10 Binding Complex Memories: The Role of Reactivation and the Hippocampus

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We briefly outline a general cognitive framework called a multiple-entry, modular memory system (MEM). MEM is an attempt to sketch a cognitive architecture that takes into account the wide range of functions memory serves (Johnson, 1983). MEM is one way of summarizing many of the findings and theoretical ideas about attention, perception, and memory that have been generated over the last 100 years of empirical research. As the framework has evolved, we have found it increasingly useful for thinking about many fundamental issues in cognition including dissociations among memory measures (Johnson, 1983), amnesia (Johnson, 1983, 1990, 1991; Johnson & Hirst, 1991, 1993), confabulation (Johnson, 1991), source monitoring (Johnson, Hashtroudi, & Lindsay, 1993), the relation between memory and emotion (Johnson & Multhaup, 1992), and aspects of consciousness (Johnson & Reeder, in press).

This chapter and Johnson (1992) explore in some detail a particular component process of the MEM framework: *reactivation*. We set for ourselves a simple exercise: what might be the consequences for learning and memory of disruption in this one component process of MEM? We argue that reactivation is critical for binding together aspects of complex memories and for maintaining complex memories over time. Furthermore, we consider evidence implicating the hippocampus (and/or related structures) in this central cognitive function.[1] Although not all component processes of MEM are represented in all species (indeed, MEM provides some preliminary hypotheses about the evolution of subsystems; see Johnson & Hirst, 1993), we assume that reactivation is normally operative in rodents as well as humans and other primates. Thus we consider the potential role of reactivation in understanding not only findings from human amnesics but also findings from controlled lesion studies with animals.

We compare and contrast an impairment in reactivation with other explanations of memory deficits (see the articles in the July 1992 issue of *Journal of Cognitive Neuroscience*). We emphasize that reactivation is a component in a process analysis of memory function, and we compare subsystems defined in terms of processes with alternative subsystem accounts of memory. Finally, we consider some general conceptual issues raised by efforts to define

subsystems of memory. Among other things, we suggest that any particular subsystem involves a number of structures and circuits and that an architecture in which subsystems interact reflects a balance between efficiency and flexibility.

1 THE MULTIPLE-ENTRY, MODULAR MEMORY SYSTEM AND REACTIVATION

We begin with an overview of MEM (Johnson, 1990, 1992; Johnson & Hirst, 1993). We then more specifically identify the role of *reactivation* within MEM.

1.1 The Multiple-Entry, Modular Memory System

Memory is a record of the operations of cognitive processes. Different environmental demands and personal goals recruit combinations of these component cognitive processes, which results in such diverse memory phemonena as classical conditioning and autobiographical recall. A useful architecture of memory would specify the nature of the component processes underlying memory and the relations among them. We have proposed one such architecture, a multiple-entry, modular memory system, or MEM (Johnson, 1983, 1990, 1991; Johnson & Hirst, 1991, 1993; Johnson & Multhaup, 1992). Within MEM, component processes are organized into subsystems, which gives memory a modular quality. Here, "modularity" is not used in the same sense of "modularity" proposed by Fodor (1983) and adopted by others (e.g., Moscovitch & Umiltà, 1990; Schacter, 1989); that is, the subsystems in MEM are not "encapsulated." MEM's organization permits subsystems to work independently but allows interactions among them as well.

MEM's subsystems reflect a number of fundamental distinctions among types of cognitive processes. First is the distinction between processes initiated by perceptual stimuli and processes that are more centrally generated. This distinction between what we call "perception" and "reflection" arises repeatedly in theoretical discussions of memory and cognition (Craik, 1986; Goldman-Rakic, 1987; Johnson & Raye, 1981; Lindsay & Norman, 1977; Locke, 1959), although various versions of the distinction do not always divide up cognition in exactly the same way (see Johnson & Hirst, 1993). Second, among perceptual processes, some processes support learning without necessarily producing the phenomenal experience of objects and events, while other processes are more essential for creating such a phenomenal world (Johnson, 1983). For example, the processes that allow you to learn to reach for an object are not the same as those that allow you to identify what the object is (e.g., Jeannerod, 1986). In MEM, the P-1 subsystem is largely responsible for the first type of perceptual processing, and the P-2 subsystem is largely responsible for the second type of perceptual processing. Third, among reflective processes too, there seem to be two fundamental types:

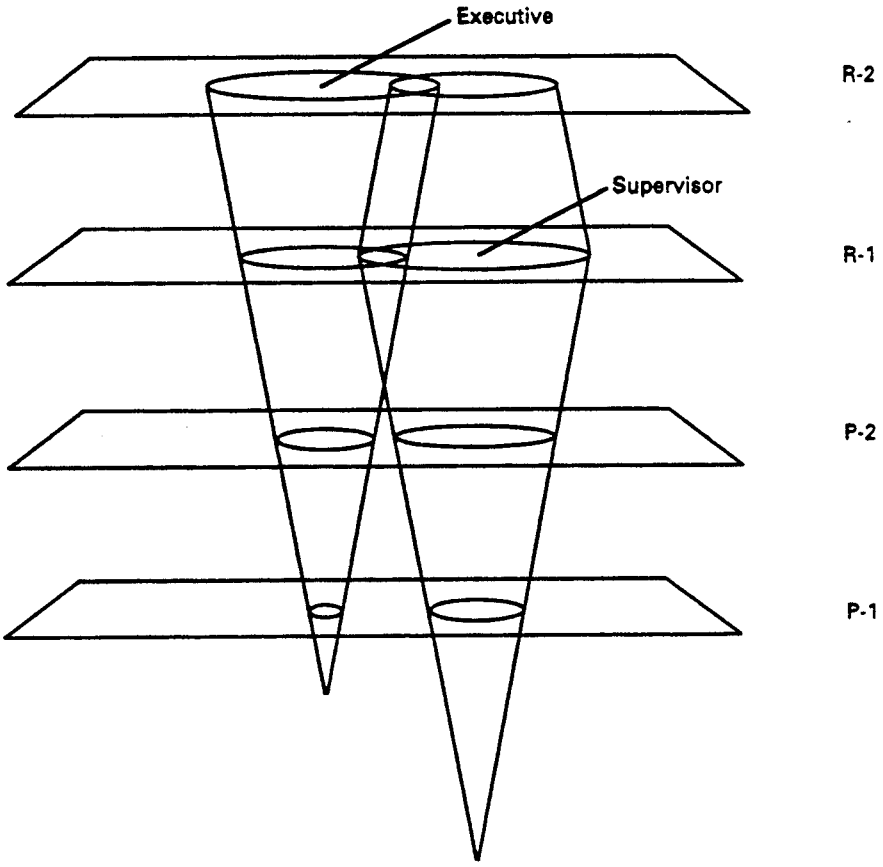

Figure 1 A multiple-entry, modular memory system consisting of two reflective subsystems, R-1 and R-2, and two perceptual subsystems, P-1 and P-2. Reflective and perceptual subsystems can interact through control and monitoring processes (the supervisor and executive processes of R-1 and R-2 respectively), which have relatively greater access to and control over reflective subsystems than perceptual subsystems. (Adapted from Johnson, 1991).

those that can be recruited to express and satisfy relatively simple self-generated goals or agendas and those that communicate or negotiate between multiple goals or agendas (compare Shallice, 1988). In MEM, reflective processes are organized into two subsystems, R-1 and R-2. R-1 and R-2 can each function alone, but they can also function interactively, which gives rise to reflection with a recursive or self-communicative quality.

With these three general distinctions as background, consider figures 1 and 2, which show a schematic representation of the proposed component processes of each of MEM's subsystems and their configuration. The proposed components are derived from and attempt to integrate a variety of theoretical ideas and findings from work on perception, attention, and learning and memory. The minimal requirements for a flexible P-1 subsystem capable of learning are four component processes:

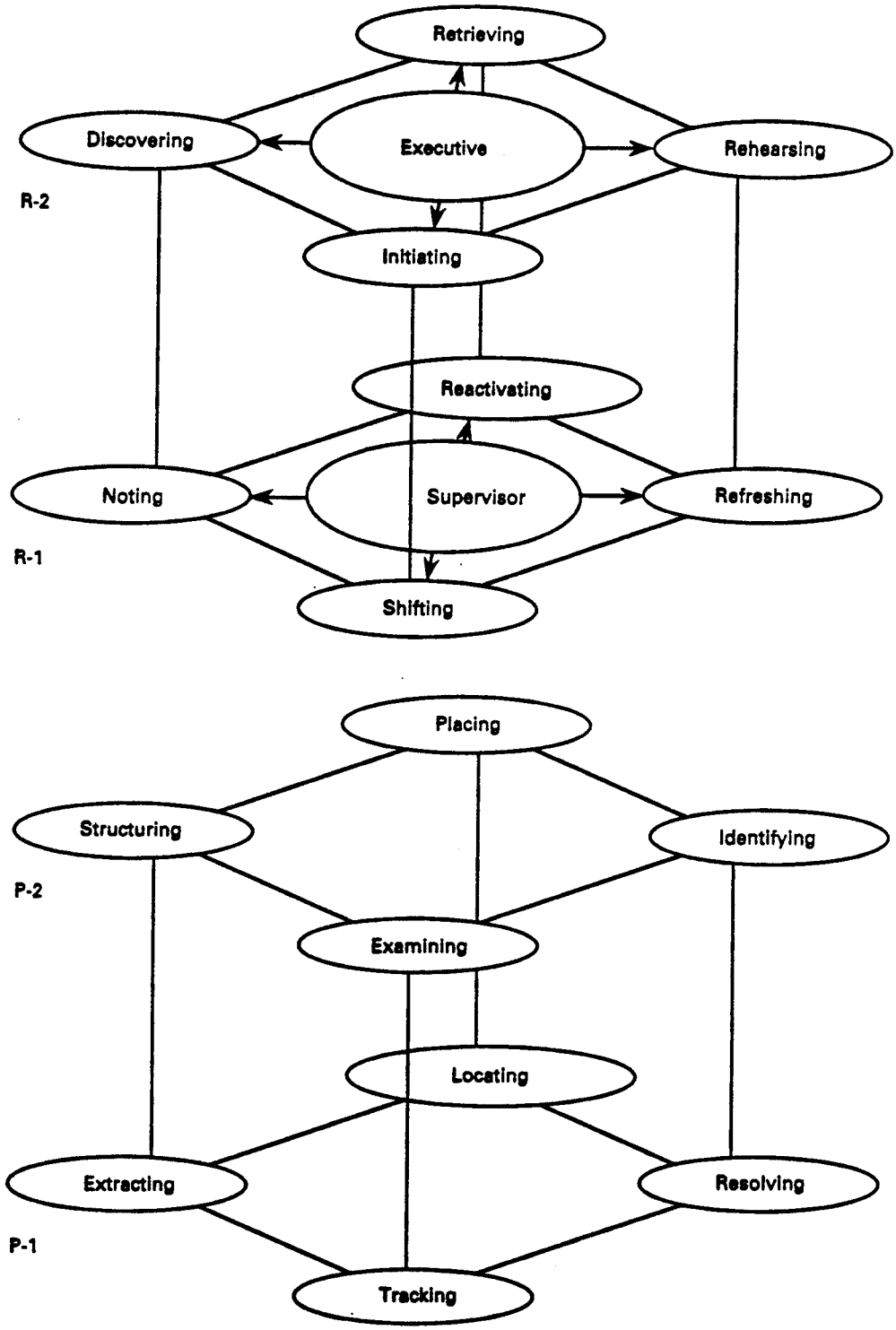

Figure 2 Component processes of R-1 and R-2 (top) and of P-1 and P-2 (bottom). (Adapted from Johnson, 1991).

- Mechanisms for *locating* stimuli, for instance, from abrupt changes in illumination (Posner & Peterson, 1990; Weiskrantz, 1986; Yantis & Johnson, 1990).
- Mechanisms for *resolving* a perceptual array into basic perceptual units by, for example, identifying edges (Marr, 1982), decomposing it into geons (e.g., Biederman, 1987), recovering primitives (Treisman, 1986), or deriving structural descriptions (Riddock & Humphreys, 1987; Schacter, 1992).
- Mechanism for *tracking* stimuli in motion (e.g., Kowler & Martins, 1982; Pylyshyn, 1989).
- Mechanisms for *extracting* invariants from perceptual arrays, e.g., texture gradients, flow patterns, horizon ratios (Gibson, 1950).

These P-1 processes are involved in the acquisition of many types of perceptual-motor skills and often can be tapped with priming procedures.

The minimal computational requirements for a P-2 subsystem capable of learning from phenomenal experience of objects and events are these:

- Mechanisms for *identifying* objects and events, that is, mechanisms by which combined perceptual primitives or integrated geons or computed structural descriptions yield a sense of what something may be or is (e.g., Biederman, 1987).
- Mechanisms for *placing* objects or events in spatial relation to one another, for example, knowing that the dog is closer to the car than to the house. Evidence from a variety of sources indicates that *placing* and *identifying* should be considered the result of different component processes (e.g., Mishkin, Ungerleider, & Macko, 1983). Furthermore, it may be that certain types of agnosias result from a disruption in the interaction between *placing* and *identifying*, producing a jumble of separately identified features (e.g., Luria, 1966).
- Mechanisms for *examining* or perceptually investigating one or many aspects of a stimulus array. That is, perception involves active observers who examine stimulus arrays, which changes the mental pattern that is most active (Hochberg, 1970; Peterson & Hochberg, 1983). Interactions between the component processes of *identifying* and *examining* provide a mechanism for testing perceptual hypotheses, for schema-guided perception (e.g., Hochberg, 1970; Palmer, 1975).
- Mechanisms for *structuring* or abstracting a pattern of organization across a temporally or spatially extended stimulus, e.g., mechanisms for extracting syntax from sentences or melodic structures from notes (Fodor, Bever, & Garrett, 1974; Krumhansl, 1990).

Working in combination, these P-2 component processes give rise to a rich phenomenal world of objects and events, some of which seem novel and some familiar and which together make up the meaningful environment we experience and remember.

In contrast to perceptual processes, reflective processes are important for internally generating and manipulating information. That is, they allow mental

experience to take place without perceptual input. The computational requirements for an R-1 subsystem capable of minimal reflective functions are the following:

- A mechanism for *reactivating* currently inactive information.
- A mechanism for *refreshing* that prolongs ongoing activation.
- A mechanism for *shifting* or changing perspective to activate alternative aspects of stimuli.
- A mechanism for *noting* or identifying relations among activated information.

Working together, these component processes permit such "higher level" functions as identifying and establishing connections among objects or events, even when they are spatially or temporally discontinuous, by creating cognitive contiguity, e.g., the organization involved in free-recall learning (Tulving, 1962).

The R-2 subsystem permits even greater degrees of planning and complexity in reflective operations. R-2 consists of more strategic versions of the processes that make up the R-1 subsystem, namely, mechanisms for (1) *retrieving*, (2) *rehearsing*, (3) *initiating*, and (4) *discovering*. The component processes represented by R-2 can best be understood in relation to the analogous operations of R-1. For example, *noting* can compute overlapping relations from associations activated by two items (e.g., "dog" and "cat" both activate "animal"), whereas *discovering* finds relations that are less direct, for example relations that depend on other relations, as in computing analogies (e.g., Gentner, 1988). Prior related information might be *reactivated* in a relatively nonstrategic manner in response to noting a particular relationship among currently activated information (e.g., Faries & Reiser, 1988); in contrast, prior information would be *retrieved* in a strategic and deliberate manner in response to generating a specific memory cue for yourself in order to remember specific information (Baddeley, 1982; Reiser, 1986). *Refreshing* simply prolongs ongoing activation, whereas *rehearsing* cycles information in a self-generated format (e.g., via verbal or visuospatial representations) in order to remember it or use it in some other way (Baddeley, 1986). *Shifting* produces a change in activation by increasing activation of weakly activated aspects of stimuli, as when one shifts activation from the idea that a fire is hot to the idea that it is bright; in contrast, *initiating* produces a change in perspective by strategically looking for ways to activate unactivated aspects of information, for example, separately listing the items available in a problem-solving task in order to escape the mind-set established by the current activation pattern (Glucksberg & Weisberg, 1966).

Component processes of reflection can be recruited and organized in MEM by reflective *agendas* (e.g., the goal to construct a hypothesis about binding in memory). Agendas include the control and monitoring described, for example, by Miller, Galanter, and Pribram (1960), Nelson and Narens (1990), and Stuss

and Benson (1986). In figure 1, reflective agendas are shown as cones passing through the planes representing the different subsystems. To distinguish R-1 and R-2, we collectively refer to R-1 agendas as *supervisor* processes and R-2 agendas as *executive* processes. The procedures represented by the concept of *agenda* are composed of component processes in MEM as applied to particular data structures. Agendas are mechanisms for two important functions: they are programs or recipes of component processes for accomplishing simple and complex cognitive actions, and they provide a means by which reflective and perceptual processes can interact (as represented by the cones in figure 1). For example, the reflective agenda to build a house can activate the perceptual schema that allows you to find a hammer in a visually cluttered environment. Further, agendas may also serve to temporarily group or bind perceptually and reflectively processed information (Johnson, 1992).

The engagement of any of the component processes of MEM constitutes attention. Moreover, it is easy to see that within MEM attention is not simply a matter of quantitative variations along a single dimension. The attention instantiated in *locating* is different from the attention instantiated in *initiating*, for example, although both may illustrate the concept of attention in a general way. Similarly, MEM makes clear that although cognition varies in amount of effort or control (Hasher & Zacks, 1979; Norman & Shallice, 1986; Posner & Snyder, 1975; Shiffrin & Schneider, 1977), such effort or control probably does not vary along a single dimension. In MEM, the phenomenology of effort or control arises from which component processes are recruited and how many are needed for a given task (e.g., R-2 processes yield a greater sense of control than do P-1 processes). Similarly, in MEM, consciousness is not represented as a separate component of the architecture (as, for example, in Schacter's, 1989, DICE model); rather, different types of consciousness arise from the operation of various component processes (Johnson & Hirst, 1993; Johnson & Reeder, in press). For example, the type of self-consciousness that arises when R-2 monitors and controls the operation of R-1 is different than the type of consciousness that arises when R-1 monitors and controls P-2 (Johnson & Reeder, in press; compare Stuss, 1991).

In short, MEM is a process-based cognitive architecture that distinguishes between the processing of externally derived information and the processing of internally generated information; also included are mechanisms for the control and monitoring of, as well as the interaction between, these types of processing. The four hypothesized subsystems represent cognitive processes available to an intact adult human. Development from infancy to adulthood and breakdowns from overload, stress, and organic brain damage are presumed to reflect the architecture. We hypothesize that the four subsystems have evolved in the order P-1, P-2, R-1, R-2 and that they represent increasingly complex variations on several themes: respectively, identifying and keeping active the objects of perception and thought (*resolving, identifying, refreshing, rehearsing*), changing the activation the system experiences (*tracking, examining, shifting, initiating*), organizing relations across time or events

(*extracting, structuring, noting, discovering*), and situating or going back to earlier objects of perception and thought (*locating, placing, reactivating, retrieving*). A comparative study of learning and memory should thus find commonalities between the proposed subsystems and component processes for different species, although how the specific themes are played out will likely vary somewhat across species.

1.2 Reactivation as a Component Process of MEM

A detailed consideration of all MEM's component processes is beyond the scope of this chapter. Here we will primarily consider the component processes involved in R-1, with special reference to *reactivating*. Our primary goal is to clarify the role that such a component process plays in learning and memory. Because there is no common agreement in the literature about the meanings of terms such as "reactivating," "retrieving," and so forth, it may help to highlight the differences among *reactivating* and other component processes in MEM with which it might be confused.

