Subjects are asked to complete a word from a fragment: e.g., c _ _ _ l _ for castle. Memory is inferred from a higher rate of success and/or shorter time to completion for fragments of previously presented words than for fragments of words not previously presented, and/or a greater tendency to complete fragments with previously studied words than with words not previously presented that would nevertheless also complete the fragment.
<b>Word Identification I (also called Tachistoscopic Identification; an instance of a perceptual identification task)</b>	Subjects are asked to identify a word from a very brief exposure (e.g., 50 msec). Priming is shown by faster or more accurate identification of recently studied words than of words not previously studied.
<b>Word Identification II (another perceptual identification task)</b>	Subjects are asked to identify a word from perceptually degraded features (e.g., very faint letters and/or pieces of the letters). Memory is inferred from quicker or more accurate identification of recently studied words than of words not previously studied.
<b>Lexical Decision Task</b>	Subjects are asked if a string of letters constitutes a real word or not. Priming is indicated by subjects making these decisions more quickly for previously studied words and nonwords than for ones not previously presented.
<b>Category Instance Production</b>	Subjects are asked to generate instances of a category: e.g., "name as many articles of furniture as you can in 30 sec." Memory is inferred from a greater tendency to produce previously studied words (e.g., "armoire") than is the case when these words have not been previously presented.
<b>Priming of Utilized Phrases</b>	Subjects are given the first word of a pair and are asked to provide the second word: e.g., sour - _ _ _ for sour-grapes. Memory is inferred from a greater tendency to produce the word "grapes" if the phrase "sour grapes" was recently presented than if it had not been previously presented.
<b>Priming of New Associations</b>	Subjects are given the first word of an arbitrary pair of words and are asked to free associate to a second word: e.g., ship - _ _ _ when previously presented pairs included "ship - castle" and "table - window." Memory is inferred from subjects' greater tendency to produce previously studied words and to produce them to the appropriate cue word. Comparison groups are those who studied no word pairs or pairs with the same cue words but different second words. This test probably requires both explicit and implicit memory.

word that comes to mind (see TABLE 2). Priming is shown by a facilitated tendency to give previously presented items in response, as if they come more easily to mind.

Various experimental manipulations, and different types of brain damage, have different effects on recall and recognition than on priming. For example, priming effects are strongest when the perceptual characteristics (surface features) of the items presented at test most closely resemble the perceptual characteristics of the

TABLE 2. Continued

<b>Spelling</b>	Memory is inferred when subjects give the less common spelling of a homophone when they had recently encountered that less common word (e.g., if they offer the spelling "reed" rather than "read").
<b>Anagram Test</b>	Subjects are given anagrams of studied and unstudied words (e.g., "ecalts" for castle). Memory is inferred from superior performance in solving anagrams of studied rather than unstudied words.
IB. Priming of Non-Verbal Information (e.g., Pictures)	
<b>Object Fragment Completion (analogous to Word Fragment Completion or Word Identification II)</b>	Subjects are shown fragmented pictures and are asked what object the fragments represent. Memory is inferred from more accurate, and/or quicker, identification of recently presented objects than of objects not previously studied.
<b>Identify an Object from a Brief Exposure (analogous to Word Identification I)</b>	Subjects are asked to identify an object from a very brief exposure to it (e.g., 50 msec). Priming is shown by faster or more accurate identification of recently presented objects than of objects not previously studied.
II. MOTOR SKILL LEARNING	
<b>Pursuit-Rotor Task</b>	Subjects must learn to keep the point of a stylus on a moving target. Improved performance, or sustained good performance on later re-test, is taken as evidence that previous exposure to the task has been "remembered" at some level.
<b>Lifesaver Task</b>	Subjects must learn to maneuver a lifesaver candy along a metal rod and around a right-angle turn. Improved performance, or sustained good performance on later re-test, is taken as evidence that previous exposure to the task has been "remembered" at some level.
<b>Mirror Tracing</b>	Subjects must learn to keep their pencil within a narrow, winding path on the paper while looking at the reflection of this path in the mirror. Improved performance, or sustained good performance on later re-test, is taken as evidence that previous exposure to the task has been "remembered" at some level.
III. OTHER	
<b>Mirror Reading</b>	Subjects learn to read certain words in the mirror, and then show improved performance in reading other, new words in the mirror, and in reading the studied words, despite a failure to recognize that these words had been presented before.
<b>Visual Discrimination (also called Pattern Discrimination or Object Discrimination)</b>	Subjects learn, from feedback about whether their response was right or wrong, which of two stimuli (e.g., a cross or a square) is correct, regardless of its spatial location. Memory of which stimulus is correct is shown by successful performance.

items presented during study. These surface features include sensory modality (e.g., seeing a word versus hearing the word), pictures versus words (e.g., a picture of a car versus the word "car"), or the same words handwritten versus typed. The closer the match between these characteristics of the stimuli at study and test, the more robust the priming. Thus, earlier study of pictures enhances performance on picture fragment completion, but not on word fragment completion; words prime word

fragment completion better. Recall and recognition performance, however, is generally not affected by the surface features of the stimuli, and sometimes is affected in the opposite direction. For example, recall or recognition of a word is better if a picture of the word was studied earlier, rather than the word itself (the opposite of what is found for priming of a word). Amnesic patients often show preserved priming, despite severely impaired recall and recognition. Findings such as these have prompted Schacter and many others to postulate that explicit and implicit memory are fundamentally different forms of memory, dependent on distinct neural systems.

Schacter further proposes that within the implicit memory domain there are multiple subsystems (independent modular processors). He calls the subsystems collectively underlying priming the "perceptual representation system" (PRS). Perceptual representation subsystems "process information about the *form* and *structure* [of stimuli], but do not represent *semantic* or *associative* information about them." Each subsystem is an independent entity with its own particular neural basis. Schacter identifies two such subsystems: the "word form system" and the "structural description system." The word form system is dedicated to the representation and/or retrieval of the visual form and structure of words, but not their meaning. The structural description system represents information about the form and structure of common visual objects, but not information about what the objects are or how they are used.

For evidence of these two implicit memory subsystems, Schacter turns to the distinctive patterns of deficits and preserved functioning found in patients who have acquired reading disorder (alexia) and patients who are unable to recognize familiar objects (patients with visual agnosia). These two classes of patients had not heretofore been considered in relation to issues of implicit versus explicit memory. Schacter marshals the evidence convincingly, however. For example, some patients can no longer tell you the meaning of printed words, but they can pronounce even irregular words (e.g., "blood") correctly. The correct pronunciation of irregular words cannot be derived on the basis of phonological rules; it can only be done on the basis of memory. Such patients, therefore, appear to have preserved access to stored representations of the word's visual form, but not to stored representations of the word's meaning. Other patients are forced to rely on grapheme-to-phoneme conversion rules and hence mispronounce irregular words as if they were regular. Such patients appear to have an impairment in the word form system.

Roediger, Rajaram, and Srinivas, however, wonder whether the evidence really warrants postulating separate systems for implicit and explicit memory, much less fractionating these still further into more circumscribed subsystems. They examine whether 4 criteria for distinguishing separate memory systems have been satisfied. The criteria are: (1) functional dissociations among tests, (2) different underlying neural substrates, (3) stochastic independence, and (4) functional incompatibility.

Dissociations are, in fact, found in the effects of different variables on priming versus recall and recognition, but Roediger and his colleagues point out that the type of memory (implicit or explicit) required by a task has typically been confounded with the type of information processing required by the task. Tests of implicit

memory have usually required only perceptually based (data-driven) processing. Such processing relies heavily on the match between perceptual (surface) features at study and test; it can often be supported by superficial study (e.g., reading a word out of context or counting the number of vowels in the word). Tests of explicit memory, on the other hand, have generally required semantic or elaborative (conceptually driven) processing (e.g., thinking about what the word means or generating the word from a conceptual clue). Such processing is little affected by changes in perceptual features of the material.

To disentangle type of memory from level of processing, Roediger and Blaxton administered a perceptually driven explicit memory test ("graphemic cued recall," in which subjects were asked to recall the word from the previously presented list that most looks or sounds like the cue word), a conceptually driven explicit memory test ("semantic cued recall," in which subjects were asked to recall the word from the previously presented list that is closest in meaning to the cue word), and perceptually and conceptually driven implicit memory tests. They found that each independent variable had the same effect on all perceptually driven tests, and the opposite effect on all conceptually driven tests. Moreover, experimental manipulations had dissociable effects on the performance of tests thought to rely on the same hypothetical memory system (implicit or explicit) when those tests required different types of information processing. Hence, information processing requirements appear to account for the data better than type of memory system. Performance on each memory test was better if the encoding operations during study matched the retrieval operations during test. This illustrates the general principles of encoding specificity and transfer appropriate processing, which state that what is important is the match between the way information is processed at study and test. Roediger, Rajaram, and Srinivas conclude that previous demonstrations of dissociations between performance on tests of implicit and explicit memory have been due to the processing requirements of the tests, not the type of memory required.

While the first criterion poses problems for multiple memory system views, the second criterion poses problems for transfer appropriate processing views. Damage to different neural systems does, in fact, produce very different effects on memory test performance. It is difficult to reconcile the pattern of impaired and preserved memory in amnesia with the notion that there is but one memory system.

Stochastic independence (criterion #3) refers to the lack of correlation between what information is recalled or recognized and what information is primed. It is not simply that memory of an item must be more robust for it to be recalled or recognized, so that recalled items form a subset of the primed items; *different* items are recalled from those primed. This would suggest that the memory systems supporting recall and priming may be separate. However, stochastic independence can also be found among priming tests or among recall tests. A well known example is that priming for the word "assassin" tested by "a \_ \_ a \_ \_ \_" is uncorrelated with priming for the same word tested by "\_ ss \_ ss \_ \_"

Functional incompatibility (criterion #4) is the principle that one should postulate independent systems only if there is evidence that there are functions that a single system could not perform. For example, certain functions might be incompati-

ble with other functions of a system if they require fundamentally different principles of operation.