Activation occurs as a consequence of perceptual or reflective processes. Activation of a particular representation or pattern within a network presumably dissipates quickly if no further processing occurs. *Refreshing, rehearsing, reactivating,* and *retrieving* are qualitatively distinct proposed mechanisms by which activation can be sustained or revived. *Refreshing* is a process by which activation can be prolonged. In effect, refreshing extends the life of an already activated representation or pattern, allowing it to bridge brief gaps between activation patterns. *Rehearsing* includes processes by which self-generated codes for activated information are produced and recycled, usually with the intent to keep information highly available in a reportable format. Typically, rehearsing bridges much longer gaps than does refreshing (as in rehearsing a telephone number) and gives rise to a stronger phenomenal sense of holding something in mind. It is possible for an item to be refreshed (i.e., kept active through its relation to an ongoing agenda) without being intentionally rehearsed.

In contrast to *refreshing* and *rehearsing, reactivating* and *retrieving* are processes that operate on information that is no longer active; they are mechanisms by which information is brought back. *Reactivating* often takes place through a fortuitous combination of agendas and cues. For example, a previous pattern may recur from the concurrent activation of agendas and cues that were previously active together. *Retrieving* requires an additional step: the search for and self-presentation of cues that are not currently active. For example, if you are asked, "What were the exciting papers at the conference last November?" the combination of the agenda to remember and the cues "conference" and "November" might reactivate memories of papers you saw at the conference. If these cues are not sufficient, you might engage in additional processes of *retrieving*. For example, you might try to remember the topic of the conference and think about people who might have been there

in order to cue yourself about what was said. Or you might try listing the people to whom you might have subsequently described the conference in order to cue memory for your report about the papers. In other words, both *reactivating* and *retrieving* require summation of cues to revive activation, but *retrieving* involves a more active self-generation of potential cues that does *reactivating*. A major assumption of the MEM framework is that exploring distinctions such as these, as opposed to grouping a number of processes under a single label such as "retrieval," will be useful for understanding memory development, memory disruption, and other memory phenomena.

Consider this illustration of how a subset of MEM's component processes (those from R-1) might work together in learning to recall a list of randomly selected words: An *agenda* to find a connection between the concepts "passion" and "politics" might *shift* activation around the data structures representing each concept until a relation is *noted* (e.g., both concepts may involve attempts to persuade). In this case, because of the temporally contiguous activation of the agenda, the concepts, and the noted relation, the agenda will now have some capacity to *reactivate* this information in the future. For example, the subsequent presentation of another concept with "persuasion" as part of its activation pattern (e.g., "advertisement") in the presence of this agenda will likely result in the reactivation of the previously noted relation between "passion" and "politics," which will thus strengthen the relation among the associated concepts and lay the groundwork for additional reactivations and a bound, organizational unit involving all three concepts.

Reactivation refers, then, to internally generated repetitions derived from reflective processes. This form of repetition can be contrasted with externally generated, or perceptually derived, repetitions. To help distinguish the two types of repetition, we refer to representations of external stimuli as *reinstatements* (e.g., when an item or relation is presented again by the experimenter). Both reactivation and reinstatement have the capacity to increase the probability that various features of an experience will become bound together and to increase the strength between bound elements. Although some learning and memory can occur without reflective reactivation (e.g., through single presentations or repeated reinstatements), reflective reactivation is crucial for normal memory in a wide range of learning and memory tasks across a wide range of species. Here we illustrate how fundamental this process is by considering the consequences of disrupting it. Our main thesis is that reactivation is crucial for binding information into new relations when essential elements are temporally, spatially, or cognitively discontinuous. For example, if *A* and *B* overlap temporally, they may be bound without reactivation. However, to bind information *A* and subsequently occurring information *C* requires reactivation of *A* in the presence of *C* to provide cognitive contiguity; this cannot be accomplished if reactivation is disrupted. Furthermore, even cotemporal bindings (e.g., between *A* and *B*) miss opportunities for being maintained or strengthened if they cannot be reactivated on subsequent occasions. Reactivation therefore serves a dual role: *to promote opportunities for binding and to*

strengthen existing relations. The more a particular behavioral task or reflective agenda recruits reactivation (in either capacity) under normal circumstances, the worse the memory deficit should be if reactivation is disrupted as a consequence of brain damage or experimental procedures (e.g., distraction).

The following sections propose that the array of deficits seen in hippocampally lesioned animals and in human amnesia patients arises from a reactivation deficit. Reactivation can be disrupted in several ways: the reactivation process itself can be disrupted, a link between reactivation and another process or a particular content domain may be impaired, or the relationship between an agenda and the process of reactivation can be disrupted. The basis for the disruption of reactivation is not crucial for the arguments we make here. In whatever manner reactivation is disrupted, the resulting general pattern should be similar, although the underlying mechanisms may be different. In what follows, we do not assume that reactivating operates exactly the same across species. For example, the conditions and the temporal parameters that distinguish *reactivating* from *refreshing* or *retrieving* may be different for different species. Thus, we are claiming not that the evidence reviewed here provides an unambiguous definition of reactivation but rather that a notion such as reactivation can help clarify relations among diverse memory phenomena. In short, we consider whether the varied reports of memory deficits in hippocampally lesioned animals and amnesic patients can be understood and unified by using reactivation as a central explanatory concept.

2 ANIMAL HIPPOCAMPAL LESIONS AND REACTIVATION

The hippocampus has been implicated in learning and memory across a range of species. We will review several studies demonstrating memory deficits observed in animals with hippocampal lesions and attempt to understand the findings by applying the reactivation framework proposed here. Further, we will describe several studies that, as a group, are difficult to understand within any one model of hippocampal functioning but that can be parsimoniously understood by using the reactivation component of the MEM framework. Several models have been proposed that describe the functional role of the hippocampus in memory. These include models that posit that the hippocampus is important for encoding and recollecting spatial information (Nadel, 1992; O'Keefe & Nadel, 1978), temporally discontinuous information (Moore, 1979; Rawlins, 1985; Solomon, 1980), configural associations (Rudy & Sutherland, 1992; Sutherland & Rudy, 1989), or contextual information (Hirsh, 1974; Mayes, Meudell, & Pickering, 1985); for supporting working memory (Olton, 1986) or declarative memory (Eichenbaum, 1992; Squire, 1987); or for selecting information or decreasing interference (e.g., Moore & Stickney, 1980; Rudy & Sutherland, 1992; Winocur, Rawlins, & Gray, 1987). Despite these diverse characterizations of the role of the hippocampus in memory, we suggest that there is commonality underlying these approaches. Two common themes cutting across various theories have been noted by Eichenbaum

(1992): the fundamental role of relational representations and the ability to use such memories in novel situations. We believe there is yet another useful level of abstraction to be made: reactivation at encoding allows relational associations to be developed among incoming stimuli or with prior information; reactivation at retrieval allows for flexibility by calling up prior information in novel situations.

Latent inhibition observed in normal animals illustrates the role of reactivation in learning. In this paradigm, the experimental group is preexposed to an unpaired conditioned stimulus (CS) several times a day for several days, while the control group receives no preexposure. In the second phase, both groups receive normal pairings of conditioned stimuli and unconditioned stimuli (UCS). Animals preexposed to the CS demonstrate significantly fewer responses to the CS than do animals in the control group, which thus demonstrates latent inhibition (Lubow & Moore, 1959). We hypothesize that reactivation is important to latent inhibition in the following manner: Animals that were preexposed to the CS reactivate this information in the pairing phase, noting that there was no pairing with a UCS during the preexposure period. This inconsistent information about the cue value of the UCS slows down learning of the pairings. If reactivation is in fact crucial to latent inhibition, we would not expect to see latent inhibition in animals with hippocampal lesions. This is in fact the case (Kaye & Pearce, 1987a, 1987b; Solomon & Moore, 1975). Preexposed animals with hippocampal lesions do not demonstrate latent inhibition relative to hippocampally lesioned animals that were not preexposed. This is not simply due to an overall decrease in responding rates; hippocampally lesioned animals that had no preexposure to the CS responded at the same rate as control animals (Solomon & Moore, 1975).

It has been well established that simple classical conditioning is normal in animals with hippocampal lesions (Berger & Orr, 1983; Ross, Orr, Holland, & Berger, 1984; Schmaltz & Theios, 1972; Solomon & Moore, 1975; Solomon, Solomon, Vander-Schaaf, & Perry, 1983), and preliminary evidence suggests this may also be the case in humans with brain lesions that include the hippocampus (Daum, Channon, & Canavan, 1989; Daum, Channon, Polkey, & Gray, 1991; Weiskrantz & Warrington, 1979; Woodruff-Pak & Corkin, 1991). We suggest this is true because reactivation is not required for acquisition of a simple conditioned response. When the CS and UCS are paired, onset of the CS typically occurs during the UCS, and the offsets of the CS and UCS occur simultaneously (the CS and UCS are time-locked), so reactivation is not required for successful pairing. Consider, however, a case where a temporal lag is introduced between the CS and UCS. Reactivation of the CS information would then be required for successful pairing of the CS and UCS. In the trace conditioning paradigm, such a lag is introduced between the CS and UCS. Control animals are not affected by this manipulation, while hippocampally lesioned animals are impaired in acquiring conditioned responses (James, Hardiman, & Yeo, 1987; Moyer, Deyo, & Disterhoft, 1990; Port, Romano, Steinmetz, Mikhail, & Patterson, 1986; Solomon, Vander-Schaaf, Thompson,

& Weisz, 1986). This simple difference between standard conditioning (also termed "delay conditioning") and trace conditioning cannot be understood by employing ideas of hippocampal function that stress spatial information (O'Keefe & Nadel, 1978), configural information (Sutherland & Rudy, 1989), or the ability to compare and contrast information (Eichenbaum, 1992). A view that considers the hippocampus crucial when there are relatively long temporal lags (e.g., Moore, 1979; Rawlins, 1985; Solomon, 1980) could be used to understand both latent inhibition, which requires information from the preexposure phase to be available in a later pairing phase, and trace conditioning, where CS information must be available later when the UCS information becomes available. However, an advantage of the hypothesis of a reactivation deficit is that it can also be invoked in nontemporal paradigms, in which a temporal view would not apply. In such cases where conditions for temporal contiguity are met, however, reactivation is necessary for cognitive contiguity and to promote opportunities for binding information together. We describe such a paradigm next.