Although the answer to whether there is more than one memory system, and if so, how many, is not presently known, Schacter as well as Roediger, Rajaram, and Srinivas do an excellent job of clarifying the issues and of presenting the arguments for the two principal opposing views.

## SECTION 6: SPATIAL ORIENTATION, REPRESENTATION, AND MAPPING

Acredolo and Nadel address questions of how we keep track of where things are, and where we are, as we move about in space. In her own work, Acredolo has concentrated on the development of landmark knowledge in human infants. Nadel's work, on the other hand, has been on the development of spatial representation in rat pups, and on the neural bases for spatial representational abilities. In particular, his work has concentrated on the hippocampus, which he considers to be the core of the system concerned with representing relations between landmarks (i.e., **cognitive maps**). Both Acredolo and Nadel agree that there appear to be at least three kinds of spatial systems, each depending on a different neural circuit and having a unique developmental time course.

The most primitive system and the first to appear in development relies on "**dead reckoning**." Here, one's movements are organized with respect to frames of reference organized around one's own body (e.g., head-centered coordinates or retinal coordinates). One remembers how to find a reward by remembering the turns, or responses, one made to get there. This system relies solely on an internally generated record of one's movements (e.g., kinesthetically or through the use of the vestibular system), but it can involve quite sophisticated computation of the velocity and direction of one's movements.

Next to appear is the ability to use objects or events outside of oneself as guides or **landmarks**. However, each landmark is considered individually, rather than in relation to other landmarks as part of a larger system. Successive individual landmarks may be coordinated with movements to generate "route knowledge."

Third to appear is the ability to use landmarks in a relational, maplike manner. Here, multiple landmarks are considered simultaneously, in relation to one another. This requires that one have an internal representation or map, within which the relations among landmarks and between the landmarks and oneself are represented.

Thus, there appears to be a progression from "**egocentric**" spatial encoding using exclusively "**response**" information, to "**objective**" responding, using "**place**" information. In more general terms, the progression is from being centered on one's own body (and ignoring everything else), to attending to something in the outside world (but only one thing), to attending to more than one thing sequentially (but not yet simultaneously), to finally being able to attend simultaneously to more than one piece of information (and to being able to relate one piece of information to another). This progression can be seen in diverse domains, and may be a general principle of cognitive development. It is illustrated again in Diamond's discussion

(Diamond, Section 7) of the developmental progression on the object retrieval task, a transparent barrier detour problem.

Nadel alludes to yet a fourth system important for spatial representation: a system capable of keeping track of the temporal and spatial context, so that one knows and remembers when and where different events have occurred. Here, one is reminded of the theory of frontal cortex involvement in memory put forward by Schacter and Tulving.<sup>11</sup> They have proposed that memory for the spatial and temporal context in which something happens is distinct from memory of what has happened and, unlike memory for the latter, depends specifically on frontal cortex.

Nadel points out that spatial exploration emerges abruptly, full-blown in rat pups, from none to all in one day. To try to understand this abrupt phase transition, he introduces concepts from percolation theory. He demonstrates how a smooth, continuous increment in the underlying substrate (e.g., in the number of cells or synapses, or level of neurotransmitter) might produce an abrupt change in the observable behavior. This has implications for aging and for brain damage, as well as for development. A system may be able to withstand considerable damage or degradation without observable behavioral consequences, but then when a critical point is reached the system might suddenly, dramatically break down, or even shut off completely. In another vein, Nadel also discusses the role of early experience, and particularly stress, on hippocampal development.

Acredolo goes on to discuss how motor development may affect cognitive and perceptual development. In particular, the onset of crawling may play a critical role in the development of spatial representation and mapping. Also, as Acredolo points out, improvement in one cognitive ability (e.g., memory) may have important consequences for other cognitive abilities (e.g., spatial understanding), as once infants can keep a representation of the spatial environment in their head, constant visual monitoring is no longer so critical.

## SECTION 7: INHIBITION AND EXECUTIVE CONTROL

All of us have, on occasion, made slips of the tongue or slips in our behavior, where we intended to do one thing but did something else instead. Such slips can be considered failures of inhibition or of executive control. A slip of the tongue may occur if we are thinking of something but don't want to say it (i.e., if we fail to inhibit an inappropriate comment). A slip in behavior, and occasionally a slip of the tongue, may consist of our doing the usual, habitual, or most easily elicited action, when it was really something else we intended. On such occasions it is as if we let ourselves run on "automatic pilot" when we should have been paying attention (i.e., when we should have been exercising executive control).

<sup>11</sup>SCHACTER, D. L. 1987. Memory, amnesia, and frontal lobe dysfunction. *Psychobiology* 15: 21-36.

TULVING, E. 1989. Remembering and knowing the past. The crucial difference between remembering personal experiences and knowing impersonal facts. *American Scientist* 77: 361-368.

A few examples from the many provided by Reason, who has made an extensive study of the slips of action that occur in everyday life, can serve to illustrate this phenomenon:"

"We now have two refrigerators in our kitchen, and yesterday we moved our food from one into the other. This morning I repeatedly opened the refrigerator that used to contain our food."

"On starting a letter to a friend, I headed the paper with my previous home address instead of my new one."

"I intended to go home a different route in order to stop at a shop. . . I entered the circle, went past the turn I wanted and took the normal one home. It took about 15 seconds of pedaling down the wrong road (normally the right one) before it clicked."

"I intended to phone a friend in Leeds, but I started to dial my home phone number in London."

"On leaving the room to go to the kitchen I turned the light off, although there were several people there."

"In the course of making a cup of tea, I picked up the coffee jar instead of the tea caddy. To open the former requires an unscrewing of the lid, while the latter simply requires the lid being lifted off. My hand (apparently recognizing what it was holding, though my mind did not) unscrewed the lid of the coffee jar, and I deposited three spoonfuls of coffee into the teapot. It was only when I poured my boiling water in, and smelled the coffee that I realized my mistake."

Reason and Mycielska (1982) summarize this phenomenon thusly: "A slip of habit is one in which our actions, words or, less obviously, our thoughts are diverted by the lure of some well-trodden pathway" (p. 62). "A large proportion of absent-minded errors actually take the form of intact, well-organized segments of skilled action that are suitable for the environmental context most of the time, but not when changed circumstances require some alteration of normal practice, or when new goals demand the modification of existing routines" (p. 39-40). One may discover the mistake at various points in the execution of action (e.g., you may realize it as soon as you begin to dial the first wrong digit, or you may not realize it until someone answers the phone at the other end).

"It is perhaps appropriate that the section devoted to the deviation of action from intention should not have gone as intended. It had been intended that James Reason would discuss this phenomenon as it occurs in normal adults and in adults with frontal lobe damage. That would then be compared with Diamond's discussion of the occurrence of this phenomenon in human infants, infant monkeys, and in monkeys with frontal lobe damage. However, at the last minute Reason was unable to attend the meeting. Some of the material that would have been covered by Reason is very briefly summarized here.

The examples provided are from Reason, J. & Mycielska, K. (1982), *Absent-Minded? The Psychology of Mental Lapses and Everyday Errors*. Englewood Cliffs, NJ: Prentice-Hall. Other work by Reason on this topic includes: The price of automatization. In G. Underwood & R. Stevens (Eds.), *Aspects of Consciousness: Volume 1, Psychological Issues* (pages 67-89). NY: Academic Press, and in Reason, J. (1989). *Human Error*. NY: Cambridge University Press.

Don Norman has also written about this in normal adults (Norman, D. A. 1981. Categorization of action slips. *Psychological Review* 88: 1-15), and Tim Shallice has written about these tendencies in patients with frontal lobe pathology (Shallice, T. 1986. Failures of supervisory control: Neuropsychological parallels. *Proceedings of the Fourth International Conference on Event Perception and Action*, Trieste, Italy).

Laboratory demonstrations of this type of behavior include the well-known Stroop effect. Here, the names of colors are printed in the ink of another color. The task is to say, not the word, but the color of the ink. Normal, healthy adults who understand the instructions, and start out performing correctly, often have difficulty sustaining correct performance because the tendency to read the word and ignore the color of the ink often overrides our intention.

Patients with damage to frontal cortex show an exaggerated Stroop effect<sup>6</sup> and an exaggerated tendency for their actions to go on "automatic pilot" generally. Their behavior is often captured by a stimulus and often deviates from their intention. They are easily distracted and pulled off course.

Numerous instances of this have been noted by Luria.<sup>7</sup> Thus, for example, if a patient with frontal cortex damage is instructed to tap on the table once when the experimenter taps twice, and to tap twice when the experimenter taps once, the patient will often start out following the instructions but will quickly revert to mirroring the experimenter's actions.

Patients with frontal cortex damage have no difficulty looking to a visual cue. However, if they are instructed *not* to look at the cue, but to look in the opposite direction instead, frontal patients (especially those with damage to the frontal eye fields in dorsolateral prefrontal cortex or to the supplementary motor area) are severely impaired at inhibiting the natural tendency to look to the cue.<sup>8</sup> Similarly, when presented with an object, a patient with damage to frontal cortex may automatically reach for it unthinkingly, even if instructed not to reach, and even if it is not an object the patient "wants": "Taking a pack of cigarettes, he hesitated a moment, then opened it and drew out a cigarette. He looked puzzled at it, being a nonsmoker" (p. 246).<sup>9</sup> In cases of severe damage to the supplementary motor area and perhaps premotor cortex, the grasp reflex will become disinhibited, and patients will automatically grasp anything placed in their hand.

The errors made by human infants, infant monkeys, and infant and adult monkeys with lesions of dorsolateral prefrontal cortex on the A $\bar{B}$  and delayed response tasks described by Diamond appear to be another instance where actions "are diverted by the lure of some well-trodden pathway." On these tasks the response of reaching to the first hiding location is strengthened by the reward of retrieving what was hidden. To change that response, and reach to the other hiding location, subjects must not only remember that the object is now hidden at the new location, but they must inhibit the tendency to repeat their earlier, reinforced response. Human infants, infant monkeys, and infant and adult monkeys with lesions

<sup>6</sup>PERRET, E. 1974. The left frontal lobe of man and the suppression of habitual responses in verbal categorical behavior. *Neuropsychologia* 16: 527-537.