Phillips and LeDoux (1992a) investigated standard conditioning of fear to both central cues and environmental contexts in normal animals and in animals with hippocampal or amygdala lesions. We will concentrate on their results from normal controls and hippocampally lesioned animals. In normal animals, fear can be conditioned both to a tone cue and to the context of the experimental chamber. Further, conditioning to the tone cue occurs prior to conditioning to the context, although both the cue and the context are present simultaneously with the aversive UCS (Phillips & LeDoux, 1992a). We suggest that animals are at first more likely to note and associate the relationship between the salient, time-locked CS and the aversive UCS. Subsequently, the UCS information may be *reactivated* and associated with the available context information. If this characterization is correct, we would expect normal fear conditioning to the tone in animals with hippocampal lesions. Contrariwise, if conditioning to the context requires reactivation of the UCS information, we would not expect normal conditioning to the context. This pattern was indeed observed (Phillips & LeDoux, 1992a).

Although contextual notions of hippocampal functioning (e.g., Hirsh, 1974) would also have predicted such an outcome, the contextual hypothesis does not predict that hippocampally lesioned animals could condition to context in the absence of a time-locked CS. This prediction (Johnson, 1992) does fall out of a reactivation characterization, however. If a time-locked CS is not present during a UCS, then context should be able to be directly associated with the UCS because animals have the opportunity to *note* the contiguity between context and UCS without competition from an actively noted relation between CS and UCS. Thus, under these circumstances, conditioning to context can occur without reactivation. Consistent with this prediction, preliminary data (Phillips & LeDoux, 1992b) indicate that both intact and hippocampally lesioned rats show a conditioned freezing response to the environmental context when there is no CS. In fact, the time course of conditioning to the

context under these conditions is equivalent to the time course of conditioning to the CS when there is both a CS and context. The pattern of conditioning to context in hippocampally lesioned animals is normal when reactivation is not required. However, these same animals do not condition to context when UCS information must be reactivated to associate the information. A contextual characterization cannot readily account for the pattern of preserved conditioning to context under certain conditions and disrupted conditioning to context under others. Furthermore, such a characterization cannot readily account for the pattern of preserved classical conditioning and disrupted trace conditioning. Similarly, although a temporal view of hippocampal functioning can account for the standard-delay and trace-conditioning data, applying this view to the fear-conditioning data would not be appropriate, because both the tone cue and the context are present when the UCS is presented in this paradigm. In contrast, the reactivation view can account for all data from the paradigms presented thus far.

Lastly, consider two studies that lead to seemingly opposing conclusions about the encoding of conditional associations and the effect of changing contexts between acquisition and testing phases for hippocampally lesioned animals. In a study by Eichenbaum, Mathews, and Cohen (1989), both sham-operated and fornix-lesioned animals learned an odor discrimination task involving two pairs of two odors ($A+$ $B-$ and $C+$ $D-$). When the correct odors ($A+$ and $C+$) were subsequently paired with different odors ($A+$ $D-$ and $C+$ $B-$ for example), sham-operated controls continued to perform the discrimination at a level equivalent to that for the old pairings, while the fornix-lesioned animals now performed at chance. These data seem to suggest that hippocampal animals can learn only conditional associations and that content information (A and C) cannot be expressed in the absence of its contextual information (A plus B and C plus D).

Before settling on this interpretation, consider the following study as well. Penick and Solomon (1991) trained normal controls and hippocampally lesioned animals in a standard classical conditioning task in one of two contexts (A or B). When contexts were changed after animals reached learning criterion in the first context (i.e., animals trained in A were tested in B and vice versa), conditioned responses decreased for normal controls but not for hippocampally lesioned animals. When the context remained the same, both groups continued to perform at the established criterion rate. From this study we might conclude that hippocampally lesioned animals *cannot* make conditional associations and that content information (the link between CS and UCS) is expressed *independently* of contextual information. This is precisely the opposite conclusion from that suggested by the data of Eichenbaum et al. (1989). None of the current prominent views of hippocampal functioning predict both of these opposing patterns of results. We suggest that a process analysis of these paradigms, including an attempt to understand where reactivation is crucial, indicates that the reactivation explanation can account for both of these outcomes.

In the Eichenbaum et al. (1989) task, animals were given, for example, *A* plus *B* to encode at acquisition and were then presented with *A* plus *D* at test. Presented with *A* plus *D*, normal animals can reactivate the *A* plus *B* information at test and note that the *A* information is consistent between the phases and respond correctly. Presented with *A* plus *D*, hippocampal animals cannot reactivate the *A* plus *B* information, however; they simply do not respond to *A* plus *D*, for which they have never been rewarded previously. Reactivation is necessary at test in this paradigm.

In the Penick and Solomon (1991) task, as in the Phillips and LeDoux (1992a) study, classical conditioning initially occurs to the salient, time-locked CS-UCS pair. UCS information is then reactivated and paired with the context. Hippocampally lesioned animals cannot accomplish this pairing, because it requires reactivation. Thus because the context has been changed between phases, it is now a poor cue for the normal animals, and their conditioned-response rate decreases. However, when the context is changed for the lesioned animals, there is no relevant difference to be noted, because contextual information was never acquired, so there is no concomitant decrease in their conditioned-response rate. Reactivation is necessary at encoding to establish the UCS-context association in this paradigm.

A similar process analysis, with specific reference to reactivation, can be used to understand the performance, on classic memory tasks, of primates with lesions that include the hippocampus (the amygdala is often additionally lesioned in many of the studies reviewed here) relative to normal controls. Here we will focus on performance of the tasks of delayed retention of object discrimination and a delayed nonmatch to the sample.

In delayed retention of object discrimination, an animal chooses one object of a pair that is baited with a food reward. After an intertrial interval of 15 seconds, the same pair is presented again. Each day 20 trials are given until subjects are able to correctly choose the baited object in 9 of 10 consecutive trials. After a 48-hour delay, 20 test trials are administered. If a reactivation deficit is induced by a bilateral hippocampus-amygdala (HA) lesion, then animals lesioned prior to learning should show poor retention relative to intact controls. Zola-Morgan and Squire (1985) found that performance on the object discrimination task after a 48-hour retention interval was significantly poorer in HA-lesioned animals than for control animals. This deficit is presumably due to the inability of the lesioned animals to reactivate the prior object-discrimination information during the testing trials that occur 48 hours after initial training.

Another task in which HA-lesioned animals typically show deficits is the delayed nonmatch-to-sample task. In this task, a single object baited with a food reward is presented to the animal. After a short delay (typically 8 to 10 seconds), two objects—the previously presented object and a novel object—are presented to the animal. The natural response of the animal, to choose the novel object, is rewarded. Twenty trials, all using new object pairs, with an

intertrial interval of 30 seconds, are presented daily until the animal achieves the criterion performance. Mishkin (1978) and Zola-Morgan and Squire (1985) have shown that animals with HA lesions are impaired in acquiring the criterion performance relative to normal animals. After all animals reach criterion performance in the training trials, test trials are administered in which the delay between the object-presentation phase and the pair-presentation phase is increased from 8 seconds to either 15, 30, 60, or 120 seconds. Retention of the object information is increasingly impaired for the HA-lesioned animals as the delay between the trial phases increases (Mishkin, 1978; Zola-Morgan & Squire, 1984; Zola-Morgan & Squire, 1985). Increasingly longer delays, whether they occur between trials or between phases of a single trial, should increase the likelihood that reactivating information will be necessary for successful performance (e.g., with increasing delays the animal is more likely to be distracted by other stimuli). Acquiring the task requires the reactivation of previous trials during new trials to compare and note the behavior needed to receive the reward. Since the intertrial interval is 30 seconds in this task, reactivation is likely necessary to acquire cross-trial information. Thus it is difficult for lesioned animals to acquire the nonmatch-to-sample task. During the test trials, increasing the delay between trial phases increases the need to reactivate information about the previously seen object when the animal must choose the novel object. Again, this is difficult for lesioned animals. Adding a distracting task while increasing the delay simply amplifies this deficit (Zola-Morgan & Squire, 1985). Performance by lesioned primates on a delayed match-to-sample task (Malamut, Saunders, & Mishkin, 1984) is analogous to that on a delayed nonmatch-to-sample task; the main difference between these tasks is that the total number of trials to acquire the delayed match-to-sample task is approximately three times greater for both the control and lesion groups. The same argument for a reactivation deficit in the delayed nonmatch-to-sample task holds for the delayed match-to-sample task.

From the animal literature, it appears that characterizing the performance of hippocampally lesioned animals as reflecting disrupted spatial, temporal, or contextual memory is inadequate. As an alternative, we suggest that a process analysis focused on whether reactivation is involved at encoding, retrieval, or both can provide a more comprehensive account of the behavior and memory deficits in hippocampal animals. We do not suggest that reactivation is the only function of the hippocampus, and we suspect that hippocampal lesions represent only one method of disrupting reactivation processes. Since behavior in humans and animals with diencephalic lesions overlaps significantly with that stemming from hippocampal lesions, diencephalic lesions may represent another method by which reactivation processes are disrupted. In short, in this section we conclude that associative learning or binding can occur with hippocampal lesions if multiple features are simultaneously available (especially with repeated external reinstatements) or when simply *refreshing* information is sufficient to bridge temporal, spatial, or cognitive gaps between important

features or stimuli of a task (i.e., when cognitive contiguity has not been interrupted). However, when reactivation is necessary at encoding, testing, or both phases, a memory deficit will be observed in proportion to the degree that reactivation is necessary for successful completion of the task.

3 HUMAN AMNESIA AND REACTIVATION

In this section we will show how the reactivation-deficit framework can also be used to understand amnesic deficits in human memory tasks. (For the most part, the patients studied have medial temporal or diencephalic damage, but other areas may be damaged as well.) We begin with human memory tasks that overlap with those reviewed in the previous section (conditioning and delayed nonmatch-to-sample tasks) and additionally consider performance on recall and recognition tasks and memory for autobiographical events. As with the animal studies reviewed earlier, we expect that human memory will be disrupted to the degree that reactivation is necessary during the encoding and/or testing phases of a given memory task.