<sup>7</sup>LURIA, A. R. 1973. *Higher Cortical Functions in Man*. Basic Books. New York.

<sup>8</sup>GUITTON, D., H. A. BUCHEL & R. M. DOUGLAS. 1985. Frontal lobe lesions in man cause difficulties in suppressing reflexive glances and in generating goal-directed saccades. *Experimental Brain Research* 58: 455-472.

<sup>9</sup>LHERMITTE, F. 1983. 'Utilization behavior' and its relation to lesions of the frontal lobes. *Brain* 106: 237-255. Note that in normal social interaction when someone holds out an object to you, as if offering it, the typical response is to take the object from the person presenting it. Normal adults, however, can inhibit this tendency if we are instructed to do so, or if it doesn't happen to be an object we would like.



of dorsolateral prefrontal cortex often fail to inhibit that tendency; they reach back to the well that used to be correct.

Reaching back to the well that had been correct seems very similar to reaching back to the refrigerator where the food used to be, to writing one's old address instead of one's new address, or to taking one's usual route instead of the new route one had intended. In all of these instances one must pay attention (i.e., exercise executive control) at the choice point so that one's strongest or habitual response is inhibited and another behavior is executed instead.

Typically these errors do not occur when a salient, visible stimulus specifies the correct response.<sup>5</sup> They occur when you have to keep in mind the correct or intended response in the absence of environmental support. Diamond proposes that it is a hallmark of tasks that depend on dorsolateral prefrontal cortex that they typically require both inhibition of a dominant response and "memory" or "sustained attention" (one must keep one's mind focused on what one intends to do despite a brief delay or possible distraction). Schacter points out, however, that tests of temporal order memory, failed by patients with damage to frontal cortex, do not appear to require inhibitory control. Such errors sometimes appear to be similar to other common errors noted by Reason and Mycielska, made by normal adults when we fail to keep our attention focused on what we are doing:

"I was spooning tea into the teapot, and I realized I had no idea of how many teaspoonfuls I had just put in."

"I was cleaning the car windows. I started with the windshield and then went around the car cleaning the other windows. When I got to the front I started cleaning the windshield again quite unnecessarily."

"When I settled down to write, I found I had everything except my pen—which I was convinced I had picked up."

In each of these instances, the person knows he or she has spooned tea, cleaned the windshield, and picked up a pen in the past. The error is in remembering *when* this occurred.

Although memory (of where the reward was hidden, in which refrigerator the food was placed, what one's new address is, or what route one intended to take) is a prerequisite for a correct response in most of these situations, poor memory alone cannot account for the errors made by human infants, infant monkeys, and monkeys or human adults with damage to frontal cortex. For example, monkeys with lesions of the hippocampal formation, who have poor memory, do not show the same kinds of errors. In addition, some of these errors occur even when memory is not required (as when transparent covers are used in the  $A\bar{B}$  task), or when the subject appears to remember the correct response, even though he or she is making the wrong response (as when infants look to the correct well even as they are reaching to the wrong well, or when patients with frontal cortex damage tell you the correct sorting criterion on the Wisconsin Card Sort Test even as they continue to sort the cards incorrectly by the previously correct criterion). Thus, infants and frontal patients sometimes show

<sup>5</sup>For example, if there is a big sign on the old refrigerator that says, "Go to the other fridge," you will usually not make the mistake of opening the old refrigerator.

<sup>6</sup>For other examples see Ellen Langer's work on mindlessness and her 1989 book, *Mindfulness*, (Addison-Wesley).

an apparent dissociation between what they know and what they demonstrate in their behavior; their behavior appears to be captured by more automatic, prepotent response tendencies that are not inhibited as they should be. Avoiding such errors requires keeping your intention firmly in mind, and controlling your behavior so that it expresses what you intend.

Diamond points out that cognitive development appears to be the result, not only of progress in *acquiring* new behaviors and knowledge, but also of progress in *inhibiting* more automatic reactions that interfere with children demonstrating knowledge and understanding they may have long possessed. To some extent, infants appear to know more than their behavior indicates. As their ability to exercise inhibitory control increases, cognitive abilities are revealed that may have been present for some time.

The development and neural basis of inhibitory control has also been investigated in a situation where the goal is always visible and where the response tendency that must be inhibited is not created by reinforcement but is a predisposition built into the organism. Here, in a task called "object retrieval," Diamond places a much desired object inside a transparent box open on only one side. When subjects see the object through a closed side of the box, they must inhibit the natural impulse to try to reach straight for what they want. They must keep in mind the requirement to detour around to the opening in the face of a very strong perceptual pull to try to reach straight through the side they are looking. Indeed, when that perceptual pull is eliminated by the use of an opaque box, performance is greatly improved. Despite the fact that the object retrieval task differs in many ways from the  $A\bar{B}$  and delayed response tasks, human infants improve on all 3 tasks during the same time period (7–12 months of age), infant monkeys improve on all 3 tasks during the same time period (1½–4 months of age), and performance on all 3 tasks is impaired by damage to dorsolateral prefrontal cortex, but not by damage to parietal cortex or the hippocampal formation.