3.1 Tasks from the Animal Literature

The literature addressing classical conditioning in human amnesic patients is currently very limited. Weiskrantz and Warrington (1979) found that two amnesic patients (one Korsakoff, one postencephalic) were able to acquire a conditioned eye-blink response to an airpuff and to retain that response across 10-minute and 24-hour delays. Despite the acquisition of the conditioned response, only one patient was able to verbalize any component of the task episode when questioned. Daum, Channon, and Canavan (1989) also found that three amnesic patients with bilateral temporal-lobe damage demonstrated an initial conditioned eye-blink response and increasing frequency of conditioned responses in a time course typically reported for normal subjects. However, neither of these studies included normal controls. Recently, Woodruff-Pak and Corkin (1991) assessed the abilities of H.M. and a healthy, age-matched control subject to acquire a conditioned eye-blink response. H.M. acquired the eye-blink response after 473 trials, while the control subject acquired the response after 316 trials. Throughout the experiment, H.M. could neither recognize nor recall the conditioning procedure. Woodruff-Pak and Corkin concluded that H.M. was able to successfully acquire the conditioned response, although the rate of acquisition may have been somewhat slowed because of long-term dilatin treatment for seizure control. Similarly, Daum, Channon, Polkey, and Gray (1991) tested the ability of both amnesics and normal controls to acquire a conditioned eye-blink response. They tested four groups: patients with unilateral right temporal lobectomies, left temporal lobectomies, frontal lobectomies (all lesions for intractable epilepsy), and normal controls. There were no differences among these groups in the time course of their first conditioned response. However, significantly fewer subjects

in the temporal lobectomy groups reported being aware of the conditioning procedure than did those in the frontal lobectomy group and the normal control group.

Daum et al. (1991) also investigated the performance of these groups on a conditional discrimination task. In reinforced trials, a colored light (e.g., red) was followed by a tone and then an airpuff; in unreinforced trials, a different colored light (e.g., green) was followed by a tone but no airpuff. Both the normal control group and the frontal-lobectomy group were able to acquire this conditional discrimination. However, the temporal-lobectomy groups showed an equivalent level of conditioned responses to both the reinforced and unreinforced lights; that is, they did not learn the conditional discrimination. We assume that it is helpful in learning such conditional discriminations to be able to reactivate information from previous trials during a given trial in order to compare and note which lights have been reinforced. The inability to reactivate prior trial information makes it difficult for a conditional-discrimination response to develop.

The studies by Daum et al. (1991) and Woodruff-Pak and Corkin (1991) are currently the only studies that demonstrate both intact and impaired conditioned responses in amnesic patients relative to controls. However, Disterhoft (1992), together with his colleagues, are currently investigating performance by both amnesics and controls in the delayed- and trace-conditioning paradigms. We would expect relatively normal performance by amnesic patients both in the delayed-conditioning paradigm and in the trace-conditioning paradigm with a very short trace interval. However, as the trace interval increases or distraction is introduced, which thereby increases the requirements for reactivation of prior information, we would expect the performance of the amnesics to become significantly poorer than that of normal controls.

We can also look at the performance of amnesic patients on memory tasks on which primates with hippocampal lesions are impaired. In the same delayed-retention object-discrimination task described previously for primates, both Korsakoff amnesics and alcoholic controls successfully learned to choose a baited object from a single pair of objects (Squire, Zola-Morgan, & Chen, 1988). Following a 1- or 10-day delay, memory performance on this task was evaluated. Korsakoff patients performed significantly more poorly than controls after both the 1- and 10-day delays. If we assume that reactivation of the previously learned discrimination is necessary at test, we can understand this result as a reactivation deficit on the part of the Korsakoff patients. Korsakoff amnesics have difficulty reactivating information and therefore perform significantly more poorly than the alcoholic controls. The HA-lesioned primates and Korsakoff amnesic patients demonstrated parallel deficits on this task.

We can similarly understand human performance on the delayed nonmatch-to-sample task (Squire et al., 1988) and the delayed match-to-sample task (Oscar-Berman and Bonner, 1985). To use the delayed nonmatch-to-sample study as an example (the delayed match-to-sample study parallels this study

in subject population, methods, and results), Korsakoff amnesics and alcoholic controls were first presented with a penny-baited object. After a 5-second delay, subjects saw a pair of objects, one of which was the previously viewed object; here the novel object was baited with a penny and subjects were to choose the novel object. A new pair was used for each trial, and the intertrial interval was 5 to 10 seconds. Trials continued until the learning criterion was reached. Following this, test trials were administered, where the delay between phases was 5, 15, or 60 seconds. Half of the 15- and 60-second delays were filled with a distractor task. We expect behavior in humans to be similar to that in primates; that is, as the delay between phases increases, we expect that the need to reactivate information about the first-viewed object will increase and amnesics will show a concomitant deficit in performance relative to controls. Adding a distractor task during the delay should serve to magnify the deficit. Squire et al. (1988) found that amnesic patients were significantly poorer than controls after all delays and that performance did not decline further as a function of delay length. Further, the distractor task added during the delay decreased performance to the same degree after both 15- and 60-second delay durations. Neither increasing delay nor increasing delay and introducing a distractor led to a decrement in the control performance. The impairment demonstrated in the Korsakoff amnesic patients is more severe than that predicted by a reactivation deficit alone. We expect that damage in Korsakoff patients, relative to that in unilateral- or bilateral-temporal-lobectomy patients, leads to other deficits in addition to a reactivation deficit (e.g., inability to maintain a set or an agenda). Such processing deficits in addition to a reactivation deficit should further impair performance on memory tasks.

It is clear that humans are impaired on tasks in which other animals also demonstrate memory deficits. Preliminary data for intact classical conditioning in humans parallel the animal data, while difficulties in conditioning paradigms that call for reactivation of information for successful performance also appear to be similar. Further, performance by amnesic patients on tests designed for primates indicates findings analogous to the primate data.

3.2 Recall and Recognition Tasks

We now apply the notion of a reactivation deficit to tasks in which observed impairments have provided the benchmarks for human amnesia: recall and recognition. The most salient symptoms of anterograde amnesia are the dramatic disruptions in recall and recognition of information and events experienced subsequent to the onset of the amnesia. As we discuss in this section, *reactivation* plays a fundamental role in normal recall and recognition both in providing the cognitive contiguity necessary for binding together information and in strengthening such relations. Thus a deficit in this component process of the memory system could produce severe impairments in recall and recognition, such as those found in amnesic patients.

Theoretical ideas about recall repeatedly highlight the importance of relational processing, as for example, in work illustrating the benefits of interactive imagery and mnemonics (e.g., Bugelski, Kidd, & Segmen, 1968; Paivio, 1969; Wollen, Weber, & Lowry, 1972), organization (e.g., Mandler, 1967; Miller, 1956; Tulving, 1962), comprehension (e.g., Bransford & Johnson, 1972; Dooling & Lachman, 1971), and schemas, scripts, and story grammars (e.g., Rabinowitz & Mandler, 1983; Schank & Abelson, 1977; Thorndyke, 1977). Most theorists agree that one reason relational processing is powerful is because at acquisition it sets up the structures among cues that are necessary for later recall (e.g., Tulving & Pearlstone, 1966; Seamon & Chumbley, 1977). Reactivation is essential for developing new cue structures among unrelated elements and strengthening relational bindings important for recall.

To take the hypothetical example given earlier of the subject engaged in free recall, the activation of "passion" in the context of the item "politics" allowed the subject to bind these items together via the noted relation of "persuasion." The later activation of the concept of "persuasion" by the item "advertisement" (in the context of the agenda to continue organizing the list) reactivated "passion" and "politics." This reactivation had two consequences: it allowed the binding of a new item to items that occurred some time previously, and it strengthened the earlier binding of "passion" and "politics" to the same organizational unit. Thus some ideas, such as "persuasion," come to function as recall cues for other ideas, such as "passion," "politics," and "advertisement."

In contrast to recall, where the subject must come up with the target items on the test, in a typical recognition experiment the targets are presented along with distractor items, and the subject's task is to identify (or recognize) which came from the target list. Again, having engaged in elaborative processing that sets up relational bindings among items on the list or between list items and extralist cues (e.g., schemas, semantic knowledge) helps recognition, although the effects here are often less dramatic than typically found for recall. Theories differ in how they conceptualize the mechanisms underlying the effects of elaborative or relational activity on recognition. One proposal is that when a subject sees the test item, they may engage in a "retrieval check" (Atkinson & Juola, 1972; Mandler & Boeck, 1974). According to this idea, the subject may attempt to recall a list via recall mechanisms, such as those described above that capitalize on relational information; finding an item via a recall process would be sufficient evidence for "recognizing" that the item was from the target list. To use the same example as above, for the test item "politics," the subject would use the activated cue "persuasion" to recall items from the persuasion category. If "politics" appeared in this set of recall items, it would be recognized. Another idea is that relational processing at acquisition increases the chances that the activation prompted by the item at test will include source-specifying information (e.g., color, spatial location, etc.), which is taken as evidence for recognition (Johnson, Hashtroudi, & Lindsay, 1993; Raye, 1976). For example, noting where on a screen an item occurred will help bind item and location. Later activation of this location information when the

item is presented could, because of its specificity, be used as evidence that the item occurred on the acquisition list. In this way, elaboration might help subjects "consciously recollect" the specific episode of the presentation of the item (Jacoby, 1991). Another possibility is that the rapidity of activation of elaborative information could alone be taken as a cue that the item has been seen and acted on before (Raye, 1976). Whatever the underlying mechanisms by which elaborative or relational processing aids recognition, it is readily apparent that reactivation plays a crucial role, because it is instrumental for creating occasions for noting relations, for strengthening relational binding through subsequent reactivations, and for making useful information available at test. Disrupted recognition might therefore result from relational processing deficits as a consequence of disrupted reactivation.