Fox and Bell, like Diamond, report that infants improve on the object retrieval task over roughly the same time period as that for  $A\bar{B}$ , and performance on these two tasks is highly correlated despite the surface dissimilarities between the tasks. Fox and Bell have found 2 patterns of development on these tasks: Some children mature faster than others so that they can withstand delays of 12 sec or more on  $A\bar{B}$  by 12 months of age and they perform at ceiling on object retrieval by 9 or 10 months. Other children mature more slowly: They can withstand delays of only about 2 sec on the  $A\bar{B}$  task as late as 12 months of age, and they do not reach criterion on the object retrieval task until 11 or 12 months.

Fox and Bell go on to discuss the relationship between (a) the developmental progression in human infants' performance on the  $A\bar{B}$  and object retrieval tasks, and (b) developmental changes in the brain electrical activity of the infants as indicated by EEG recordings. They find that the two developmental patterns in  $A\bar{B}$  and object retrieval performance are associated with two distinct developmental patterns in the EEG. In particular, they report that the pattern of EEG activity in the frontal lobe is strongly associated with performance on both tasks.

<sup>7</sup>The EEG recordings were taken while the infants were sitting quietly on their parents' laps before the behavioral testing; each infant was followed longitudinally on both the EEG and behavioral measures.

The EEG parameters on which Fox and Bell focus are “spectral power” over individual recording sites and “coherence” between two different recording sites. By spectral power, Fox and Bell mean the ratio between a higher frequency power band (6–9 Hz) and a lower frequency band (1–4 Hz). They take this to be indicative of the level of neural activity in an area; the greater the power ratio, the more neuronal activity is thought to be generated in that region, although the source of the neuronal activity (its spatial location) cannot be precisely determined from the EEG, especially when a limited number of leads are used. They interpret coherence between two electrode sites as indicating the degree of functional interconnection between those two brain regions.

The infants who improve the most on  $\overline{A\overline{B}}$  and the fastest on object retrieval show an increase in spectral power over the frontal and parietal recording sites, and an increase in coherence between left hemisphere recordings over the frontal and occipital lobes. Infants who are unable to tolerate increasing delays on  $\overline{A\overline{B}}$  and who display only more gradual improvement on object retrieval do not display an increase in frontal activity. They show a change in the spectral power ratio only over parietal cortex (not over frontal cortex) and no increase in frontal-occipital coherence nor in the coherence between any other recording sites. Thus, an increase in frontal activity appears to coincide with increasing ability to tolerate long delays on the  $\overline{A\overline{B}}$  task and to detour around a transparent barrier on the object retrieval task for the infants who show substantial progress on the tasks. No changes in frontal activity were found to be associated with performance on a third, control task.

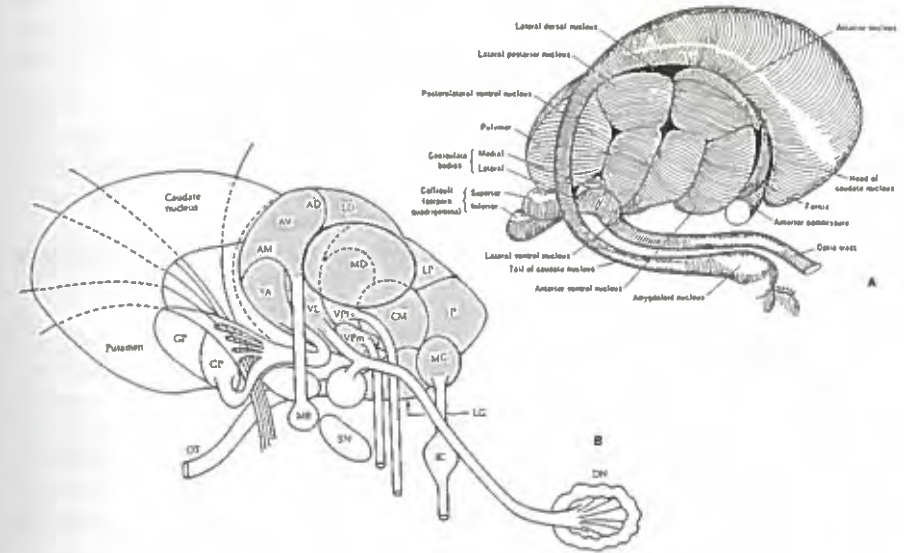
Dorsolateral prefrontal cortex, which appears to subserve the cognitive functions necessary for success on  $\overline{A\overline{B}}$  and object retrieval, does not act in isolation. It acts as part of a circuit of interrelated brain structures. What are the other structures in this neural system? What are the other structures critical for the cognitive abilities subserved by dorsolateral prefrontal cortex?