Although the potential impact of relational processing on recognition is incorporated into most recognition models, most models emphasize the role of nonrelational, item-specific familiarity responses in old/new recognition. Again, theories vary in what they assume to underlie this familiarity response. For example, Underwood (1972) suggested that the mnemonic representation of an item builds up frequency increments whenever the item occurs; this frequency information yields the familiarity response that produces recognition (see also Atkinson & Juola, 1974). Underwood explicitly noted that not only external presentations but also covert occurrences of items increase their familiarity value. (In terms of MEM, covert occurrences are a result of *refreshing, rehearsing, reactivating,* and *retrieving.*) In the language of several recent models for recognition, one could say that such covert occurrences add episodes to memory that figure into "echo intensity" (Hintzman, 1988), add to the "global familiarity" computed from degree of total activation (Gillund & Shiffrin, 1984), increase intraitem integration (Mandler, 1980), or add to processing "fluency" (Jacoby, 1991).[2] In any event, anything that would decrease the frequency of reflectively generated reactivations should also produce a disruption in recognition when such reactivations play a central role in the normal case of recognition. It is clear, then, that a reactivation deficit could impair recognition memory, both by disrupting the accrual of information that subserves familiarity (here including frequency increments, echo intensity, global familiarity, item integration, and fluency) and by eliminating the binding opportunities that subserve the ability to recollect specific featural and relational information, which is also important for recognition.

Thus there are a number of variations on the general theme that normal recognition can be based either on a response to undifferentiated information (e.g., "familiarity" with no sense of an item's specific features) (Johnson et al., 1993) or on more specific characteristics of complex memories, such as perceptual detail, spatial location, semantic relations among concepts, and so forth (i.e., information that could be used to infer sources). This raises the question of whether subjects with memory disorders are equally impaired in using familiarity and in using the other bases of recognition. Several investigators have suggested that old/new recognition judgments based on familiarity

alone may be preserved in amnesia, whereas judgments about source are impaired (Cermak & Verfaellie, 1992; Verfaellie & Treadwell, 1993; Weinstein, 1987). For example, Weinstein found quite similar levels of recognition for amnesics and controls when the original processing at acquisition had been directed toward perceptual characteristics of the items (counting the number of black objects in a sequence of colored objects, such as an orange rabbit, a black umbrella, and a pink train) and the distractors on the test were new items in unfamiliar colors (a green goat). In contrast, when the distractors were constructed to elicit familiarity responses based on semantic knowledge (green pepper), the amnesics showed a significant recognition deficit relative to controls. Weinstein suggested that the amnesics could use differential familiarity of targets and distractors as a basis for recognition decisions in the first condition but had difficulty in the second condition sorting out the source of their familiarity responses. Consistent with this interpretation, the fact that amnesics have difficulty separating items that are familiar from day 1 from those familiar from day 2 (Huppert and Piercy, 1976) suggests that they experience familiarity responses to previously encountered stimuli but cannot identify the source of the familiarity. Verfaellie and Treadwell make a similar point in interpreting their study (1993). Prior to the test phase, subjects saw some items as anagrams, read others, and heard still others. Subjects were then tested under two conditions: say "yes" to all old items (whether heard, seen as an anagram, or read) and "no" to new items; or say "yes" only to items that had been heard and "no" to new items, anagram items, and read items. Whereas control subjects decreased "yes" responses to anagrams and read items in the second condition relative to the first, amnesics were equally likely to say "yes" to anagrams and read items in the two conditions. Again, the results suggest that amnesics experience familiarity responses but have difficulty identifying the source of the familiarity. Taken together, these various studies suggest that the processes that newly bind specific, relational, or featural information are disrupted in amnesia. If so, a disruption in reactivation should particularly disadvantage relational information, which is in greatest need of strengthening.

Although reactivation affects both recall and recognition, an important difference between them is that recall characteristically depends more on reactivation than does recognition. For preexperimentally unrelated items, such as words or pictures, normal recognition is often excellent after a single presentation, whereas recall is generally quite poor (Shepard, 1967). That is, a single presentation appears to set up a representation that is easily activated by another presentation of the same stimulus (i.e., by *reinstatement*) but is surprisingly difficult to access through reflective processes (i.e., by *reactivation*). At the same time, recognition generally improves with additional presentations that do *not* allow for reactivation and reflective processing of the material, whereas recall may not (Tulving, 1966). This is because familiarity, supported to some degree by *reinstatement*, is a good basis for recognition (given that the distractors are not too similar to the targets or do not have any other

basis for familiarity) but is not particularly good for recall. Reflectively generated cue structures, which depend on *reactivation*, are more likely to be needed in recall.

If amnesics suffer from a reactivation deficit and if recall typically depends more on reactivation than does recognition, we would expect to see variations in amnesic performance on recognition and recall tests that can be traced to variations in reactivation requirements. Although we need better tests of this proposition, several findings in the literature are consistent with it. Complex or abstract pictures, which are difficult to label verbally, are likely to receive relatively more perceptual than reflective processing; in particular, they may undergo few reflective reactivations (e.g., Loftus, 1974). If reactivation does not play much of a role in how control subjects process such pictures, amnesics should do relatively well. A number of studies of complex picture recognition (summarized in Johnson & Kim, 1985) indicate that amnesics seem to do quite well. For example, in a study conducted by Johnson and Kim (1985), subjects saw each of several abstract "paintings" (each composed of three of five possible colors) either 1, 5, or 10 times. On a forced-choice recognition test, amnesics' performance improved as a function of frequency of presentation, as did normal subjects' performance. Furthermore, on an additional recognition test administered 20 days later, the amnesics' recognition rate for items seen 10 times was 78 percent, versus 86 percent for controls. These findings suggest that amnesics, like controls, build up familiarity over repeated presentations, which will support recognition as a function of perceptual reinstatements after substantial intervals (see also Huppert & Piercy, 1982).

One can capitalize on this intact aspect of amnesic processing to equate the recognition performance of amnesics and controls by giving amnesics more presentations of material than controls. Alternatively, one can attempt to limit the self-initiated reactivations and elaborations in normal subjects by presenting material more quickly to normal subjects than to amnesics (see Huppert & Piercy, 1977). By combining both techniques, Hirst, Johnson, Phelps, and Volpe (1988) actually found superior recognition for unrelated words in amnesics than controls: the recognition rate for amnesics, who saw a list twice at a rate of 5,000 milliseconds per item was 85 percent, versus 77 percent for controls, who saw the list once at 500 milliseconds per item. The subjects in the study by Hirst et al. were tested for recall of the list items as well. Amnesics recalled about 7 percent of the words, whereas controls recalled about 16 percent (see also Hirst, Johnson, Kim, Phelps, Risse, & Volpe, 1986). The fact that recall was relatively more disrupted than recognition is consistent with our argument that recall depends more on reactivation and that reactivation is disrupted in amnesia. However, this general line of reasoning does not depend on amnesics invariably showing disproportionate disruption in recall as compared with recognition. Because both recall and recognition recruit various processes depending on the situation, it may be that under certain conditions recall and recognition tasks may *not* differentially draw on

reactivation processes (see Johnson et al., 1993; Verfaellie & Treadwell, 1993). Under such circumstances, amnesics should show equal deficits in recall and recognition (e.g., Haist, Shimamura, & Squire, 1992).

Although profoundly memory-impaired subjects can be brought up to relatively high performance levels on recognition by adding presentation trials, it is almost impossible to bring them up to such levels of recall using the same strategy. Presumably, the extreme difficulty amnesics have in recall tasks arises because additional reinstatements alone are insufficient to establish and strengthen appropriate cue structures and their relations, which are normally established by reflectively guided reactivations. Consequently, the various elements of experience do not become bound into complex, coherent, voluntarily accessible representations (see Hirst, 1989).

In suggesting that amnesics have a disruption in reactivation processes, are we simply postulating a "retrieval" deficit theory of amnesia? The usual objection to the idea that anterograde amnesia involves a retrieval deficit is that anterograde amnesics do not necessarily have difficulty retrieving everything: they may be able to retrieve semantic knowledge and autobiographical information encoded prior to the onset of amnesia (e.g., Hirst, 1982; Shimamura, 1989). However, this critique does not take into account two considerations. First, the state of learned information is not necessarily the same for recently acquired information and information that has survived the test of time and interference. That is, the preonset events represented in memory are probably not exactly like the postonset events that are not remembered. As discussed above, preonset events (and knowledge) have likely benefited from prior reactivations and other organizational processes that improve their chances of being revived relative to newly acquired events and information. Second, there is more than one type of retrieval from memory. Retrieval can be prompted or controlled by external cues, such as the retrieval involved in priming, the retrieval involved in familiarity-based recognition, or the retrieval involved in highly overlearned information, as when someone asks your name and you reply. Even these kinds of retrieval, all prompted by external cueing, are not necessarily alike. More important, these kinds of retrieval all differ from the retrieval that is not controlled by external cues but is prompted by reflective processes. Retrieval can be prompted or controlled by internal cues, as when subjects attempt to recall items without benefit of cues in a free-recall task. Here internal cues are set by agendas and strategic plans or are self-initiated cues, as opposed to external cues, which are set by information presented in reinstatements. In MEM, we begin to differentiate various kinds of reflective retrieval by distinguishing among *refreshing*, *reactivating*, *rehearsing*, and *retrieving* (see Johnson, 1990, 1992; Johnson & Hirst, 1993) and further postulate *reactivating* as what is specifically disrupted in amnesia.[3] By postulating a reactivation deficit, we are also postulating that deficits will be observed not only at test but also in learning and maintaining information. If reactivation cannot be recruited to provide the cognitive contiguity necessary for binding aspects of memory together, then complex cue

structures will not be properly encoded. If reactivation cannot be recruited to provide the strengthening of reinstatement-generated relations, then relations will not be properly maintained. Thus postulating a deficit in the MEM component process of reactivation is an encoding, maintenance, *and* retrieval account of amnesia.