The other cortical structure most strongly interconnected with dorsolateral prefrontal cortex is inferior parietal cortex (see FIG. 2 above). Damage to inferior parietal cortex in the monkey, however, does not result in deficits on  $\overline{A\overline{B}}$ , delayed response, or object retrieval. Similarly, human adults with damage to parietal cortex do not generally have difficulty with memory or with inhibitory control.

Another area with which dorsolateral prefrontal cortex is strongly interconnected is the **mediodorsal nucleus of the thalamus** (see FIG. 6). Indeed, dorsolateral prefrontal cortex is defined anatomically, in part, through its pattern of connections with the mediodorsal nucleus. **Best, Weldon, and Stokes** report that rats with lesions of the mediodorsal nucleus do, in fact, show a characteristic failure to inhibit their behavior. However, this failure does not appear to be due to an inability to exercise inhibitory control; it appears to be due, instead, to a failure to notice the rearrangement of familiar stimuli.

When an animal has become familiar with a particular environment, any significant change in that environment will cause the animal to orient to those changes and arrest (i.e., inhibit) his or her ongoing activity. If the animal does not arrest or orient, one can infer from this that the altered cues are not an important part of the information the animal is attending to or processing.

Best, Weldon, and Stokes trained rats in 2 similar runways. Then they swapped the goal boxes at the end of each runway. On the trial in which the goal boxes were



**Figure 6. The human thalamus. A: A three-dimensional view of the thalamus from the lateral side in the context of adjacent structures.** (Reprinted from Figure 22-2 in E. L. House & B. Pansky [1967], *A Functional Approach to Neuroanatomy*, NY: McGraw-Hill Book Co., drawn by Ben Pansky.)  
**B: Nuclei and afferent fiber systems of human thalamus in sagittal plane as viewed from the medial side (from the third ventricle).** The mediodorsal nucleus (MD) covers parts of VL, VPI, CM, and LP, the outlines of which are dashed behind MD. (Adapted from Figure 8-3 in J. B. Angevine & C. W. Cotman [1981], *Principles of Neuroanatomy*. NY: Oxford U. Press, drawn by Maureen Killackey.) Abbreviations (in approximate clockwise sequence):

Thalamic nuclei	Afferent fiber systems and other structures
AV, AM, AD = anterior nuclear group	OT = optic tract
MD = mediodorsal nucleus	IC = inferior colliculus
CM = centromedian nucleus	DN = dentate nucleus of the cerebellum
LD = lateral dorsal nucleus	GP = globus pallidus
LP = lateral posterior nucleus	MB = mammillary body
P = pulvinar	SN = substantia nigra
LG = lateral geniculate nucleus	
MG = medial geniculate nucleus	
VPM = ventral posteromedial nucleus	
VPI = ventral posterolateral nucleus	
VL = ventral lateral nucleus	
VA = ventral anterior nucleus	

switched, the control animals took significantly longer to reach the goal because the familiar goal box in a new context caught their attention and caused them to briefly halt their progress toward the goal. Rats with lesions of the mediodorsal nucleus of the thalamus, on the other hand, proceeded to the goal seemingly oblivious to the transposition of the goal boxes. *Best et al.* demonstrate that this failure to show arrest

and normal orienting is not due to a sensory deficit, an inability to notice change in the surroundings, or an inability to inhibit their behavior because rats with the same lesions show a pronounced arrest reaction to the same stimuli when the switched goal box is totally new to them, and not simply in a new context.

Lesions of the superior colliculus caused a different type of deficit: rats with superior collicular lesions oriented to the stimulus change and arrested their behavior when a new tactile stimulus was introduced, but not when the change was in visual stimuli. This makes sense as the superior colliculus (see FIGS. 1 and 6 above) is particularly important for the processing of visual information.

Now, on the object retrieval task, one problem that infants and prefrontally operated monkeys appear to have is in paying too much attention to the *sight* of the object and too little attention to the *tactile* information specifying whether a side of the box is open or closed. **Kuypers** and **Diamond** speculate that inhibition of the superior colliculus by dorsolateral prefrontal cortex may be critical for success on this task. Similarly, the failure of patients with damage to frontal cortex on Guitton's anti-saccade task, which requires that subjects *not* look at the cue, appears to be in their inability to inhibit saccadic eye movements generated by the superior colliculus. Thus, the projection from dorsolateral prefrontal cortex to the superior colliculus (either directly or via the substantia nigra) may be one important component of this circuit.

Another important component may be the communication between dorsolateral prefrontal cortex and the visual areas in the occipital lobe, as the results of **Fox and Bell** suggest. The only source of information a subject has on where the reward was hidden on an AB or delayed response trial is the visual information provided by watching as the hiding occurred.

Finally, monkeys with reduced levels of the neurotransmitter, dopamine, due to injections of MPTP show the same errors on the object retrieval task as do monkeys with lesions of dorsolateral prefrontal cortex. **Diamond** speculates about the importance of the dopaminergic projections from the substantia nigra and the ventral tegmental area to the ability of dorsolateral prefrontal cortex to perform its functions.