3.3 Recollection of Autobiographical Events

For the most part, cognitive theories of recall and recognition have been based on empirical studies of normal subjects learning and remembering relatively simple lists or stories. However, studies of memory for naturally occurring autobiographical events, or relatively complex laboratory simulations of autobiographical events, also indicate the importance of reactivation in personal memory. For example, Rubin and Kozin (1984) found that people's more vivid memories were also those they reported having talked about more often. In an extended study of her own memory, Linton (1978) recorded daily events in her life (e.g., "I had coffee with Jeff before his colloquium presentation, and we talked about his research") over a period of years. She regularly tested her memory for such events, and one notable finding was that reactivating memories by cueing them on test trials markedly improved their retention on subsequent tests (see also Allen, Mahler, & Estes, 1969; Hogan & Kintsch, 1971; Landauer & Bjork, 1978). Furthermore, previous reactivations had greater effects on earlier memories; that is, the effect of reactivations was not necessarily seen until much later. In studies conducted in our lab, Aurora Suengas (Suengas & Johnson, 1988) had subjects engage in various "mini-events," such as visiting a computer lab, making a clay pot, and having coffee and cookies. Using cues to control which memories were reactivated, she subsequently had subjects think about some events but not others. Subjects later rated the subjective qualities of their memories, such as the perceptual and spatial detail or the emotional characteristics, for the various events. Their ratings indicated that thinking about events maintained subjective qualities over time more than not thinking about events. Characteristics such as perceptual detail and the spatial relations among objects and people give memory its episodic quality; such details are what distinguish autobiographical memories from more abstract "semantic" memory (Johnson, 1988). Mental rehearsal or reactivation of unique, one-time autobiographical events may be the single most important determinant of which personal memories survive to become part of our autobiographical narrative. An amnesic who cannot think back to earlier events after even only a few minutes cannot make use of reactivation in maintaining the qualitative details necessary for event memory (Johnson, 1988).

As we have noted (Johnson, 1992), there are undoubtedly large individual differences in the amount of reactivation of event memories that people normally engage in or the types of memories to which reactivations are

directed. Similarly, there are likely circumstances that generally tend to lower (or raise) the frequency of such reactivations. For example, chronic stress, lung disease, abuse of alcohol or other drugs, and other conditions that influence cognitive functioning might reduce the frequency of naturally occurring reactivations, either because of a direct reduction of cognitive functioning or because of a reduction in the social interactions likely to prompt autobiographical remindings. If so, such individuals should have fewer well-consolidated autobiographical event memories and should therefore be at greater risk for retrograde amnesia in the event of brain damage. Thus some of the temporal gradient that retrograde amnesia often shows, with greater deficits for preonset memories closer in time to the onset of amnesia than for memories from longer ago, may actually be produced by a "progressive anterograde amnesia," which, with time, increasingly disrupts reactivation of events and information, as often observed in alcoholic Korsakof patients (Butters & Albert, 1982).

Reactivation is crucial in many tasks for encoding and developing cue structures, for maintaining relations, and for retrieving information. This component process is important for promoting cognitive contiguity and for spanning temporal, spatial, or cognitive lags where other processes are insufficient. We have argued that reactivation is important not only for tasks typically considered "declarative" or "explicit" memory tests (i.e., verbally mediated recall and recognition) but also for tasks, such as classical conditioning, that are not considered declarative or explicit. We also expect deficits in procedural, or skill-based, tasks when there is a reactivation component to the task (Phelps, 1989). Further, if a priming paradigm requires reactivation, then performance on that task should also be impaired. The point here is that the categorizing complex tasks into such dichotomies as procedural/declarative or implicit/explicit does not clearly delineate which tasks will be impaired in amnesia and which will not. Instead, a process analysis of individual tasks is required to predict and understand where memory impairments will be observed. This point begins to highlight the differences in memory deficits as conceptualized within the MEM framework and as conceptualized in other subsystem memory models. We will further explore these differences in the next two sections.

4 MEMORY SUBSYSTEMS IN THE MULTIPLE-ENTRY, MODULAR MEMORY SYSTEM

We have described how reactivation might play a role in a variety of tasks, contributing to, for example, latent inhibition in classical conditioning, performance on delayed nonmatch-to-sample tasks, familiarity in recognition, organization in recall, and maintenance of the qualitative details characteristic of memory for autobiographical events. Disruption in this one component process of mental activity has far-ranging consequences for memory. How does

this view of memory deficits as resulting from a disrupted component process compare with views positing a disrupted memory subsystem, such as a disrupted declarative, spatial, or episodic subsystem (e.g., Cohen & Squire, 1980; Nadel, 1992; Schacter, 1992; Squire, 1992; Tulving, 1983)?

Most subsystem models of memory rest on dichotomous distinctions based on broadly defined task content or task categories, such as episodic/semantic (Tulving, 1972, 1983), procedural/declarative (Cohen, 1984; Cohen & Squire, 1980), taxon/locale (O'Keefe & Nadel, 1978), and habit/memory (Hirsh, 1974; Mishkin, Malamut, & Bachevalier, 1984). Johnson's (1983, 1990) initial description of MEM (which posited three subsystems: two perceptual and one reflective) also adopted a relatively broad dichotomy in terms of perceptual and reflective processes, based on Johnson and Raye's earlier distinction between perceptually derived and internally generated information (Johnson & Raye, 1981; Johnson, Taylor, & Raye, 1977). However, MEM differed from many dichotomies in that the basis of the distinctions among the three proposed subsystems was along types of *processing requirements* of tasks rather than classifications between tasks. The MEM framework has evolved to encompass four interacting subsystems, each comprising a specific set of component processes (Johnson, 1992; Johnson & Hirst, 1993).

To understand how subsystems are defined in MEM, consider what is meant by "system" and what the goals are in making distinctions between systems. The homeostatic system of the human body is an example of what can be meant by "subsystems." The system of the human body is made up of such interacting subsystems as the respiratory, circulatory, and central nervous systems. Each subsystem is further made up of component processes; for example, the respiratory (sub)system includes processes for taking in air, cleaning air, and exchanging carbon dioxide for oxygen. Here, a system is a functional unit with interacting interdependent subunits and processes that markedly expand the functional capability of the system as a whole. A subsystem is simply a secondary or subordinate system, which means that it too consists of interacting, interdependent subunits. This same use of "system" can be seen in the MEM framework in the four interacting subsystems (P-1, P-2, R-1, and R-2) making up the memory system and in the specific sets of component processes making up each of the memory subsystems.

The conceptual use of subsystems seen in MEM, which allows for (or even requires) interactions among subsystems, might be characterized as the "weak" view of systems described by Sherry and Schacter (1987). However, distinguishing between the "strong" view, in which components within subsystems can interact but subsystems themselves do not, and the "weak" view seems more appropriate for subsystem memory models that identify subsystems with distinctions among task *content* than for those that define subsystems in terms of *processing* components. In our view, many tasks performed by adult humans recruit processes from most, if not all, subsystems for successful completion. Some tasks, such as perceptual identification or word recognition, will recruit more perceptual processes from P-1 and P-2, but these processes

in turn are likely to activate reflective processes to set up cue structures or strengthen relations between current information and prior related instances. Other tasks, such as recall or free association, access relatively more reflective processing from R-1 and R-2, but these processes also activate some perceptual processes when, for example, examining the environment for potential recall cues. The coherence of processes within subsystems increases efficiency, but the interaction of processes across subsystems promotes flexibility and increases the complexity of the tasks that can be accomplished. Further, a system based on task-processing requirements avoids the potential danger of proliferating subsystems presumed to operate by entirely different principles, as experienced by content-based subsystem memory models (Johnson and Hirst, 1993). As Johnson and Hirst (1993) note, the MEM framework does not require unique subsystems with different operating principles for face, language, or spatial memory, for example. That is, the processing subsystems posited in MEM should be replicated across content domains.

In the psychologically defined functional subsystems of MEM, the components are seen as working together for purposes that can be defined in mental and behavioral terms (e.g., to reason, to find food). Behaviors vary greatly in the cognitive complexity they represent, and we have no way to estimate the relative roles of evolutionary pressures and accidental factors in producing variations in complexity of cognition and memory across species. But in any event, we assume that the architecture of human cognition and memory reflects something of our evolutionary history. In figure 1, eliminating subsystems from the top to the bottom would in each case leave intact a viable organism with considerable learning capacity. For example, if we build up from the bottom of figure 1, a fully functioning organism with only a P-1 subsystem should be able to engage in a variety of learning, for example, by connecting incoming stimuli to motor responses. An organism with an additional P-2 subsystem should be able to attach responses differentially to recognized individuals, objects, and locations. Adding in an R-1 subsystem would permit comparing and connecting events across time and the expression of intention and control. Adding in an R-2 subsystem would allow the discovery of relations among many internally generated representations. To give more concrete examples, P-1 processes might be sufficient for acquiring skill at chasing prey, but P-2 processes are needed to recognize a familiar environment. R-1 processes are important for considering whether the current environment is preferable to yesterday's environment, but R-2 processes are needed to contrast one's own idea about the relative desirability of two environments with someone else's idea.

These examples are only to give some flavor of the types of activities each subsystem adds to the cognitive repertoire. The descriptions should not be taken as subsystem labels. For example, we do not want to say that the P-1 subsystem is *for* associating stimuli with motor movements, because associating certain motor movements with stimuli may be greatly helped by activities of subsystems higher than P-1 (e.g., when one learns a new dance, it may be

useful to have a reflectively based representation of the overall structure and theme of the piece). Conversely, P-1 may participate in memory feats that are not entirely motor-based, as when starting to dial a phone number helps you remember the names of the digits.