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I would particularly like to thank the graduate students who commented on an earlier draft of this introduction: Howard Geyer, Dave Cameron, Sue Coldwell, Tim Davis, Carrie Haskins, Teresa Pantzer, and Randy Seeley. I hope that this book will be useful for graduate seminars and upper-level undergraduate courses, and I have tried in the introduction to help make the book accessible to students and non-experts. The comments of the graduate students at the University of Pennsylvania were very helpful to me in this. I would also like to thank a wonderful colleague and friend, Robert Savoy, who commented on the introduction during many of the stages along the way to its completion.

# Preface

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Two people, more than any others, made possible the scientific meeting on which this book is based. I had first conceived of bringing together developmental psychologists, neuroscientists, and cognitive scientists who were studying the same behavioral paradigms when I was a postdoctoral fellow. A postdoctoral fellow does not call together a meeting of established scientists, however. A new assistant professor does not either, but to make conversation one day during the break in the seminar we were co-teaching, I mentioned my vision of such a meeting to Mike Posner. To my great surprise, Mike said he thought it was a wonderful idea and that he was chairman of the Committee on the Cognitive Neuroscience of Attention and Perception of the James S. McDonnell Foundation, and he thought his committee might be interested in providing some seed money to help make the meeting a reality. All of a sudden, the meeting became a realistic possibility.

With the legitimacy that initial funding from the McDonnell Foundation brought, I could begin trying to raise the rest of the required funds. I called NIMH. The person who answered the phone connected me with someone I had never met up until then, Richard Nakamura. I told Richard about my idea for a meeting. I wasn't sure whether NIMH supported meetings or not, but Richard responded almost the same way Mike Posner had. He thought it was a wonderful idea, and he thought NIMH would be very interested in supporting it.

Michael Posner and Richard Nakamura deserve a great deal of the credit for making the meeting and the resulting book possible. They took a chance on supporting someone who was young and largely unknown. That took more than a little courage, I would think. I am deeply grateful to them for that. Richard Nakamura, in addition, provided welcome advice throughout the planning of the meeting. NIMH has demonstrated its continuing interest by establishing a new program, Behavioral and Neural Approaches to Cognition, for the funding of grants in this area of research.

I would like to thank the McDonnell Foundation, the NIMH (both the Cognitive and Behavioral Neuroscience Research Branch and the Basic Behavioral and Cognitive Sciences Research Branch), the EPA, and the University of Pennsylvania for providing the funds that supported the 5-day meeting of 70 scientists at the Sugarloaf Conference Center on the outskirts of Philadelphia, Pennsylvania, from May 20-24, 1989. I am particularly grateful to the McDonnell Foundation for the initial funding that made all the rest of the funding possible and to the Cognitive and Behavioral Neuroscience Research Branch of NIMH for providing the largest share of the funds.

The meeting was staffed by four graduate students: Jim Cummings and Marie

Perri of the Institute of Neurological Sciences, University of Pennsylvania School of Medicine, Teresa Pantzer of the Department of Psychology, University of Pennsylvania, and Judy Chasin, now in the Department of Psychology, University of Michigan. They did an *outstanding* job and worked exceedingly hard, doing everything from registering people as they arrived, to working the slide projector at every session, taking notes during the discussions following each paper, and, in particular, noting the name of the person making each comment, to cleaning up after everyone left. In addition, Judy Chasin and Teresa Pantzer worked with me throughout the year as we prepared for the meeting. They coordinated travel arrangements for everyone to be picked up from, and delivered to, the airport. They handled the duplication and mailing of the meeting's papers so that all participants received copies before the meeting. They ordered supplies, collected background material on all the participants for the meeting brochure, and handled endless other details. And in everything they did they were thorough, professional, and meticulous.

The staff of the Sugarloaf Conference Center also did an excellent job, as did Trinity Limo, Inc., which handled all travel arrangements, and Jenny DeRose's Audio-Visual Center, which handled the extensive microphone and taping arrangements required so that transcripts of the discussions following each presentation could be typed from the recordings.

Transcribing and editing the discussions following each of the 27 papers was a gargantuan task. It would not have been possible without the exceptionally able assistance of Janet White, who handled all of the typing.

I would particularly like to thank the editorial staff at the New York Academy of Sciences: Bill Boland, Executive Editor, and Linda Mehta, Associate Editor in charge of this book. Linda did a terrific job. She edited the papers exactly as one would want. The book was brought to press quickly and expeditiously. Both Linda Mehta and the printer, Science Press, deserve credit for that.

I would also like to thank John Taggart for taking the photograph of the conference participants and Susan Mills for designing the cover of the paperback version of the book.

Last, but certainly not least, I would like to thank the meeting participants themselves, both the presenters and the discussants. They were a wonderful group of people, and they did an outstanding job. The presenters have written superb papers, and they were responsible enough to get them in months before the meeting. People arrived at the meeting prepared, and every session was packed despite the grueling schedule. Finally, it was a personal pleasure for me to introduce Doug Frost and Helen Neville to one another's work, to introduce Dick Thompson to work on classical eyeblink conditioning in infants, and to bring together others from different scientific disciplines.