A second crucial point is that functionally important cognition within subsystems typically requires the joint action of several component processes. For example, getting more skilled at chasing prey likely involves *locating*, *resolving*, *extracting*, and *tracking*; learning to critically evaluate empirical evidence likely involves *rehearsing*, *initiating*, *retrieving*, and *discovering*. Thus the overall architecture of MEM represents two features of cognition that are evident across species: the capacity of certain cognitive components to work easily together to support functionally important behaviors and also the expansion of functionally important behaviors, resulting from the addition of new, smoothly interacting sets of components. The architecture also reflects such ideas as that interactions are more easily accomplished among component processes from adjacent than nonadjacent subsystems, that recursive reflection (e.g., thinking about thinking) can be accomplished with only two interacting subsystems as long as each has the capacity for control and monitoring (thus R-1 and R-2 resolve the "homunculus" problem), and that component processes going up the vertical edges of the cubes in figure 2 may represent variations on computational themes or provide some clue about the evolutionary history or structural basis of successively evolving components (Johnson & Hirst, 1993).

To understand the cognitive requirements of tasks, we have suggested a process analysis of tasks rather than categorizing tasks according to their content. We believe that comparing radically different tasks, such as perceptual identification and recall, can take us only so far in analyzing component processes of cognition. To tease apart the action of component processes, we have to pay more attention to the detailed requirements of superficially similar tasks. Different theorists have been able to use largely the same data to support different theoretical frameworks because processing requirements tend to be unequally distributed across tasks (e.g., perceptual and skill tasks draw largely on P-1 and P-2 processes, and recall on R-1 and R-2 processes), though the correlation between type of task and type of processing requirements is not perfect. In addition to advocating a processing analysis, we have also posited that a deficit in a single component process within a subsystem can account for a wide range of memory impairments. This notion is more specific than the suggestion that impairments in memory are a result of deficits in particular memory subsystems (e.g., Cohen & Squire, 1980; Nadel, 1992; Schacter, 1992; Squire, 1992; Tulving, 1983). What has been the basis for taking subsystems as the unit of disruption? The main basis for making these distinctions has come from single and double dissociations of task performance in both amnesic and normal subjects (e.g., Schacter, 1989). However, single and double dissociations can also be taken as evidence for disrupted processes, especially when dissociations occur across proposed subsystems

(e.g., Blaxton, 1992, although see Dunn & Kirsner, 1988, for problems in using dissociations as evidence for multiple subsystems or processes). In MEM, we do not think that each of the four postulated subsystems breaks down in an all-or-none fashion. Rather, each subsystem could break down in several ways, just as the respiratory (sub)system could break down in multiple ways from problems with the diaphragm or the alveoli. Here we have focused on a disruption in *reactivating*, a single component within a subsystem, with consequences for many memory tasks.

Although we have focused on reactivation, most memory tasks require the participation of a number of component processes, and the more complex the task, the more complex combinations of component processes are necessary. Consequently, especially for more complex tasks, there should be a number of ways to disrupt their performance. For example, organization often involves not only *reactivating* information but also *shifting* from one aspect of meaning to another for the same item, *noting* relations, or *refreshing* information. While hippocampal damage may primarily affect reactivating, we expect that damage to other areas, such as the frontal lobes, primarily affects some of these other component processes (e.g., shifting, noting). All of MEM's component processes are necessary for normal adult human memory, so we should see different patterns of memory disruption, depending on the locus of the brain damage and the information tested. Thus we expect that even if reactivating were intact, brain damage that affected some of these other processes should show up when processing demands for normal performance are high. Consistent with this, patients with frontal lobe lesions do relatively well on tests that require relatively few organizational processes, such as *shifting* and *noting*, but show disruption on tasks requiring relatively more of these processes (Moscovitch, 1989; Shimamura, Janowsky, & Squire, 1991; Smith & Milner, 1984).

As this example suggests, MEM subsystems are not confined to single structures in the brain (e.g., R-1 is not *in* the hippocampus). Rather, subsystems involve brain circuits that cross neuroanatomically defined regions. Similarly, component processes within subsystems may also involve circuits that cross neuroanatomically defined regions (e.g., a neocortex to hippocampus to neocortex circuit). Thus, although we have proposed that the hippocampus is central for *reactivating*, it is important to remember that to reactivate, the hippocampus operates in combination with various other brain structures (e.g., those subserving activated agendas, those subserving the representations to be reactivated). Furthermore, we assume that these component processes and subsystems operate in a "distributed" fashion. For example, within the hippocampus, reactivation mechanisms are evidently replicated: unilateral hippocampal damage appears to disrupt reactivation of verbal information more than visual information or vice versa. Likewise, different areas of the frontal lobes may be important for shifting or noting different types of information. With increasingly sensitive cognitive tests and better characterizations of corresponding brain damage, it should become useful to make finer and finer

distinctions among areas within specific brain regions, such as the hippocampus and frontal lobes. At the same time, we expect to see common processing components, as represented in MEM, across distinctions that reflect input modality (auditory, visual), form (pictures, words), and content (faces, animals).

In sum, the subsystems of MEM contrast with the subsystems of many other memory models in that they provide a means of analyzing task-processing requirements rather than a means of distinguishing task content. Furthermore, MEM differs in the characterization of memory deficits: rather than positing a disruption of an entire subsystem, we posit a disrupted component process within a subsystem, which can lead to impairments of encoding, maintenance, and retrieval. When we eventually map MEM subsystems and component processes to brain regions, we expect that some, if not all, will be supported by circuits that may cross anatomically distinct brain regions. Tracing out these processing circuits is a great challenge and represents an opportunity to merge insights about processing derived from cognitive analyses with insights about structure derived from neurobiology.

5 CONCLUSIONS

An important goal of this book is to bring together and consider similarities and differences among subsystem approaches to memory. To distinguish between subsystems within a particular memory model, one can use any one (or many) of several criteria. For example, one might use biological interrelatedness as a criterion: we see this in Squire's (1992) model, in which the hippocampus and associated cortical regions are especially important for declarative memory, as opposed to nondeclarative memory, and in Moscovitch's (1992) model, in which the hippocampus is important for explicit, episodic, associative memories and the basal ganglia are important for procedural memories. In contrast, one might use computational similarity as a criterion for defining a subsystem: Rudy and Sutherland (1992) highlight a subsystem important for creating configural memories, as opposed to creating elemental memories. Another criterion for positing subsystems is the content of memories, such as speech-based information versus visuospatial information (Baddeley, 1992) or spatial information versus nonspatial information (Nadel, 1992). Clearly these criteria are not exclusive of one another: biologically based subsystems can be computationally distinct and computationally distinct subsystems can be biologically distinct. Rather, the situation is that a particular type of criterion tends to be used preferentially within particular models.

A useful by-product of the different criteria employed for defining subsystems is that work from different laboratories highlights different aspects of memory. Thus, categorizing memory tasks as implicit versus explicit (e.g., Metcalfe, Cottrell, & Mencl, 1992; Schacter, 1992) has emphasized the importance of awareness and its relationship to memory. Again, categorizing tasks as declarative versus procedural (e.g., Cohen & Squire, 1980) has highlighted

voluntary access versus skill acquisition. Similarly, categorizing memories as perceptual versus reflective (e.g., Johnson, 1990, 1992; Johnson & Hirst, 1993) emphasizes the important distinction between externally derived and internally generated components of the acquisition, maintenance, and retrieval of memories. Problems with some of these dichotomies have been discussed elsewhere (Johnson & Hirst, in press; Shimamura, 1989; Squire, 1987).

Problems aside, however, the various proposals about amnesia based on these categories do characterize different important manifestations of what we believe is a common *processing* disruption. For example, declarative, episodic, spatial, explicit, and relational memory all require binding together enough information to differentiate similar representations. We have tried to show how this binding of potentially differentiating featural information is greatly facilitated by reactivation processes that operate both within a session (e.g., when a subject becomes momentarily distracted and then thinks back to recent events or information) and over longer intervals (e.g., when one thinks about what happened yesterday). That is, reactivation is important for noting relations, creating declarative access through structured cues or through self-generated practice, and keeping alive episodic detail, for example. In short, positing a reactivation deficit provides a common processing account of these various characterizations of memory impairments in amnesia.

Researchers proposing subsystems intend for the subsystems to be more than useful alternative categories of description; they hope to capture a structural or functional architecture of the mind/brain that can sensibly organize as much existing data as possible and predict new data. MEM attempts a schematic representation of interactive interdependent functionality. Each of MEM's subsystems consists of sets of component processes that continually work interactively to accomplish functionally important tasks. The organization of component processes into subsystems (as opposed to an undifferentiated collection of components) creates coherence and efficiency in processing routines that call on components within a subsystem. Segregation among components created by subsystem "boundaries" reduces interference among processes and increases our capacity for multitasking (*tracking* a tennis ball and simultaneously *discovering* an opponents' strategy). The capacity for interaction between subsystems, especially under the control of executive and supervisory agendas, allows us access to useful information across subsystems, as, for example, when a reflectively *noted* relation calls up appropriate perceptual *structuring* processes. At this point in development, MEM's architecture attempts to reflect this functional analysis of memory and to suggest a vocabulary for beginning to describe differences in task-processing requirements.

In sum, we have proposed that a single process within a memory subsystem, namely *reactivation*, is important for both binding together and strengthening aspects of memory. These consequences of reactivation are, in turn, important for the encoding, maintenance, and retrieval phases of many memory tasks across a wide range of species. Hippocampal damage appears to be

one means by which disruptions in reactivation may occur in humans specifically and animals more generally. Disruption of a single component process can have consequences that are profound for all of memory, cutting across subsystem distinctions made in other memory models.

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NOTES

1. Throughout this chapter, "hippocampus" means "hippocampus and/or related structures." As pointed out by most investigators in this area, experimental lesions of the hippocampus may vary in size and are sometimes not confined to the hippocampus. Lesion variability is, of course, an even bigger problem in the study of amnesics.

2. In fact, some theorists propose that the effect of elaboration is no different than the effect of repetition: elaboration has its effect by adding to an item's overall level of familiarity by increasing the covert frequency of occurrence of any item entering a relationship with another item (Shaughnessy & Underwood, 1973) or by increasing the number of links contributing to the total amount of activation, which is assessed for familiarity (Gillund & Shiffrin, 1984).

3. Component processes may build on each other. For example, the potential success of *retrieving* will be influenced by prior successful *reactivations*.

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